Good Urodynamic Practice: Measuring Urethral Continence Function
Workshop 32
Tuesday 24 August 2010, 09:00 – 12:00

9:00	9:20	Biomechanical Principles of Sphincter Function	Werner Schaefer
9:20	9:40	Clinical view: What do we want/need to know about urethral closure function to improve diagnosis and/or treatment?	Charlie Nager
9:40	9:55	Biomechanical concepts of stress incontinence	Margot Damaser, Werner Schaefer
9:55	10:10	Pressure measurement - the details	Margot Damaser
10:10	10:30	The standard urodynamic investigation of urethral function – what does it offer	Gunnar Lose
10:30	11:00	Break	
11:00	11:15	Urethral pressure measurement - the problems	Werner Schaefer
11:15	11:30	The sleeve catheter - can it solve all problems?	Charlie Nager
11:30	11:45	Reflectometry - a different approach	Gunnar Lose
11:45	12:00	Round table discussion	

Aims of course/workshop

This workshop is intended to be a first step towards developing guidelines for good urodynamic practice of urethral pressure measurement, signal quality control, and interpretation. Thus, the aim of this workshop is not to provide any simple recipes for routine clinical application, but rather a critical analysis of the many problems of currently established clinical methodologies and research applications. Further, the workshop aims to evaluate the promises of new developments and try to define criteria for what we ideally want.

Educational Objectives

The first step will be to develop a correct understanding of the pressure in the closed urethra: its physiological origin and role, its physically correct definition and the specific conditions of measurement. In principle two different approaches are in common use today. The direct intraurethral measurement with a probe in the urethra (e.g. air and water filled balloon, microtips, different perfusion) are most commonly used to measure the pressure distribution along the length, the urethral pressure profile, UPP, at rest and under stress, and indirect measurement by increasing intravesical pressure and forcing the urethra to open so that leakage can occur, the so-called leak point pressure, LPP. With the simple physical principle that leakage can only occur when the intravesical pressure is equal/higher than the urethral pressure, it is also immediately clear that if all these methods would measure what they claim to do, all should result in the same values for the maximum urethral pressure. Obviously they do not.

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We will outline the various physiological and physical aspects that can contribute to these differences and identify the most common artifacts which currently limit the clinical usefulness. We will discuss the potential of new concepts, such as the sleeve catheter which should allow a better measurement of maximum urethral pressure, and the additional information on the biomechanics of urethral closure function which can be gained by reflectometry. Finally, in interaction with the audience, we will try to elaborate a set of criteria to identify "perfect" physical and physiological measurement of urethral pressure, and what the clinical relevance of such a parameter could be.

(all presentation times include discussion)

There can hardly be a greater difference between theory and praxis: the biomechanical principles of urethral continence function are straight-forward and simple. As long as the urethral pressure is higher than the intravesical pressure, no leak can occur. However, the precise definition is not easy at all, and ultimately the precise measurement of urethral pressure is one of the most difficult urodynamic tasks.

The theory of urethral pressure is simple, taking normal anatomy at rest: urethral closure means that the mucosal surface is in full contact, at least in some cross-sectional area, acting on each other with the urethral pressure. The mucosa is surrounded by the lamina propria, and the longitudinal smooth, and circular smooth and striated muscle, and distribution and amounts of all these structures are different along the urethral length. A pressure can be build up by contraction of sphincteric muscle, i.e. active force in circular structures. The active force in a muscle depends on pre-stretched and contracted length, which are for the sphincter determined by the volume of enclosed tissue, respectively blood volume present, mainly in the lamina propria. However, it is not the blood volume which determine the urethral closure pressure but the tissue pressure which determines the blood volume present together with blood pressure.
From this simple physiological model it is clear that pressure with which the soft easily deformable mucosal surface close on each other cannot show any directional difference. A pressure difference between two surfaces which act on each other is simply impossible, because they “generate” this pressure by their interaction only and there are no other force involved. Further, it is reasonable to assume that within the tissue volume enclosed by the sphincter a radial pressure gradient cannot exist, only an axial/longitudinal pressure gradient can exist within the longitudinal smooth muscle.

As a further complication, the urethra is embedded in very different surroundings which can have an impact. The urethra extends from the bladder neck, - normally under the influence of the abdominal pressure from the outside and the intravesical pressure from the inside -, through the pelvic floor, where any activity of these complex structure will influence urethral pressure, to the distal meatus at the external body surface, where by definition the urodynamic zero pressure is. Thus along the length the urethral pressure will be different due to contributions from various internal and external structures, and the longitudinal urethral pressure profile UPP goes from intravesical pressure to some sphincteric maximum pressure down to zero. Somewhere the gradient from abdominal to zero pressure will be superimposed to this UPP.

In addition to these original urethral structures most of the surrounding structure can change voluntarily and involuntarily, directly or indirectly, as function of time. Further, the urethra can be significantly deformed and/or moved due to changes in posture and most dramatically due to various forms and degrees of prolapse.
In consequence: even if we can precisely measure the intraurethral pressure along the length, we will often not be able to attribute this pressure or its changes clearly to a distinct structure.

Problems in Urethral Pressure Recordings.
A huge body of literature exists dealing with urethral closure function and a large variety of urodynamic measurements, different techniques and different catheters, but with little attention to artifacts. However, all catheter-based measurements in the urethra will create a signal in interactions between the catheter and the urethral wall. In all these catheters

with a sensing surface, like microtips, or a perfusion hole, the recorded signal will have inevitably a **directional component**. This, however, is not the urethral closing “pressure” we aim to measure, but a classical artifact = the directional component is caused by the catheter and not existing in the urethra without a catheter present. Only with system which do not record directionally, such as e.g. balloons or ring-like perfusion openings such directional component can be avoided.

Further, the **dynamic resolution in time** of the signal is mostly unclear. It is very obvious and well established that the dynamics of any perfusion pressure signal depends on the perfusion rate, and is rather slow. Similarly any signal recorded with an air-filled balloon will have a reduced sensing capability of fast signals. In addition, air-filled system like microtips will have the hydrostatic **reference level** at the site of primary sensing element, i.e. inside the urethra. This makes it difficult to establish a common reference level with intravesical and abdominal pressure.

As the urethral pressure is different along the length, it is of utmost importance to control the localization of the **sensing site**. Only when the sensing site is fully controlled it can be identified if pressure changes are real local changes in time or just changes in position. As the urethra is quite mobile, particularly in patients with stress incontinence, it is impossible under conditions such as a moving patient, under straining or coughing to control the precise location of the measurement. This is a most critical artifact in the large variety of stress-UPPs, usually ignored, often interpreted “pathophysiologically”. This “location” artifact may be avoided by the sleeve-catheter, or can be corrected with multiple measuring sites.

Some Biomechanical Aspects of Continence Function

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The investigations of incontinence and outflow obstruction are key issues in urodynamics. The investigations of bladder storage and voiding function are well standardized. For the investigation of urethral closure function, however, we have no standard method of measurement. Numerous different techniques have been developed and tested, and extensively applied in the clinic. Overall, the results have been disappointing. Accuracy and reproducibility are still under discussion, and correlation to pathophysiological changes and the clinical degree of incontinence are poor, so that none of the methods is generally accepted as a standard. There are a variety of reasons for this. In the context of this paper it is not possible to give a comprehensive review of only the most popular methods for urodynamic measurement of urethral closure function, with a useful analysis of their specific technical problems, limitations as well as their potential information. This is already complicated by rather unclear terminology. Therefore I will have to add quite some introductory statements. Most of my discussion deals with the theoretical aspects of continence and the bladder closure mechanism. I know from previous discussions with clinicians the argument that this is just theory and therefore has nothing to do with clinical or physiological reality. However, I think this is a misunderstanding of what theory can offer in reality. The biggest problem we have today in this field of urodynamics is the non-organized combination of an abundance of poorly controlled data with speculative interpretations and anecdotal reports about clinical observations using vague terminology. What we are missing is careful discussion of the quality and value of the information we have, clear identification of the information we need, and a careful consideration of theoretical principles which may allow to transform the current confusion to a scientific approach (1–3). Therefore my theoretical considerations will be performed on a very basic level which is sound and transparent. Only then we can avoid the situation whereby the current confusion of unclear data may just be covered up with an equally obscure theory.

URETHRAL PRESSURE

For many years an attempt has been made to measure urethral closure function as “urethral pressure” and to derive a “urethral closure pressure” according to ICS Standardization (4). This is where the problems begin because a generally accepted and precise, physically correct and physiologically meaningful definition of “urethral pressure” is not in use. It is not uncommon in medicine that a parameter such as pressure—which is strictly defined in physics—is used clinically in a form which is not in agreement with the original physical definition. Such a loosely descriptive terminology, however, makes it very difficult—if not impossible—to compare different published methods of measurement and to analyze critically their results for artifacts and precision on a physically and physiologically meaningful level. They all use the term “urethral pressure” differently and in fact measure slightly or significantly different things. This is a real problem when we compare static and dynamic urethral pressure profiles, when we compare perfusion pressures with microtip-transducers, and when we compare urethral closure pressure with the variety of different leak point pressures. If properly defined and measured, all these methods should give consistent results, which they obviously do not.

A good definition for “urethral pressure” can be derived following the ICS standard terminology: “incontinence occurs when intravesical pressure exceeds the urethral pressure”. Here, “urethral pressure” is the fluid pressure needed to force the urethra open, and: urethral closure pressure (UCP) is the difference of urethral minus intravesical pressure. When the UCP is positive, the urethra is closed, when UCP is negative,

the urethra leaks. This definition of a fluid pressure is an idealized concept but it is clear and in agreement with the physical definition of pressure. Such a "urethral pressure" allows a definite control of signal quality, independent of the method of measurement used, as well as a meaningful interpretation.

It is easy to see that such a pressure can be recorded by using the perfusion technique, which physically "simulates" a urethral pressure as a local intraluminal fluid pressure. This technique allows good recordings of static closure pressures but has a limited dynamic response when a cough or stress profile is recorded. During the 1970s the so-called microtip-transducer catheters (MTC) became very popular in urodynamics and a vast number of publications report endless details about clinical measurements. MTC pressures do have some specific features which need to be considered. First, in a fluid-filled surrounding MTC can measure the local fluid pressure very accurately but the reference height may be difficult to determine as the reference level is at the transducer itself. If the MTC is not used inside a fluid-filled surrounding but in the closed urethra, then this differs from the perfusion technique as the MTC will not imitate the situation of a local intraurethral fluid pressure but record a pressure as interaction of a transducer surface with urethral tissue. To repeat this: a MTC senses the force of tissue on its transducer surface, and the signal is processed so that it gives a pressure, e.g. where 1 gram force on a area of 1 mm^2 results in a pressure of 100 cmH_2O. It is acceptable to discuss the local tissue/transducer interaction in terms of pressure; it is not acceptable to take this tissue/transducer pressure as an accurate measure of closure function in terms of urethral pressure. This distinction is important. MTC weight or bending or any inhomogeneity in the urethral wall tissue can lead to local directional tissue/transducer interaction which will superimpose the desired urethral pressure signal. Clearly, therefore, these MTC-pressures show directional differences of "pressure" recording when the transducer is rotated in the urethra. These directional MTC signals are interpreted in many ways, mostly incorrectly, because they are not a urethral pressure according to the definition above (5, 6). In addition we can use a formal argument, that in physical definition a pressure is a scalar and thus does not have a direction and therefore cannot show directional differences. This shows the value of a precise definition of urethral pressure and UCP. From this definition it is clear that the reported directional differences are artifacts. In more descriptive terms this means: the reported directional differences of urethral pressure do not exist in the urethra without the MTC present; or: the derived directional signals show that the local interaction between the catheter and the urethral wall are related to many details of MTC and the urethra, but not to urethral closure function. It may be possible to correlate such directional signals with anatomical structures in or around the urethra. However assessing these structures or assessing closure function in terms of urethral pressure are very different issues and need different approaches. For such structural assessment we would need a standard MTC, preferably completely rigid and straight, and presumably with a larger diameter to provide a better directional resolution. For assessing closure function as urethral pressure we ideally should use a very thin, weightless, and extremely flexible MTC to minimize artifacts (7–11).

These artifacts become very significant and totally uncontrollable when cough or stress urethral pressure profiles are recorded which try to approximate more closely the typical situation when stress incontinence occurs. This also leads to additional problems. The events at coughing are rather fast and usually these patients show a significant mobility of pelvic floor and urethra. Under these conditions it becomes very difficult—if not impossible—to control the precise location of a pressure recording site and to make sure that the recording site does not change during a cough. Any relative movement of the measuring catheter in the urethra will have a strong impact on the recorded signal, just according to the different pressures along the urethral length. In addition, MTC acceleration and bending will influence the signal. Thus, we must conclude that recording of a UCP with MTCs under stress conditions will require a very strict signal quality control as many potential artifacts can obviously occur. Signal quality control, however, is only possible with a strict definition of what we want to measure. Nevertheless, the most common reaction to these difficulties in measurements has been just pragmatism or even ignorance. I think that intraurethral cough pressure signals are so critical that a real signal quality control may only be possible with an automated electronic approach, such as compensation of these directional components. At least, such electronic compensation is much more meaningful than the computer generation of an "asymmetrical three-dimensional urethral closure pressure profile".

LEAK POINT PRESSURES

In recent years, various forms of measurement of a "leak point pressure" (LPP) have become popular and are under investigation or even in routine use in many clinics. At a first glance LPP appears to be a simple method to assess closure function which should avoid most of the problems of urethral pressure discussed above. In principle it should give results consistent with our definition of urethral pressure, as a "leak point

pressure" actually seems to imitate the situation described in the ICS definition of incontinence and urethral pressure. However, the variety of detrusor and abdominal, of cough and Valsalva "leak point pressures" already indicate the lack of reproducibility and standardization (12, 13). Similar to the "stress profile" the measurement of a "cough leak point pressure" is complicated not only by the fast dynamic changes in intravesical, abdominal, and urethral pressure, but also by tissue deformation and acceleration. In addition, the direct or indirect observation of urine leak and its correlation to any definite pressure value of a cough spike which only lasts 1 sec or less is not realistically possible and will lead to significant subjective errors. Further, considering the complexity and variability of the pelvic and urogenital anatomy, the variety of active and passive structures, the different forms of coughing and straining, which will lead to different forms of stress and different impact on the bladder closure mechanism, it seems to me obvious that any LPP value—be it intravesical, abdominal, detrusor—somehow will reflect the complexity and variability of the system. I think the simplistic concept of a "leak point pressure" is in conflict with reality. At a closer look, a rapid dynamic pressure change which will cause leakage is actually not at all a simple parameter which can be easily defined and reproducibly measured. Recently, Höfner et al. (14) have presented a computerized approach to "leak point pressure". This method seems to have some technical advantages, as it measures the pressure spike and the urine leak at high dynamic resolution. But the principal problem of correct synchronization of leakage flow and pressure remains, and this problem may just become more transparent now, as this computerized method shows rather different pressure/leakage patterns. It is interesting to note from their first results they find only a good correlation between such a "precisely" measured leak pressure value and the leakage volume. This result is trivial and it brings us back to pre-urodynamics times. Nevertheless, I think that a precise measurement of leakage flow and pressure may lead to a better understanding of some details of the event of incontinence. However good measurement of a LPP is as difficult as a UCP, and I think the potential information is different. Careful urethral pressure recordings will allow the investigation of the pathophysiology of urethral closure function, while the combined leakage and pressure recording will help to understand the dynamics of incontinence. Any LPP-value determined by simple observation of leakage and some pressure can hardly be converted to a standardized and reproducible procedure.

In summary, there is a huge body of literature about the urodynamic measurement and quantification of urethral closure function. This has to be viewed with great caution because all investigational methods have specific problems which make it impossible to compare them in a meaningful way. When we follow the ICS-derived definition of an idealized urethral pressure, and when all described methods identify and define clearly what they really measure, then all these methods should come to consistent results, ideally to consistent pressure values. So it seems that we are still far away from a standard urodynamic method for the investigation of urethral closure function and the pathophysiology of incontinence. But nevertheless, I have no doubt that with a very careful and critical urodynamic approach, information about urethral closure function can be derived which is accurate and reproducible, objective and of clinical relevance.

URODYNAMICS AND INCONTINENCE

When we compare the urodynamics of continence function with the urodynamics of voiding function then we can identify some remarkable differences. Incontinence is a clearly defined sign and a symptom which can be objectively demonstrated without any urodynamic measurement. Thus, no urodynamic parameters or criteria are needed to identify incontinence, and we can rely on the leakage volume to quantify incontinence. This is very different with bladder outflow obstruction. Only after many years of systematic urodynamic research have we learned that there are no clear signs or symptoms of obstruction but the only way of objectively identifying and quantifying obstruction is by urodynamics. In this way obstruction is a urodynamic diagnosis, incontinence is not. So what is the potential role of urodynamics in incontinence? Clearly, it can become an important tool to investigate and to quantify the underlying pathophysiology in research and clinic, and actually urodynamics is the only objective and quantitative approach I can see. But first we have to learn to discuss and conceptualize incontinence in terms and parameters which can be measured urodynamically.

With respect to the current clinical practice, the need for such an investigational method may not appear to be urgent. Some hundred-plus more or less different surgical methods claim to cure incontinence with a very high success rate, with the usual tendency that the more invasive or newer techniques have the higher success rate. However, most surgical methods are based on rather vaguely described understanding or misunderstanding of the underlying pathophysiology as well as the mechanism of how the surgery does correct this. In addition, at least some of these ideas and concepts, I think, are wrong from a realistic pathophysiological point of view. And lastly some (or most?)

surgical methods are not as successful as is claimed, at least not over an acceptable period of time. However, here the situation is comparable with the urodynamic confusion: a profound lack of standards and of reliable and comparable information. It is remarkable that still to date new surgical methods are developed more in a try-and-error fashion than in a controlled scientific approach (15).

URODYNAMICS AND BIOMECHANICS

The typical urodynamic signals such as pressure and volume and their respective changes in time are mechanical parameters. Therefore I think that as indispensable first steps in a urodynamic approach to the continence mechanism we need in addition a clear understanding of physical definitions and technical limitations of measurements a clear description of the biomechanical aspects of continence. Such an explicit mechanical approach may sound irritating to physicians, but I think that closure of a soft tube and prevention of leakage, or vice versa the failure of closure and occurrence of leakage from a fluid-filled container, have many mechanical aspects or can be suitably described in mechanical terminology. Further, one could argue that surgery mainly treats incontinence by introducing mechanical changes. The urodynamics of the voiding function has only been understood after the fluid and muscle mechanical aspects of urethra and detrusor had been understood. Similarly, we will not understand the pathophysiology of continence without a sound and valid biomechanical model. Further, such a biomechanical model is indispensable for performance of proper plausibility and quality control before a meaningful pathophysiological interpretation of the urodynamic signals becomes possible.

These mechanical considerations cannot substitute for the need to understand the anatomy and physiology of all structure and tissues involved. However, the anatomy and pathophysiology of the lower urinary tract is so complex and variable that we must exploit all possible tools to investigate, and all methodologies to organize our understanding of continence function to a clinically useful and scientifically valid concept. As stated in "Good urodynamic practice, GUP" (3), we need a step-by-step approach to urodynamic signals with plausibility checks on various levels: first, we must precisely define what we want to measure and make sure that what we measure is in agreement with the laws of physics and principles of mechanics. Second, we must understand the methodology of measurement, the technical limitations, the possible artifacts, and the potential information contained in our signals. And only then, at a third step, can we start with a meaningful pathophysiological interpretation and can derive correct and clinically useful information. Particularly in the urodynamics of incontinence it is still common practice today, to produce "some urodynamic signal" and to interpret it immediately clinically in a very pragmatic and rather anecdotal form, without bothering about any of the indispensable steps in between. A typical phenomenon is the continuous re-investigation of the "directional properties of urethral closure pressure". There is no doubt that the complexity of the continence mechanism requires to use all possible methods and instruments for investigating the pathophysiology which all have their specific limitations. Apparently simple approaches such as the attempts to understand the anatomy by dissections and imaging have lead to results which remained controversial over the years. The findings are so complex that we need additional sources of information to test any functional hypothesis. It is true for all approaches that "you only see what you look for, and you only look for what you know". Thus an anatomical approach will focus on a clear description of type, orientation, and location of muscles and nerves and other tissues. This alone bares the risk of misinterpretation of its functional meaning, or even that the preparation, dissection, observation and identification may be misguided by a pre-existing functional model. Here I think mechanical considerations can be used as objective and powerful controls. Any functional interpretation of muscle contraction and relaxation and their mechanical effects in a three-dimensional space which is not in agreement with the basic mechanical concept cannot be correct. Because of the amount of unclear or even misleading information published it is better to start from a basic and most simple level. Only after we have reached agreement on this elementary level we should make the next step and developed more complex and sophisticated models. Thus here I will focus on the first and second level of urodynamic plausibility control according to GUP (3) using simple mechanical concepts and tools.

BASIC MECHANICS

There are some basic, very simple concepts and tools used in mechanics and engineering which we can apply rather easily in urodynamics and which are underutilized so far. If this is done properly we will discover that these can be powerful tools, at least in avoiding misconceptions and to identify artifacts. I will concentrate here on stress incontinence using this simple definition: a bladder closure mechanism is competent under static conditions but leaks when it is challenged by additional mechanical stress due to abdominal pressure increase. First, we will try to describe the mechanical properties of essential elements and struc-

tures separately, and then we will try to combine these structures and elements to model a functioning sphincter unit in as much agreement with the actual anatomy as possible on a simple level. Then we will discuss how this sphincter unit is possibly integrated into the pelvic floor and how it may be challenged by abdominal pressure increase and deformation. Some consequences of changes in various elements and structures will be discussed, and finally we will try to outline the mechanical impact of surgery and identify some misconceptions.

Elements and micro-structure

Passive and active elements are involved. Passive are, for example, bones, collagen, urothelium, and fatty tissues. One relevant mechanical feature of collagen is that fibers have one-directional properties and in form of fascia have two-dimensional properties. These directional properties indicate the direction of the predominant load. Urothelium and fat are more mechanically homogeneous tissues which will deform under an external load equally in all directions. All the tissues involved here have in common that they are incompressible. Usually I hear many objections when saying this, but it seems to me this is mainly due to the fact that this clear physical term is often misunderstood. Therefore I will add some explanations. Water is incompressible, i.e. a given volume of water is easy to deform, but the volume cannot be reduced under pressure. Gas is even easier to deform and a given volume will decrease under pressure, i.e. gas is compressible. Water is one of the main components of biological tissues, so that it is easy to remember that tissue is incompressible. Incompressibility is often confused with deformability, but this is a very different aspect of tissue mechanics. Deformability depends mainly on the more-or-less elastic or fibrous elements of the tissues, in particular on their directional orientation. Fat and urothelium deform easily and do not seem to have directional properties. Under constant load they will yield to a load and behave more like soft rubber, or even like a highly viscous fluid, e.g. tar. Fibrous tissues have distinct directional properties so that they can bare a strong force when pulled in direction of the fibers but less in the other direction, e.g. perpendicular to the fiber orientation. Therefore it is realistic to assume that the orientation of fibers always indicates the main direction of load.

Soft tissues often show viscoelastic properties, i.e. their elasticity and deformation depend on how fast the load varies. Also soft tissues will yield under constant load, particularly outside their elastic range. Only active elements such as muscle can ensure and restore long-term stability, so long as they are not damaged, i.e. overloaded. In this simple biomechanical approach I discuss muscle—irrespective of any anatomical subclassification, whether striated or smooth, fast or slow twitch—as a directional (fibrous) element which can change its length and generate a force within definite limits. The length/force-relationship in different muscles will depend on its type and its activation, and in general the force will depend on the length, i.e. if we change the passive or active length of a muscle we will change its possible force. It is important to keep in mind the directional aspect of both change in length as well as force. It is further important to understand that a parallel combination of muscle cells will increase the potential force, while a serial combination will increase the maximum change in length as well as the contraction velocity, but not the force (Fig. 1). All elements in a chain will bear the same force, and the weakest element will determine the maximum load for the complete chain. Again: in a serial combination of muscle cells the forces will not add up. However, the energy consumption for such a chain—the energy per unit force—will more or less multiply with the number of muscle cells in a serial combination. Thus, a serial combination of muscle fibers has no "advantage" with respect to force development, only with respect to contraction speed and potential length change. Theoretically a single muscle cell in serial combination with collagen fibers can develop the same force but consumes obviously much less energy than multiple muscle cells in a series (Fig. 1).

Only in parallel combination can the forces of muscle fibers add up. Further it should be kept in mind that muscle is incompressible and the muscle volume is quite constant, whether active or passive. This simply means that any reduction in length will result in a corresponding increase in diameter and vice versa (Fig. 1).

Before we can combine these elements and tissues to a realistic structure, we should consider some simple (bio)mechanical concepts and tools.

The equilibrium of forces

In a given structure which does not move or deform, there must exist an equilibrium of forces (Figs 2 and 3). If an additional force is introduced, the structure will move or deform until it finds a new equilibrium. Any force without balance, i.e. any resultant force, will lead to acceleration. In complex structures it can be difficult to understand the conditions for equilibrium and to identify the forces involved, particularly in a three-dimensional space. Even more so it is difficult to quantify these forces, i.e. to identify direction and magnitude of forces. There is a rather simple method in engineering which can help to identify and understand these forces, even when these are not obvious external forces but internal forces, i.e. force acting inside a

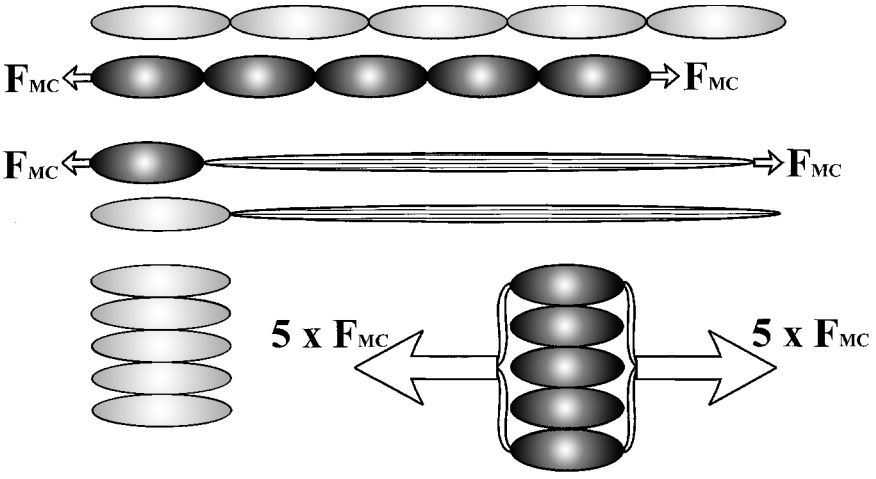


Fig. 1. Assuming a standard muscle cell with a given relaxed length and diameter, which by contraction generates a force FMC and shortens 20% in length. Because the cell volume stays constant the diameter will increase by 20%. In a serial combination the total shortening as well as the contraction velocity increases proportional to the number of cells (i.e. the muscle length), but the total force of a serial combination is not higher than that of a single cell, or a single muscle cell combined with passive tissue. When such standard muscle cells are combined in parallel, then the total force increases proportional to the number of parallel cells, i.e. muscle thickness. Neither the total shortening nor contraction velocity increase, but the total diameter increases in proportion.

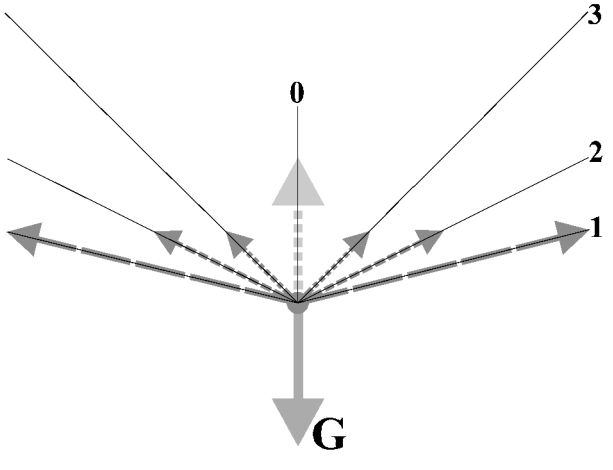


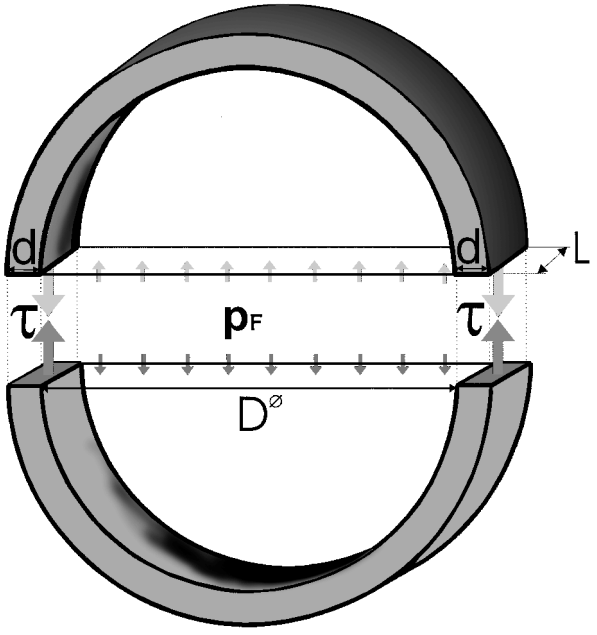
Fig. 2. The equilibrium of forces is illustrated showing the influence of direction and strength of forces. Let us consider a simple example of a weight G hanging on a rope. Here the direction of forces is obvious: it is the direction of the rope, which comparable with many "directional" anatomical structures. Four different positions (0–3) for the rope are indicated here by thin black lines. The length of the arrows represent the strength of the force. In position 1, where the rope is almost horizontal, the forces needed to balance the weight force G are very large. When the rope hangs lower down, as in positions 2 and 3, the forces in the rope required to balance the weight G become much smaller. Ultimately with a vertical rope (position 0) the force in the rope has the same strength as G but in the opposite direction. I discuss this example so explicitly because similar configurations will occur in slings and hammocks and other supportive structures. It is interesting to note how much the strength of the balancing forces depends on the direction, and how much the balancing forces can be smaller when a supporting structure descends. A different example for equilibrium of forces is discussed in Fig. 3.

structure or tissue. Conceptually, we imagine that we cut a distinct part of the structure and then we try to understand what the mechanical effect of this cut would be, i.e. how the structure would deform or even fall apart because of this cut (Fig. 3). Then we can identify and theoretically add the forces needed to avoid deformation or disintegration of the structure, i.e. to substitute or compensate for the effect of the cut. In this way we can make internal (hidden) forces to external forces which are much easier to investigate and to understand. A classic example for the application of this method in urology is the so-called "La-Place law" used to derive the relation between bladder pressure and forces in the bladder wall. This can also be used to understand the relation between urethral pressure and sphincter muscle force (Fig. 3).

SPHINCTER MECHANICS

A sphincter is an arrangement of active and passive tissues which can open and close an enclosed tubular structure. This mechanical definition as some other following statements may sound ridiculously oversimplified, but such a definition is helpful, mechanically and biologically.

The only soft tissue arrangement which can close and seal a tubular structure with pressure as well as allow an internal lumen to open is a circular arrangement of active and passive fibers. The characteristics of opening size and closure pressure will be determined by how much these fibers can change their length and exert a force, and by the spatial arrangements and



$$p_F \; D^{\varnothing} L = 2\tau \, d \, L$$

$$\frac{p_F}{2\tau} = \frac{d}{D^{\varnothing}}$$

Fig. 3. Considering a very simple sphincteric structure, i.e. a circular shell with an internal pressure pF. When we cut this shell in the midline, we can unveil the equilibrium between the internal pressure as well as the forces in the wall. In such a shell the wall forces are the same around the circumference. In the cut plain the pressure acts on an area D × L and the wall tension acts on the areas d × L. From the condition of the equilibrium we find that for given muscle strength and urethral pressure the diameter D and muscle thickness d are proportional.

dimensions. I make this very simplistic statement here because in the anatomical literature it is sometimes postulated that a "real" sphincter requires a complete circular arrangement of muscle fibers, and that any fractional circular muscle or any circular combinations of partially muscle and partially collagen fibers are often described as "incomplete" or "not a real" sphincter (16—18). According to my definition such distinction is not meaningful, as the relative contribution of muscle and of passive fibers only will have an impact of sphincter strength, speed and opening size, but not on the sphincter function in principle (Fig. 4a and b). Closure is active—i.e. requiring muscle contraction—and opening is passive—i.e. requiring muscle relaxation and an internal fluid pressure to open and unfold this internal lumen. Any active mechanical opening of such a sphincter would require radially oriented muscle fibers with external anchoring, because only in this way a circular structure could be pulled open. We can develop some more simple mechanical rules to analyze sphincter function. A sphincter is a circular and not an oval structure, as it appears sometimes in histology (17, 19–21) because only a circular structure is stable without additional forces acting from outside or inside. The wall tension needed to balance the closure pressure will be minimal in a circular configuration and will be the same around the circumference (Fig. 3). This is important but often ignored: the wall tension is the same around the whole circumference.

Simple geometrical considerations tell us that the circumferential length is in definite relation to the sphincter diameter and that the change in circumferential length is in definite relation to the size of the lumen which can open internally. The first conclusion is that a sphincter made out of circular muscle alone is impossible. Closing an internal lumen by contraction of muscle fibers alone would require contraction to zero length for the innermost fibers lining the wall of the internal lumen, simply because the circumferential length around a closed lumen is zero (Fig. 4a).

Thus, sphincter function requires some other tissue volume in the central area which may be deformed and folded easily—circumferentially and radially—by the surrounding circumferentially oriented fibers. Such central passive "filler" volume may have no specific directional mechanical properties or could have an axial, i.e. longitudinal fibrous structure. Nevertheless, this central "filler" volume is important as it determines the minimal circular muscle fiber length in a closed sphincter and the filler volume together with the area of the open lumen determines the maximum muscle length of the open sphincter. Thus, the amount of "filler" volume will determine the length change in the circular sphincter muscle between the open and closed state. The more "filler" volume the less length change is required, i.e. somewhere between the impossible 100% contraction to zero length with no filler volume to some more realistic 10–30% length change when the diameter of the "filler" volume in the closed sphincter is double or equal the size of the open lumen (Fig. 4a).

Thus, the proportion between filler volume and open lumen size and the relative length change in the circular sphincter structure is determined by geometry. In addition, the relation between closure pressure and muscle force in the circular sphincter structures depends on the "filler" volume in two ways. One is just the mechanical equilibrium: the more "filler" volume, i.e. the larger the diameter, the more force is needed to build up a given closure pressure by contraction. Some other aspect is the physiological length/force relationship of the muscle. Assuming that the normal configuration is "optimized" we should expect that whenever the "filler" volume is reduced and

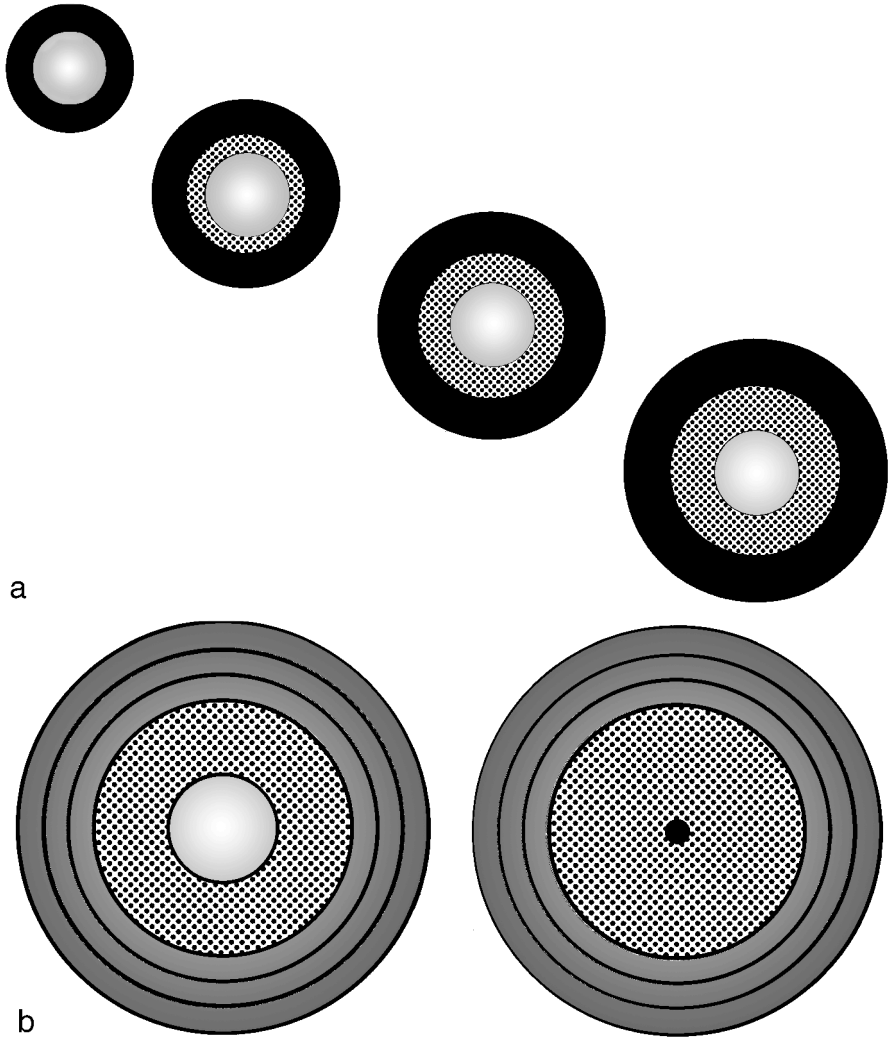


Fig. 4. (a) Because the muscle (outer dark ring) cannot contract to zero length it is not possible that the sphincter wall consists of circular muscle alone (upper left corner). If the inner lining of the opened internal lumen consists of circular muscle, then these muscle fibers would need to contract 100% to close the lumen. If around the internal lumen an area of passive filler volume (dotted area) exists equal to the area of the open lumen, then the inner muscle fibers only have to shorten 30% around this filler volume to close the internal lumen. With increasing amount of filler volume the percentage of muscular shortening needed for closure decreases rapidly, here to 20% and 13% for filler volumes double and triple the open lumen size. (b) Consider a simple sphincter where the diameter of the open lumen is approximately 20% of the total sphincter diameter, and filler volume and muscle thickness are equally 40% of the total diameter. If such a sphincter closes the external diameter decreases only 1%, and the complete circular muscle length have to decrease on average by just 5%, i.e. between 1% outside and 9% inside. If the circular muscles are combined with passive fibers then the necessary shortening will increase equivalent to the percentage passive tissue around the circumference, i.e. if half the circumferential length are passive fibers, then the average muscular shortening will increase to 10%. Thus, with significant enclosed filler volume only little change in size and muscle length will occur between the closed and the open state.

thus the final closure length is less than optimal, then the potential muscle force is lower.

In summary, assuming a "standard muscle cell", we can say that with more "filler" volume and thus increasing sphincter diameter, the sphincter muscle must be stronger for the same pressure, i.e. the number of parallel muscle fibers and thus thickness of the sphincter wall must increase, but the share of muscle fibers along the circumferential length, i.e. the number serial muscle fibers can decrease. The opposite applies with less "filler" volume (Figs 4b and 5). Thus we cannot expect a close relationship between sphincter size, volume, and pressure (22).

Without knowing the precise muscle mechanical performance of human sphincter muscle, we can still discuss the situation further qualitatively. In principle we assume that the sphincter function is performed energy efficient, i.e. with as little energy consuming muscle as needed. Considering closure to be the main function, then a circular serial combination of muscle and collagen fibers around a passive "filler" volume is mechanically the best solution. It also must be kept in

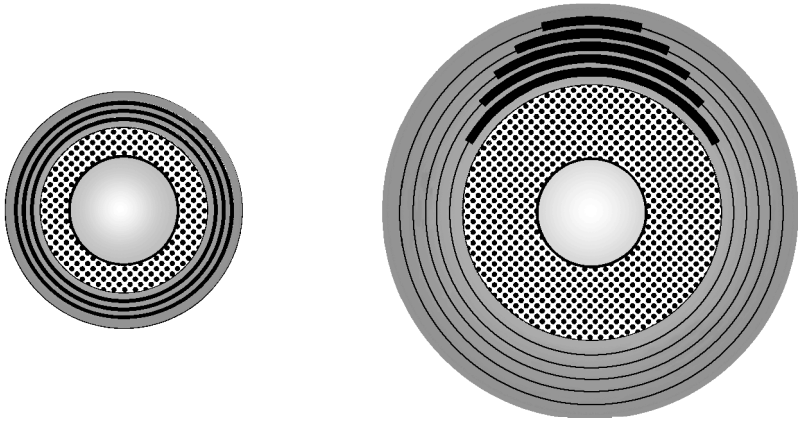


Fig. 5. When we compare two sphincters with comparable closure pressure but very different amounts of filler volume, we can use geometrical considerations and the equilibrium of forces to deduce theoretically some specific differences. With little filler volume, and thus small diameter (left), i.e. little force, a sphincter can work with a thin muscle coat (thick circular lines) which, however, needs to be quite complete around the circumference to achieve sufficient shortening. With more filler volume (right) much less shortening is needed so that a significant part of the circumference can be made of passive fibers (thin circular lines), but more force is requested so that the sphincter coat must be thicker (thick lines).

mind that the sphincter wall tension around the circumference is the same in all segments, i.e. in all active and passive elements constituting the sphincter wall. Whenever we learn from anatomical studies focusing on detection of circular muscles that a complete circular muscle ring does not exist, or that the sphincter is thicker in one segment than in others, then the logical mechanical consequence would be the search for finding the non-muscular tissue on which these muscles act to maintain a circular equilibrium. Any segment around the circumference where the muscle coat is thinner or absent (17, 18) can only be explained by other non-muscular tissue bearing part or all of the circular tension, and thus mechanically being part of the sphincter. Alternatively, we could assume that the muscle there is stronger so that it needs less muscle cross-section to bear the same force. I think this explanation is unlikely but has to be tested. The typical anatomical interpretation of an "incomplete" or "no real" sphincter would therefore be disputed from a mechanical point of view, where an optimal sphincter will be a combination of muscle and passive tissue. In the end, what should any partial circular muscular arrangement do except act on neighbor tissue? And so long as there is even just a fraction of truly circular muscle, compressing enclosed tissue is the most likely functional explanation.

Circular tension, tissue compression, and longitudinal tension

Assuming a tubular sphincter structure is pressing on enclosed soft tissue, and further assuming the enclosed tissue to have homogeneic mechanical properties without active or fibrous elements. As passive soft tissue will yield to long-term pressure and as the ends of this tubular structure will be open, it is clear that the enclosed soft tissue will be squeezed out at the open ends (Fig. 6). Thus, the enclosed soft tissue must be stabilized in an axial direction by some longitudinal passive and/or active elements. The forces which these longitudinal elements will have to bear depend on the axial pressure gradient, as shown by the slope of urethral pressure profile. If the closure pressure is constant over the sphincter length and then falls to zero at both ends, then the pressure gradient would be very high at the ends. However, with an arc-shaped axial pressure distribution we will have a minimal longitudinal pressure gradient from the central maximum closure pressure to both ends (Fig. 6). This longitudinal tension can be balanced by the combination of longitudinal muscle and collagen fibers (23) within the central "filler" volume. And as this is a long-term stabilization function it is no surprise that this is mainly collagen with relatively little smooth muscle.

However, the role of this longitudinal muscle has often been discussed as pulling the bladder neck open at the initiation of voiding (17, 18, 24). For the longitudinal muscle to exert an effective pulling force at the bladder neck it would need for equilibrium some anchorage distally along the axes, either within the sphincter or even in the distal urethra. I have no indication that such an anchorage does exist. Also, I have no indication that the longitudinal muscles contract when the circular muscle relax during the initiation of voiding.

In addition, for any effective contraction of the longitudinal muscles we must assume that with shortening the thickness of the longitudinal layer will

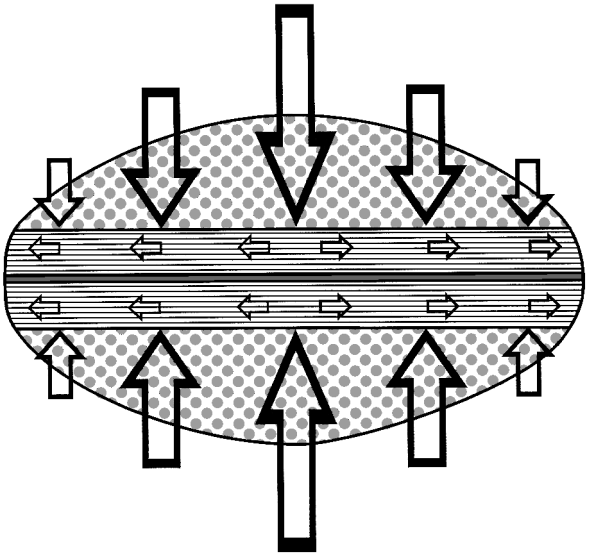


Fig. 6. The pressure of the circular muscle on the filler volume will lead to an internal pressure gradient. Therefore this filler volume needs axial, i.e. longitudinal muscle fibers, to ensure long-term stability by counteracting and balancing this pressure gradient. When these longitudinal muscles contract they will increase in diameter and thus contribute to the closure function. With the proportion shown here less than 10% of shortening would be sufficient to close the internal lumen by contraction of the longitudinal muscles alone. It is highly unlikely that the longitudinal muscles should be active for opening the bladder neck at the initiation of voiding, because on one hand their increasing diameter would be counteractive for opening and on the other hand the longitudinal muscle fibers would need some axial anchorage in the sphincteric or distal urethra to allow effective pulling on the bladder neck.

increase, which would be counteractive for opening a lumen in the sphincteric urethra. I rather think that contraction of the longitudinal muscle will contribute towards an effective sphincter closure mechanism rather than an opening. Theoretically, even a pure "longitudinal (i.e. axial) sphincter" is conceivable. In a tubular structure with a fixed diameter an internal lumen surrounded by longitudinal muscle could be opened and closed just by relaxing and contracting longitudinal muscle, and thus by decreasing and increasing the thickness of this muscle coat.

In reality, I think that all muscle, the striated and smooth circular and the longitudinal smooth muscle contribute in an intimate cooperation to closure function. Presumably some of these muscles vary in activity, which could explain the observed fluctuation in urethral closure pressure. Further, this muscular cooperation allows local length and force changes and still provides a reliable long-term closure function. This variable local activity could have a favorable influence on muscle perfusion.

Thus, for proper sphincter function, longitudinal muscle within the "filler" volume is a most valuable contributing factor to urethral closure pressure as well as ensuring long-term three-dimensional stability. This stability is most easy to achieve for an arc-shaped pressure profile.

Tissue perfusion, blood pressure, and closure pressure

When a sphincter closes with a pressure higher than the tissue perfusion pressure, it will be difficult to perfuse the enclosed tissue optimally. However, do we know the critical perfusion pressure for all components of the urethral sphincter? It seems that the lesson from artificial sphincter cuffs is that a value above 50–60 cmH_2O permanent compression may be critical for urethral perfusion. Definitely, any sphincteric pressure above local blood pressure will be bad for tissue perfusion. It is not blood pressure which controls or changes the closure pressure but the closure pressure which will determine how much blood can flow into what region of the sphincter. Therefore, I do not think that the blood volume present, e.g. in the lamina propria, has any additional and independent sealing effect. The dominant condition for blood flow is as for other fluids: it will flow from a higher to a lower pressure. Thus, urethral pressure will control the blood volume present in addition to the regulation by vascular constriction and only indirectly, i.e. passively, the blood volume can contribute to closure function.

Occasionally cardiac pulsations can be observed in urethral pressure profile recordings, particularly in the central high-pressure zone. This has been interpreted in many ways, e.g. indicating that there are more vessels with a rich blood supply in the high-pressure zone of these patients. It has even been argued that the good blood supply provides the basis for the high pressure. However, the causal relations are different: it requires a minimum compression to detect such pulsation, as is the case during blood pressure measurement. This compression is only present in the central zone of a high-pressure profile, and not at low pressure. This does not justify the conclusion that there are fewer blood vessels in a low profile. It is just that under low-pressure conditions the pulsation will not be picked up. It may be that there are indeed less blood vessels in a weak sphincter and that this is associated with deterioration of the muscle tissue. But this cannot be measured urodynamically by simple UPP recording.

THE ABDOMINAL PRESSURE SPACE

The anatomy of the lower urinary tract and the urogenital diaphragm is complex and seems to show considerable variability (17, 18, 25, 26) and is not understood in detail. I will not try to compete here with any of the various models of lower urinary tract

function and pathophysiological concepts of stress incontinence.

Important is the functional aspect which can be investigated urodynamically, and that is better described by the term "abdominal pressure space" than by an anatomic decription. The work of, for example, DeLancey and co-workers illustrates how the combination of urodynamics measurements and anatomical findings, clinical observations and imaging, with bio-mechanical modeling can be exploited in a meaningful way (27–30). Obviously our current models are not complete and I am not convinced that they are correct in detail, and I disagree often in more or less relevant details. It seems to me there exists some kind of vicious circle between urodynamics measurements, signal interpretation, and pathophysiological modeling. From poorly controlled signals, data is abstracted in an uncritical fashion, which is then used to construct models, and these same models are used to interpret the signals. I will limit my discussion here to the application of essentially the same simple mechanical tools used before, which are the strict application of correct definitions of urethral and abdominal pressure, the equilibrium of forces, and the incompressibility of tissues.

This may sound very simplistic but considering the published material it seems useful. However sophisticated the pathophysiology models may be, they cannot be valid when they contradict basic physical laws and established mechanical concepts.

In many publications and even in textbooks one finds schematic drawings where the pathophysiology of stress incontinence is illustrated using arrows to show in which direction a specific pressure is acting, and accordingly the pathophysiology is discussed in terms of directional pressures used synonymously with forces (31, 32). Similarly in discussion with surgeons it is quite common to hear, for example, that the abdominal pressure—coming from the anterior—is pushing the bladder neck downwards and backwards (27, 32). It seems to me quite clear that these "pressure arrows" actually illustrate the observed or presumed direction of movement and deformation, and an arrow is indeed a perfect way of illustrating movement, but misleading for pressure. The observation of significant movement under stress indicates that indeed the stable equilibrium of the anatomy at rest is disturbed by the increase in abdominal pressure. But a definite causal relation between proven pressure difference and movement or deformation must be substantiated for every single aspect of movement and deformation.

The analysis of mobility in a space such as the lower abdomen is another good example of differences in a primarily anatomical or mechanical view. I prefer here the term "abdominal pressure space" which can be investigated and defined urodynamically, and I would like to show that simple mechanical considerations could lead to a better anatomical view. Independent of the method of imaging used the anatomical view will focus on the identification of specific structures and organs and try to follow their movements and changes (25, 33). A mechanical view will at first focus on some physical/technical questions as to the resolution of the images in space and in time. Obviously our observations are mostly limited to two-dimensional images which make it difficult to reconstruct the three-dimensional space and its changes in detail. The dynamic resolution in time seems to me rather unclear. I am not aware of any investigation of the true dynamic resolution of standard imaging techniques used in urology and of the question whether they are fast enough to follow all movements to its full extend, or whether there are any movements faster than those which can be observed with typical equipment and the naked eye.

While an anatomical view will focus on defined structures and organs, a mechanical view will take into consideration that this space is filled with incompressible tissues. This means essentially that any deformation of structures and organs anywhere in the abdominal space will have an effect on neighboring tissues, so that there is no independent free movement. This complication seems to be often avoided in schematic drawings by showing structures and organs surrounded by quite a bit of apparently empty areas in the abdominal space (32–35). This makes it easy to illustrate any form of mobility and deformation of structures and organs without bothering about neighboring tissues.

Imagine the urogenital border zone as a completely stable inelastic lower border plain. Further there are no open cavities or other significant compressible areas within the abdominal space. Completely incompressible tissues enclosed within fixed border plains will not allow any deformation and/or mobility to occur. Thus all questions about how and where organs and structures of the urinary tract are attached or connected are only relevant when mobility and deformation occur, i.e. when the pelvic floor is an elastic border plain with some significant mobility. This makes the discussion of any causal relation difficult, e.g. is a specific structure mobile because its fixation is weakened or broken, or is it just mobile because some other neighboring structure has become mobile, and the weakening or breaking of its fixation is secondary (35, 36)? Most surgical interventions aim at targeted modification of mobility, e.g. of the bladder neck. It seems that successful outcome is linked to specific modifications of mobility. However, this does not necessarily mean that a "physiological" support is re-

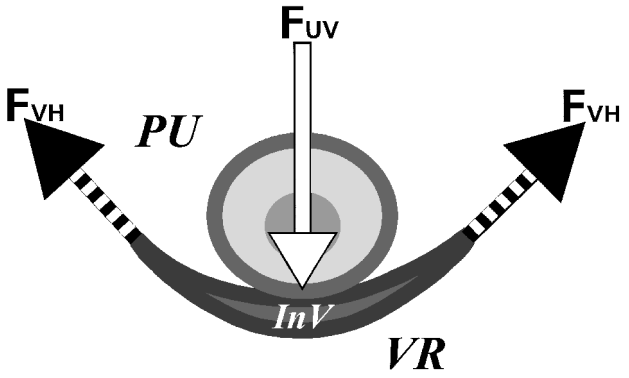


Fig. 7. The concept of a vaginal hammock has to meet a number of mechanical criteria. The force supporting the urethra FUV should be in balance with the forces in the hammock, FVH. This, however, leads to the conclusion that as a consequence of such a hammock we should find a pressure difference between the pubo-urethral space (PU) and the intravaginal (InV) and/or the vagino-rectal space (VR).

established by surgery (18, 32, 35, 37). This is different when the improved support originates from the pelvic floor (38, 39). The intra-operative observation of specific structural damages is interesting but needs more control in matching continent persons (30).

Vaginal hammock

I would like to discuss further the application of such simple mechanical reasoning as a plausibility control to a popular pathophysiological concept: the support of urethra and bladder neck by a vaginal wall hammock (27, 28, 35). Again only using concepts and parameters which can be investigated and measured urodynamically. One obvious key features of a hammock is to carry a load so that a pressure will exist between load and hammock but not below the hammock, or at least the pressure between load and hammock will be higher than the pressure below the hammock. If the pressure below this hammock would be the same as above, then obviously this cannot be a supporting hammock. This raises some questions about the simple mechanical consequences (Fig. 7):

1. Is it realistic to assume that the urethro-vaginal pressure on this hammock above is higher than the pressure below this hammock? The urethro-vaginal pressure cannot be higher than the pubo-urethral pressure, which is the abdominal pressure as is known from perivesical measurements via suprapubic puncture.
2. If only the anterior vaginal wall would form this hammock, then the intra-vaginal pressure should be lower than the abdominal pressure. This would affect the posterior vaginal wall which would have to carry the difference between the abdominal pressure existing in and around the rectum and the lower intra-vaginal pressure. I do not see any mechanism or structure which could provide these posterior balancing function.
3. If the whole vagina and its attached connective tissue form a hammock for the urethra and bladder neck, then again we must expect a pressure difference across this hammock and the question remains: how much lower is the pressure below the hammock, or how can pressure difference between the rectum and vagina exist posteriorly?

This discussion may serve as an example for the frequent confusion of an observed movement illustrated with an arrow and interpreted as a pressure. I do not think we have proof that a pressure difference across a vaginal hammock exists. It seems to me that a vaginal hammock, if it exists at all, may only become effective when the pelvic floor is weak, so that a vaginal hammock may be not a physiological mechanism to maintain continence but rather an indicator of a pathological change in other structures. However, careful discussion of mechanical effects of supportive structures and the investigation of intraabdominal pressure differences will help to better understand the pathophysiology. My discussion here is realistic for slow pressure changes where we know the exact speed of movements and the extent of deformation which can occur. But I ignore here the very fast dynamic changes with high acceleration, which make the situation much more complicated, particularly as I am not sure that we really know the maximum speed and extent of movement.

Pressure transmission

"Pressure transmission" is an intriguing and apparently simple concept which dominates most pathophysiological thinking about urinary stress incontinence (18, 37, 40, 41). However, it seems to me that there are a number of problems and it is mechanically not a simple concept. First we have to clarify terminology. Pressure transmission sounds like a pressure is generated somewhere and transmitted to somewhere else. This is rather unlikely. An abdominal pressure according to the idealized definition of ICS standards can only be generated in a confined space, i.e. "the abdominal pressure space", with enclosing borders which will bear the wall tension balancing this pressure. This pressure rise will be the same everywhere within these borders, except for a hydrostatic pressure component. The mechanics will be more difficult at coughing or sneezing with very fast pressure increase together with significant deformation and acceleration of organs and tissues. The term pressure "transmission" is misleading as pressure is not transmitted but builds up synchro-

nously in all tissues within the abdominal space. The real (anatomical and) mechanical problems arise from clear identification of the borders of this abdominal space, particularly where urethra, vagina, and rectum penetrate this border plain. The terms "pelvic floor", "urogenital diaphragm", "perineal membrane" all seem to describe a clear border plain. However, the use of multiple terms already indicates that the situation is complex and that there is no simple border plain. In the area of interest, a number of superimposing and intermingling structures and tissues balance the abdominal pressure and presumably make up an "urogenital border zone" of considerable thickness, so that actually a pressure gradient from abdominal pressure to zero surrounding pressure should be expected over some significant length, or rather thickness of the complex three-dimensional composite border structure. Any organ penetrating this border zone will be exposed to the full abdominal pressure inside and to no abdominal pressure outside (Fig. 8a). It is accepted that an abdominal pressure can be recorded in the ampulla recti and in the proximal urethra and vagina. But where is the mechanical border? All structures in this border zone should somehow show a pressure gradient where they cross this border zone. I think with careful urodynamic investigations we should be able to identify this border, at least under static conditions. Under stress it seems to be almost impossible considering the mobility and deformation of the pelvic floor in conflict with a defined local measurement.

These questions are important as it is very clear that during stress the intravesical pressure often increases to much higher values than the maximum resting urethral pressure. Thus if the urethral pressure does not increase under stress, incontinence would almost always occur. It is easy to see that in all parts of the urethra which are exposed to the same abdominal pressure increase as the bladder, the urethral pressure will increase synchronously with the intravesical pressure so that the pressure difference, i.e. the closure pressure under stress, will remain the same as under resting condition.

Further, quite sophisticated theories of active sphincter and/or pelvic floor contraction under stress are based mainly on urodynamic pressure recording somewhere in or near this border zone (5, 18, 42). It appears to be quite logical that levator ani and/or sphincters can be contracted in preparation for or during straining or coughing. However, many of the theories of active contraction during cough are based on intra-urethral pressure recordings showing "over 100% pressure transmission". So far I am not convinced that under stress the true local closure pressure increase has been quantified accurately beyond any reasonable doubt. Clearly any transducer moving within this border zone will record pressure differences

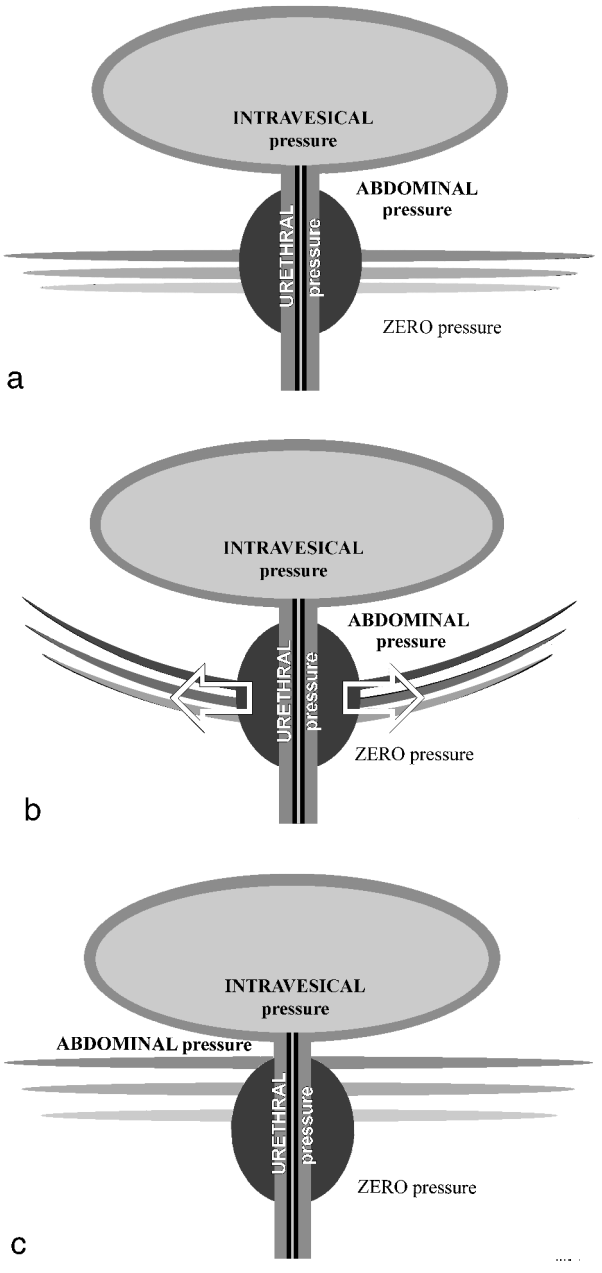


Fig. 8. The urethra has to cross the border zone which separates the abdominal pressure space from the surrounding (a). Imagine the urethral closure system were a mechanical part of this border zone. Any increase in abdominal pressure will have to be balanced by increased wall tension in this border zone and may lead to stretch and distension of this zone, which would counteract sphincteric closure function (b). If the urethral closure system is not a mechanical part of this border zone and can move within this zone, then any descent will reduce the proximal urethral length which is exposed to abdominal pressure, so that the urethral closure pressure (UCP) will be the reduced in the sphincter (c).

exactly according to the pressure gradient on the profile and its direction and extent of movement along this profile. Here I think it requires additional efforts to show that a signal with apparently over 100% "pressure

transmission", which can also be logically explained as a movement artifact or transducer/wall interaction, does indeed show fast active muscle contraction.

Mechanical combination of pelvic floor and sphincter

Increasing abdominal pressure means increasing abdominal wall tension and thus also increasing tension in the pelvic floor. This can mean deformation in many ways, including descent and distension of the urogenital diaphragm. Inevitably this also means that any structure which is mechanically a part of this "border plain" will come under the same increase of stretch and tension. Consequently, any urethral segment which is mechanically a part of this "wall", such as the urethral sphincter penetrating this wall, would come under a tension possibly directed radially with respect to the urethral axis and thus counteracting the closure forces and trying to pull the urethra open (Fig. 8b). The same is true for the vagina. The abdominal pressure increase would support the closure inside the abdominal pressure space, but still the increase in wall tension could weaken closure in the border zone. Anatomically, urethral sphincter and surrounding pelvic floor appear to be well-separated and different structures. Mechanically, however, the question remains, how are they connected, because some form of continuity must exist between these neighboring structures? If the urethra could move within the urogenital border zone, then any part descending downwards through this border zone under increasing abdominal pressure would no longer be exposed to the same abdominal pressure. If this happens with the high-pressure sphincter zone, then here the closure pressure would be diminished or may even become negative (Fig. 8c).

As stated above, the pelvic floor is not just a simple two-dimensional border wall between the abdominal and the outside pressure. In addition, there is no simple geometrical configuration where the urethra, vagina and rectum penetrate the urogenital border zone. It is not clear how they penetrate a mechanical border zone without becoming mechanically a part of it. It is, however, obvious that with increasing distension and stretch of this border zone the closure function of all penetrating structures is endangered. This could well be protected by appropriate structures within this border zone, e.g. sling-like structures which with descent and higher tension could act to increase closure pressure on penetrating lumen passively. The effective function of such support structure would have to be proven by precise urodynamic measurements.

I hope this discussion makes clear that a more careful and realistic mechanical view on this urogenital border zone provides useful additional criteria for the interpretation of the anatomical findings and a more careful and critical analysis of how urethra and vagina penetrate this zone. Further I think we should consider measuring directly the mechanical changes of these structures in terms of differential movement, acceleration, and deformation.

Mechanics of slings

Most surgical methods try to restore continence and support the urethra by some type of sub-urethral sling. This is a very logical approach, but the real effects and limits of such slings seem often not to be properly considered. Some surgeons have used slings to bring the urethra to a high intra-abdominal position. The idea behind this is that the defect is the loss of "pressure transmission", and that such slings would bring the urethra back to a location with proper pressure transmission. Further, it is a common belief that such a sling, if it could pull strongly enough, would also increase urethral closure pressure. However, the mechanical analysis of a sling around the urethra shows that a sling can only apply a force from one side and we know that this will not increase closure pressure (Figs 2 and 7). If pulled hard enough a sling can deform and kink the urethra. This already indicates that the balance to the force applied by the sling to the urethra will come from longitudinal tension in the urethra wall. Only if the sling could pull the urethra against some opposing tissue (where and what could this be?) could the urethra become compressed. Obviously then, kinking and compression will both result in urethral obstruction (43). Thus within suitable physiological limits a sling can only stabilize urethral position and prevent excessive urethral descent (10, 11).

According to the mechanical definition of stress incontinence used above, it is clear that the urethra does not necessarily have to be elevated from its resting position where it is continent without stress. The aim is only to prevent the negative impact of stress, such as further descent and deformation. This can be achieved with minimal tension in this sling. It seems logical that the full effect of such a support is best achieved when not only the bladder neck but also the mid-urethra is supported, as only then sufficient urethral length can benefit from this additional suspension. Only if a lack of exposure to abdominal pressure is the course of stress incontinence, it will be necessary to either "elevate" the urethra into the "abdominal pressure space", or to modify the mechanics of the border plain below, i.e. shift or modify the border plain of the "abdominal pressure space". This "abdominal pressure space", however, is not any anatomical definition but just a mechanical concept of an idealized abdominal pressure according to ICS definition (4).

It is now well accepted that many forms of suspension surgery are not effective over longer

periods, particularly the simple needle suspension type of surgery. Obviously the suspension loses its effect, presumably because it yields or breaks. Many different approaches have been made to correct for this. I do not think that anterior anchoring is a problem so that bone anchors can hardly offer a solution. It is interesting to note that the relationship between the forces in a sling is very sensitive to the actual shape. The forces are critically dependent on the angle of the V-shaped sling. With widening of this angle to a flat shape the forces will increase exponentially. The same is valid for any other sling-like supporting structure such as a hammock. This can be easily demonstrated with the equilibrium of forces (see Fig. 2). I think, however, the key problem will be related to the rigid inelastic nature of the artificial suspension. Such an inelastic suspension in a physiologically highly elastic structure must inevitably lead to failure. This failure should be expected at the weaker, more elastic, more mobile site which is the vaginal part. Here every sudden pressure increase, such as a cough or sneeze, will act on the soft tissue comparable with a lash with a whip. Thus not stronger but more elastic suspension is needed for providing long-lasting suspension in soft, elastic and mobile tissue. The local sling/tissue interaction will depend on forces and on the area of interaction, i.e. on the local tissue pressure. The potential danger of using very thin material, such as a mesh-like sling, is that the thin fibers over the years may cut through the tissue even at small forces.

SUMMARY

I have discussed here some aspects of the biomechanics of urethral closure function in a most basic, simplistic way. They are much more sophisticated models in the literature (44). My problem with these models, however, is that I think they may be misleading or at least there is not sufficient evidence that they are more realistic than my most simple approach. Just to take one example: Duan et al. (44) have investigated the age effects on the differential contributions of the venous plexus and smooth and striated muscle layers on the maximum urethral closure pressure and pressure transmission. Part of this model is quite similar to my simple sphincter. But here quite sophisticated calculations are developed from this model and it is concluded that a reduction in venous plexus and smooth muscle elasticity are important factors in the decrease of maximum urethral closure pressure with ageing. Without speculating about any details some simplistic plausibility control leads to question such as: what is elasticity in passive incompressible tissue and what is pressure transmission under static conditions? There are still a number of steps to make before I would accept such complex models.

I would rather like to summarize some results of my simple mechanical approach. Most of this may sound very theoretical and partly rather speculative. However, it is my experience that communication between physicians, but also physiologists and anatomists on one side and physicists and engineers on the other, is difficult. Instead of attempting to give some "final" answers I discuss here a number of questions which always come to my mind when reading urodynamic publications on incontinence. I have tried to describe a different—a mechanical view in a way—that may be understood and even may become applicable for others involved in the clinical urodynamic investigation as well as the scientific conceptualization of lower urinary tract continence function. I have tried to outline clearly the potential and limitation of some easily available mechanical tools, which I think are simple but very powerful. And finally I do think we have to start new from a very basic level if we want to develop a sound and comprehensive urodynamic methodology for application in research and clinic.

Only when we measure clearly defined parameter, such "urethral pressure", will we be able to perform signal quality control as described in GUP. We first have to clarify all physical, technical and mechanical aspects of our parameters before we can start a physiological interpretation. We should test all functional interpretations of anatomical findings or any other physiological investigations for agreement with the fundamental principles of physics and mechanics. Even very simple concepts such as the incompressibility of tissues and the equilibrium of forces allow wide-ranging theoretical conclusions on a microscopic and macroscopic level, which deserve careful experimental investigations. But I am convinced that such mechanical considerations are underutilized in urodynamics, and that they should be used to guide anatomical/physiological/clinical/surgical investigations and can serve as reliable and objective controls:

1. *Sphincter function:* The consequences of different arrangements of more or less complete circular muscle fibers for sphincter function; the need for a central filler volume; the effects of sphincter diameter and amount of filler volume; the relationship between sphincter size and closure pressure; the combination of circular and longitudinal muscle for optimal closure function; the interaction/coordination in space and time between the different muscles; the contribution of the lamina propria and blood volume.
2. *Abdominal pressure space:* The confusion of "pressure arrows" and direction of movement for the

conceptualization of the pathophysiology of incontinence; the inadequate resolution in space and time of observation and interpretation of organ movement and deformation; the difficulty of abstracting causal relations from observed movements and deformations; the fact that there is no independent movement in an incompressible space; the importance of understanding the local equilibrium of forces in a three-dimensional space; the difficulty of identifying the contribution of different structures separately; the careful investigation for local pressure and pressure transmission differences; the problem of correct definition of "pressure transmission" and its precise measurement; the critical analysis of passive "pressure transmission" and distinction from active contractions; the detailed investigation of the anatomy and mechanical properties of the urogenital border zone.

3. *Pelvic floor, sphincter, and slings:* The anatomical and mechanical integration of the urethral sphincter into the urogenital border zone; the impact of pelvic floor descent and distension on closure function; the influence of sphincter mobility within the urogenital border zone; the equilibrium of forces between urethra and a sling; the mechanical influence of a sling and the static and dynamic situation; the relationship between forces, sling geometry, and elasticity.

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Clinical View: What do we want/need to know about urethral closure function to improve diagnosis and treatment?

1. Introduction-
   - The two most commonly regarded clinical "urethral function tests" are the urethral pressure profile (UPP) and the Leak point pressure (LPP). The LPP does not directly measure urethral function. This section will focus mainly on the MUCP and only briefly review the LPP. The goal of current UPP technology is to measure the function of the urethral sphincter mechanism. When the measuring catheter (a microtip transducer catheter, a water perfusion catheter, or an air charged catheter) is slowly and constantly withdrawn through the urethra, a pressure profile of the sphincter mechanism is obtained. Various terms describe the parameters obtained with these measurements including: maximum urethral pressure (MUP), maximum urethral closure pressure (MUCP), functional urethral length (FUL), and pressure transmission ratio (PTR). Although all these parameters may be interesting from a research perspective, the only parameter that has been extensively advocated for clinical utility is the MUCP.
2. What do we want/need?
   - If continence means urethral resistance exceeds vesical pressure, commonly expressed mathematically as $p_{ura} > p_{ves}$, then theoretically the physiology of continence and incontinence should be easily measured and ascertained. It should be simple to measure these parameters and determine if someone is continent or incontinent. Yet we are still searching for a way to do these measurements and differentiate normals from abnormals, continent from incontinent. Current techniques like the UPP and, its main measure, the MUCP do not differentiate normals from abnormals- because we don't accurately measure the urethral pressure during times of incontinence?
3. We need a test that:
   - Clinically can be used to identify normals vs. abnormals during conditions of abnormality.
   - Provides information that influences clinical decision making and hopefully these changes in clinical decision improve outcomes
   - Provide prognostic (predictive) information for severity or treatment effect.
   - Is simple, easy, minimally invasive, and minimally uncomfortable to the subject.
4. What can go wrong?
   - *Tests performed or interpreted improperly*. (e.g. technology not really measuring what you want it to measure, using measures (cut-offs) from one technology to influence clinical decision making that may be very different in another technology, not understanding normal ranges or misunderstanding the data, the biology, or the physics
   - *Limitations of MUCP measures*. Position, posture, orientation of the sensor, size and rigidity of the catheter can all influence the urethral pressure profile results [1]. In a very thorough review of whether urethral pressure profile measurements are a useful diagnostic test for stress urinary incontinence, Weber [2] reviews criteria for useful diagnostic tests: 1) measurement methods must be standardized, 2) results must be reproducible, 3) calculated parameters should have clear cutoff values without significant overlap that differentiate health and disease, 4) calculated parameters should contribute to the differential diagnosis and choice of therapy, and 5) calculated parameters must correlate with the outcome of therapy for the disease. UPP measurements have difficulty with all these criteria.

5. Clinical Examples of utility and limitations of MUCP:

   - Comparative studies of conventional MUCP measures with different technologies (microtip catheter transducers, water perfusion catheters, air-charged balloon catheters); values differ depending on the technique.
   - ISD- what is it? Real or manufactured? Don't all women with SUI have ISD?
   - MUCP cut-offs- do they stand up to scrutiny? Should a normally distributed variable be evaluated dichotomously? Do these measures provide information to change clinical decision making. The proponents of MUCP testing argue that this test is helpful for: 1) contributing to the differential diagnosis of stress incontinence subtypes - intrinsic sphincter deficiency or urethral hypermobility, 2) influencing the choice of therapy, and 3) providing prognostic information for outcome of therapy.
   - Clinical utility of MUCP measures- Burch and Sling studies- When this continuous variable has been used to group patients into categories (MUCP< 20 cm/H_20 and MUCP >20 cm H_20) most retrospective studies demonstrate that surgical success rates with a modified Burch procedure are lower in the low MUCP group. [3-5]. Failure rates ranged from 12-18% when the MUCP was greater than 20 cm H_2O, but increased to 33 – 54% when the MUCP was < 20 cm H_2O. The preponderance of data would suggest that patients with low MUCP have higher surgical failure rates than patients with high MUCP. The implication is that sling procedures should be done instead of Burch procedures when an MUCP < 20 cm H_2O is found. Other data contradict the implication that MUCP provides significant prognostic information and should influence the choice of therapy. Richardson et al. found respectable Burch failure rates of 15% in low MUCP patients [6]. Sand et al., who earlier reported the 54% Burch failure rates with low MUCP patients, found later in a short term prospective randomized study that by modifying the Burch technique to a more aggressive correction, he was able to reduce the failure rate to 5% [7]. This rate was not clinically or statistically different than the synthetic sling group. In a comparative study of low MUCP patients, Maher et al. [8] found failure rates of only 10% with the Burch procedure, compared to 29% with an autologous fascia sling procedure.
   - Clinical utility of MUCP measures in midurethral sling (MUS) studies - retropubic MUS studies, transobtorator MUS studies and comparative MUS studies. In an RCT of patients with MUP < 20 , or VLPP<60 subjects did much better with a retropubic than a transobturator MUS [9] . In the recent UITN Trial Of MidUrethral Slings (TOMUS) study, VLPP or MUCP did not have an interactive effect with treatment. In other words this variable did not influence success or failure in one type of sling more than another. [10]

6. Leak Point Pressure (LPP) measurements –

   - The amount of pressure producing urine leakage by a Valsalva or coughing maneuver is typically termed the leak point pressure. LPP proponents argue that this is the preferred measure of urethral function for stress incontinence, because it is a measurement during "stress". It was initially defined as an abdominal pressure measurement and McGuire et al [11] found correlation with the clinical grade of incontinence. He also found that abdominal leak point pressures less than 60 cm H_2O highly correlated with the videourodynamically defined Type III incontinence: "…a nonfunctional open "internal" sphincter and leakage not necessarily associated with rotational descent." Management was recommended based on these measurements:
   - "In female patients without genital prolapse, a low leak- point pressure of 65 or less indicates intrinsic sphincter dysfunction; a high leak –point pressure of 100 or more usually is associated with urethral hypermobility. Most patients in the

low-pressure group have Grade III, Type III stress urinary incontinence. Most patients in the high-pressure group have some degree of urethral hypermobility and leakage is associated with that abnormality. In the middle-pressure group are patients with features of both intrinsic sphincter deficiency and hypermobility. Patients with pure hypermobility can be treated with a suspension procedure, whereas those with pure intrinsic sphincter deficiency are treated better by a sling, an injectable agent, or an artificial sphincter. Patients with features of both conditions do well with slings, but suspensions procedures may be effective"[12].

- With such strong management recommendations based on these measurements, it is not surprising leak point pressures became a cornerstone of U.S. stress incontinence evaluations. Modifications in technique developed. Many investigators utilize the vesical pressure measurement since the bladder catheter is in a fluid medium and less susceptible to artifact vagaries, like a blunted response. Although most investigators define LPP as the lowest intravesical pressure required for leakage with Valsalva or cough, others report the LPP as the increase, or change from baseline vesical pressure at the time of leakage. The LPP determinations are typically performed during filling cystometry at various volumes, in various patient positions, and either directly visualized or indirectly imaged with fluoroscopy. MUCP and LPP correlate modestly with each other and both are limited, but comparable in predicting incontinence severity [13].
- *Limitations*. LPP measurements vary based on the baseline used for measurement, patient position, catheter size, bladder volume, the technique used to confirm loss, and whether a cough or valsalva was used to produce leakage[14]. This lack of standardization has been summarized [15], and makes study comparison impossible. Few reproducibility studies have been done with LPP, but it appears to be more reproducible than MUCP measurements [2, 14]. Unlike MUCP data, there is limited outcome data to support specific treatments based only on LPP measurements. Success rates with collagen injection do not correlate with preoperative LPP levels[16].

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Biomechanical Concepts of Stress Incontinence: Theories of Continence

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In spite of the large number of women affected by stress urinary incontinence (SUI) little is known about the mechanics associated with the maintenance of continence. Detailed knowledge of the biomechanics of continence and SUI could be utilized for development of novel treatments and improvements to current therapies. Several theories have been developed for this purpose. To date, the knowledge of the mechanics of female continence is limited to three conflicting theories, *The Pressure Transmission Theory* proposed by G. Enhorning which stresses the importance of effective transmission of abdominal pressure to the urethra in the maintenance of continence; *The Hammock Theory* proposed by J.O.L. Delancy which stresses the importance of the levator ani muscle in the maintenance of continence; and *The Integral Theory* proposed by P.E.P. Petros which stresses the importance of the pubourethral ligaments in the maintenance of continence (1-4; 6; 10-13). However, the mechanics behind these theories has never been biomechanically validated.

In theory, the mechanics underlying the above theories can be investigated through the use of a complex dynamic finite element model of the lower urinary tract and pelvic floor. However, there are several modeling challenges that must be overcome before it is possible to begin construction of such a model. Therefore, in lieu of a quantitative biomechanical model testing the different theories of continence, this workshop will provide a biomechanical assessment and comparison of the models.

The Pressure Transmission Theory

The Pressure Transmission Theory focuses on the location of the proximal urethra in relation to the abdominal cavity (6; 8; 9). Enhorning theorized that, since, in healthy women, the bulk of the proximal urethra lies within the abdominal cavity, passive transmission of abdominal pressure to the urethra during such events as coughs, sneezes or strains was critical to compressing the urethra which in turn would allow continence to be maintained during these types of events (6; 8; 9). This theory further proposed that in women with SUI, the bulk of the urethra descended outside of the abdominal cavity, resulting in inefficient transmission of abdominal pressure to the urethra and causing vesical pressure to exceed urethral pressure,

leading to urine leakage. This theory gave rise to the idea that in order to treat SUI, the proximal urethra should be repositioned in such a manner so that the bulk of the proximal urethra once again lay within the abdominal cavity, enabling effective transmission of abdominal pressures and restoration of continence (6; 8; 9).

While slings & suspensions have been successfully implemented to treat SUI, their effectiveness does not depend on moving the urethra to a more abdominal location. In addition, imaging studies of continent women demonstrate a variety of urethral locations with regard to the bladder and abdominal cavity. Therefore, the Pressure Transmission Theory has not been validated by clinical studies and is not utilized as the theoretical basis for development of new treatments.

The Integral Theory

Petros and Ulmsten proposed that the vagina could be considered as three distinct segments: the anterior segment, the horizontal segment (superlevator vagina) and the zone of critical elasticity, with the anterior segment consisting of the distal two-thirds of the vagina (10-12). They refer to this segment as the *hammock* portion of the vagina since it is tightly connected to the distal portion of the urethra by a layer of dense connective tissue and propose that the urethra is supported in this region by the vagina in much the same way a body is supported in a hammock (10-12). The vagina itself is supported by the anterior and intermediate pubourethral ligaments which are anchored into the pubic bone. The insertion point of these ligaments, along with the insertion point of the anterior pubococcygeous muscle on the ventral wall of the vagina, mark the anterior border of the anterior segment and the distal border of the horizontal segment (10-12). From this border region, the horizontal segment of the vagina makes a 130º turn. The terminus of this segment is the insertion point of the uteral sacral and the cardinal ligaments, which provide support for this segment along with the levator plate which also supports the horizontal portion of the vagina (10-12).

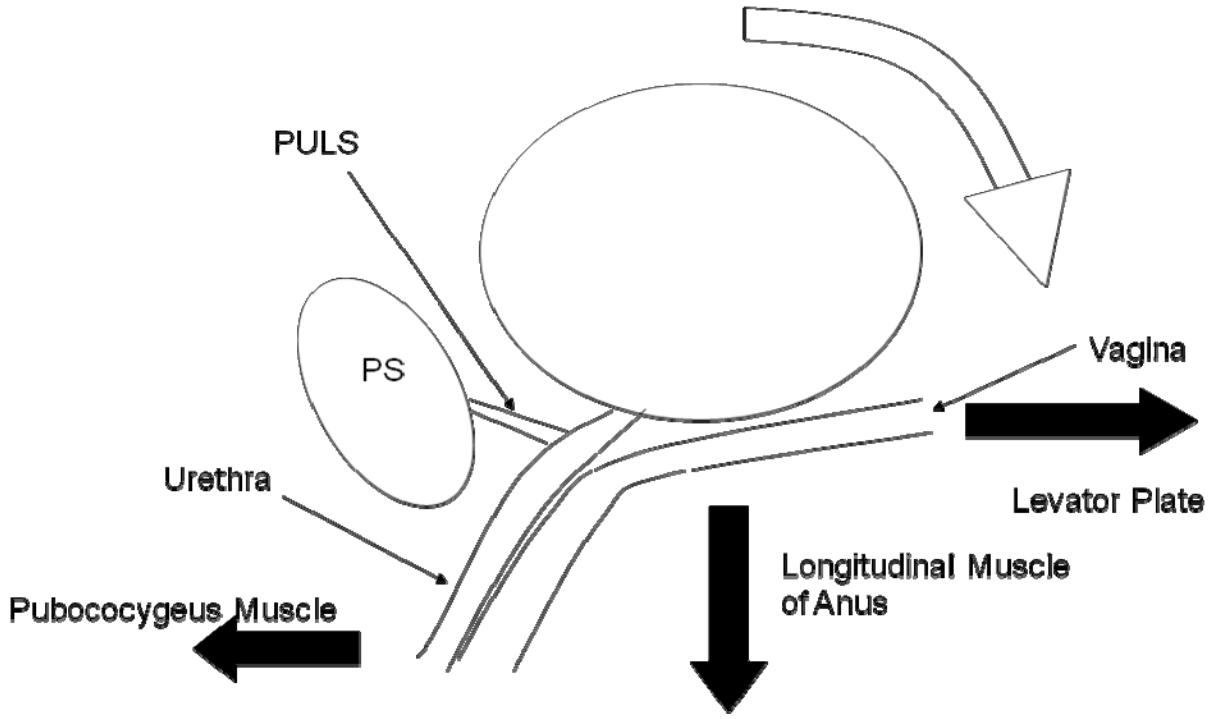

Figure 1. Forces (arrows) involved in continence, according to The Integral Theory, as adapted from PE Petros & UI Ulmsten, *Acta Obstet Gynecol Scand Suppl*. **153**:7, 1990.

The region where the superlevator vagina makes a 130º turn is termed the zone of critical elasticity by Petros and Ulmsten, who propose that this zone allows the vagina to act like a hinge and be pulled in two different directions (10-12). They theorize that this ability allows for three opposite movements to act on the vagina, enabling maintenance of continence (Fig. 1). The first mechanism of continence as proposed by the Integral Theory involves the contraction of the pubococcygeous muscle which exerts a tensile force on the anterior segment or hammock portion of the vagina, pulling the hammock-like vagina tight against the urethra.(10-12). The tightening of the vaginal hammock closes the gap between the cresta urethalis and the peiurethral striated muscle (urethral sphincter). Petros and Ulmsten reason that by closing this gap and tensioning the vagina the insertion points of the periurethral sphincter muscle are immobilized, allowing for the isometric contraction of this muscle (10-12). They propose that contraction of the pubococcygeus muscle is primarily responsible for closure of the urethra and that the periurethral sphincter muscle acts only to seal the urethra making it water tight, and lacks the force necessary to close the urethra by itself.

According to this theory, if the vagina is lax, pubococcygeus muscle contraction will not sufficiently tension the vagina enough to compress the urethra and provide continence (10-12). In addition, in the case of a lax vagina, the insertions of the periurethral sphincter will not be sufficiently immobilized to allow for the sealing isometric contraction of the sphincter to take place (10-12). Thus, a lax vagina could be the cause of SUI, according to the Integral Theory.

While the first mechanism of continence causes closure of the distal urethra, the second mechanism of continence simultaneously causes closure of the bladder neck. According to the Integral Theory, contraction of the anterior pubococcygeus muscle pulls the crescent shaped anterior wall of the vagina tight against the posterior urethral wall, immobilizing the urethra in this region (10-12). Petros and Ulmsten speculate that the pubourethral ligament, in this case, functions as a passive anchoring point which functions as a fulcrum against which the pubococygeous muscle contracts. At the same time that the pubococcygeous muscle is pulling the ventral wall of the vagina forward, the levator plate contracts, pulling the horizontal or superlevator segment of the vagina dorsally, using the pubourethral ligament as an anchor (10-12). The horizontal or superlevator vagina is then stretched dorsally in the direction of the levator plate contraction and the contraction of the levator plate muscle in the opposite direction causes the lateral portion of the pubococcygeous muscle to become semi-rigid due to the opposing forces acting on it (Fig. 2). Once the lateral portion of the pubococcygeous muscle becomes semi-rigid, the longitudinal muscle of the anus contracts, causing the pubococcygeous muscle to pulled downwards against the anchoring pubourethral ligament and causing the vagina to bend like a hinge in the zone of critical elasticity (10-12). The result of this bending in turn causes the urethra and bladder base to be tugged in a downwards direction, kinking the urethra at

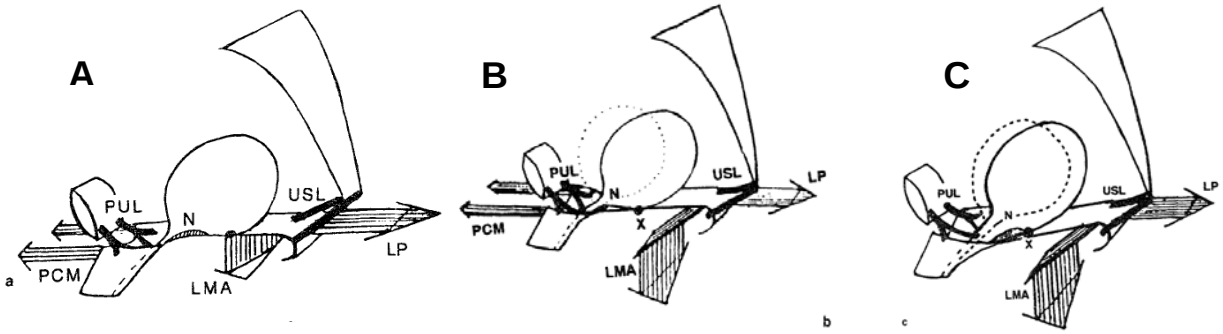

Figure 2. Integral Theory of Continence. **A.** First mechanism of continence; **B.** First and Second mechanisms of continence; **C.** Second Mechanism of continence. From: PEP Petros and U Ulmsten, Role of Pelvic Floor in Bladder Neck Opening and Closure II: Vagina, *Int Urogynecol J*, **8**:69-73, 1997.

the region where it is the least rigid.

Petros and Ulmsten theorize that in order for this mechanism to function properly, the pubourethral ligaments and the uterosacral ligaments must be intact to provide the proper support needed so that the forces of contraction function properly. In addition, isometric contraction of the external anal sphincter is also necessary to effect the contraction of the longitudinal muscle of the anus. Continuing with their idea that a lax vagina is the source of incontinence, they speculate that if the pubourethral and uterosacral ligaments do not adequately support the horizontal vagina, the forces produced by the contractions of the puboccygeous mucle, the levator plate and the longitudinal muscle of the anus will be dissipated, and as a result the bladder neck will not be tugged down sufficiently to create a kink in the urethra and resulting continence (10-12).

Both of the first two mechanism of continence are described as being under sympathetic control. The third mechanism, involving contraction of the three muscle groups associated with the puborectalis muscle, in contrast, is described as being voluntarily controlled (10-12). Petros and Umsten theorize that the contraction of one or more of these muscle groups pull the ventral and dorsal walls of the vagina and the rectum in an anterior direction, which simulates contraction of the puboccccygeous muscle and reflexively causes the first two continence mechanisms to activate.

According to the Integral Theory, SUI can be caused by laxity of the vagina, defects in the pubourethral ligament, laxity of the ureterosacral ligaments, or tears in the external anal sphincter (10-12). Damage to the pubourethral ligament is presumed to result in a reduction of restraint of the proximal urethra during contraction of the pubococcygeous muscle, causing funelling in the bladder neck instead of kinking in the urethra, when the levator plate contracts dorsally. If the uterosacral ligament is lax, the vagina would not be adequately supported and as a result, contraction of the longitudinal anal muscle will be dissipated and the bladder neck will not be pulled downward with sufficient force to pull it into position for closure of the bladder neck (10-12).

The Integral Theory cites inadequate support of the vagina as the primary cause of incontinence as without this support, the kink cannot form in the bladder neck (10-12). This support is

primarily dependent on the pubourethral ligament. The fact that this theory centers on the pubourethral ligament as the primary support structure responsible for continence is not without controversy, since it has been reported by other authors that these ligaments do not exist or that thin flimsy strands of smooth muscle tissue with none of the physical properties or mechanical strength associated with ligaments have been mistakenly named as ligaments (5; 7; 14). Nonetheless, the tension free vaginal tape (TVT) procedure developed based on the Integral Theory has proven to be a highly successful treatment for SUI (15). Since the TVT does not require a functional pubourethral ligament to restore continence, it is likely that only a portion of this theory is representative of physiology.

Hammock Theory

Similar to the Integral Theory, proposed by Petros and Ulmsten, the Hammock Theory, proposed by Delancey and Ashton-Miller, focuses on the supporting structures of the pelvic floor as the most important anatomical structures for continence (1-4; 13). The Hammock Theory deviates from the Integral Theory in 2 important aspects: the urethral sphincter (periurethral sphincter in the Integral Theory) plays a major role in the maintenance of continence and the pubourethral ligament does not (1-4; 13).

Delancey and Ashton-Miller speculate that during a hard cough the inferior abdominal contents are forced caudally due to increased abdominal pressure, basing their theory on ultrasound scans which show that the proximal urethra can be displaced up to 10 mm in a caudal-dorsal direction while the bladder neck is displaced in a caudal-ventral direction (1-4; 13). In response to this motion, it is thought that either the pelvic floor, the abdominal wall, or both must stretch to accommodate and arrest this motion. Delancey and Ashton-Miller hypothesize that this motion is arrested due to the inertial forces occurring as the pelvic floor is stretched. They speculate that these inertial forces cause a caudal cranial pressure gradient to occur in the abdominal organs. However, before this motion can be arrested, the proximal intra-abdominal portion of the urethra gets compressed against the support structures of the pelvic floor by the increased abdominal pressure. They further hypothesize that the abdominal pressure acts in a transverse manner on the urethra, such that the anterior wall is pressed against the posterior wall while the lateral walls are pressed against each other.

If this supporting structure is damaged by injuries such as those obtained during childbirth, Delancey and Miller suggest that the supportive layer becomes more compliant and the compression of the urethra by abdominal pressure is not as effective. The levator ani muscle is cited as the primary support structure responsible for allowing this compression to take place (1-4; 13). During a cough, the levator ani muscle contracts simultaneously with the diaphragm, contributing to elevation of abdominal pressure. This contraction also tenses the suburethral fascial layer, which in turns provides the support necessary to compress the urethra (1-4; 13). In short, under normal healthy circumstances, the urethra is supported by a hammock like structure consisting of the endopelvic fascia and the anterior wall of the vagina. When a cough occurs, the levator ani muscle contracts, causing a tensile load to be placed on both ends of the hammock which draws the structure taught. The rising abdominal pressure then forces the urethra into this now tight hammock compressing the anterior wall against the posterior wall supported by the hammock (Fig. 3). In addition, because pressure acts uniformly on a surface, the lateral walls are compressed towards one another. In women with SUI, due to injuries or aging, contraction of the levator ani muscle can no longer cause tightening of the hammock. As a result the support structure is not held as taught and the resulting compression of the urethra is not as effective, since the hammock deforms as a result of the compressive force, rather than resisting it.

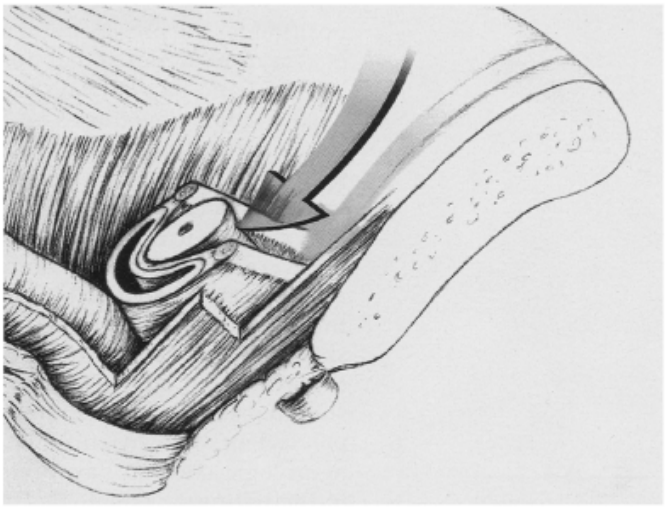

Fig. 3. Depiction of the Hammock Theory. From CM Sampselle and JOL DeLancey. Anatomy of Female Continence. *Journal of Wound Ostomy Continence Nursing* 25(2): 63-74. 1998.

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Some More Biomechanical Aspects of Continence Function: A Simple Model - Less Stress with Stress Urinary Incontinence

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The symptoms and signs of stress urinary incontinence, SUI, are rather clear and simple, and the success rate of surgical cure appears to be very high. But the diagnostic and therapeutical details are not based on profound understanding of the pathophysiological details of SUI. Much is based on speculation and experience from trial and error rather than the result of dedicated comprehensive research.

Traditional Concepts and Models of SUI

Traditionally, for many years now, the discussion of SUI circles around the more or less clear distinction of two possible main reasons for SUI: **"hypermobility",** i.e. weakness of bladder support, and **"intrinsic sphincter deficiency",** i.e**.** weakness of urethral sphincter. However both seem to be more speculative descriptive than anything which can be clearly identified and quantified.

While it is easy to accept that a weak sphincter cannot prevent leakage at abdominal pressure increase, this is more difficult to understand with hypermobility. In addition, mostly both entities occur in some combination, and both are difficult to measure. With our current catheter-based technologies any measurement of a closure pressure in a closed organ such as the urethra is notoriously unreliable. Most artifacts cannot be avoided and some are so common, so that e.g. **"directional pressure"** differences are accepted by some urodynamicists as being real and are interpreted as being pathophysiologically important, irrespective of the fact that by definition a pressure is a scalar and cannot have a direction. However, these artifacts are easy to identify, but they are difficult to correct.

Further, with our current imaging technologies it is difficult to evaluate the true mobility, as it seems that movement at cough and sneeze is definitely faster than what the naked eye

could track, faster than the time resolution of fluoroscopy and likely faster than what sonography could assess even by using fast imaging with slow motion replay.
In addition to intrinsic sphincter deficiency and hypermobility as two trivial components of SUI there are some more complex attempts of modeling such as e.g. the “integral theory” from Ulmsten and Petrou. Definitely this theory is much more impressive, but I think not more realistic. Anatomy seems to be taken into account with significant details, but basic requirements of physics and mechanics are generously ignored, such as e.g. the equilibrium of forces.

The Limits of Current Urodynamics.

So let us consider some basic facts which hardly can be debated. SUI occurs when the intravesical pressure due to increasing abdominal pressure exceeds the urethral pressure. Thus, my best urodynamic definition of stress incontinence is, that the bladder closure mechanism is continent = competent at rest, i.e. urethral pressure higher than intravesical, and becomes incontinent = incompetent under stress, here the mechanical stress occurring with abdominal pressure increase. Then the intravesical pressure increases and may exceed the urethral pressure, or even the urethral pressure may drop. We do not really know much about this stress in any details, as much as many anatomical details are debated. But we can still make some simple statements of general validity.
Good Urodynamic Practice, GUP, demands to reproduce symptoms under conditions of precise measurement. What does that mean for SUI?

Conclusion 1: Measure what we can i.e. p_{ves}/p_{abd} and p_{ura}, and indeed measurement of p_{ura} at rest shows the strength of bladder closure in form of a resting urethral pressure profile, UPP. But the contradiction to GUP is that a UPP without stress and thus without the symptom and sign of incontinence cannot give the desired information. Therefore, it is easy to understand that UPP at rest cannot be diagnostic, but it may be prognostic. Clearly, when the resting closure pressure is very low it is easy to overcome this closure function with little stress. This is difficult to repair with surgery, because surgery primarily interferes with the stress but does not increase a very low urethral closure pressure. Obviously surgery is effective in reducing this stress, but cannot completely abolish it, so when little stress already leads to leakage, then surgery cannot cure.

It is widely agreed that urethral pressure values of continent and incontinent are widely overlapping and that urethral pressure values do not change by/after successful surgery. Stress incontinence results from imbalance between closure function and load/stress; and operative therapy changes primarily the impact of load/stress on closure function, but not the closure function per se.

Conclusion 2: We should strictly follow the strategy of GUP and do measurements under load/stress, - when incontinence occurs, or actually we should measure both, stress and closure function, and ideally actually the impact of stress on bladder closure function. Now the problems start and the limitation of our urodynamic measurements becomes very obvious. We have no idea how to measure this stress, have problems of measuring the closure function, and have no concept of how to evaluate the impact of stress on the closure function. Even with little urethral and pelvic floor mobility it is impossible to keep the precise position of the catheter-based pressure transducer constant, so that we cannot determine the precise location in the urethra, and thus, correct for any dislocation. Because the UPP has strong pressure gradients on both sides, any dislocation will inevitably lead to a pressure change. We cannot tell how much urethral pressure change occurs at a specific location due to change in sphincter activity or impact of the surrounding pelvic floor, or how much of the recorded pressure change is actually only due to dislocation.

Conclusion 3: Measurement of pura under slow load/stress like at straining is difficult, under fast load/stress like during coughs and sneezing it is impossible. Such dislocation artifacts can be identified and qualitatively described, but cannot be quantitatively corrected.

A Simple New Approach

Let us build a simple model to understand the general nature of this inherent mechanical stress problem. To do this we have to be very simplistic in a first step, particularly with respect to the often unclear anatomical details. The bladder is inside the abdominal cavity and the urethra connects the bladder to the outside, penetrating this „ pelvic floor“, PF, which is treated here as a complex 3-dimensional muscular structure. It is well accepted that the sphincter surrounding the urethra and the PF are histomorphologically different,

but no doubt they must be „biomechanically“ connected, i.e. the sphincter is not freely mobile within the PF. Inside the abdomen we have the abdominal pressure and the PF provides the border ands thus also the mechanical balance to the zero reference pressure outside, i.e. a pressure gradient will exist over the PF. (Fig 1) With a strong PF the abdominal pressure can increase without any deformation and descent of the PF, i.e. no internal deformations, i.e. no movements of the bladder and sphincter will occur, because tissue is incompressible (o.k. gas isn't). Movement inside the abdominal cavity can only occur when the borders of the abdominal cavity will move. Thus the variety of „connecting“ structures such as the e.g. pubourethral ligaments, which often are ascribed an important role in providing continence function will only come under load, i.e. play a role, when the pelvic floor is weak and descends and deformations can occur.
When this is the case, then increase in abdominal pressure with deformation and descent of the PF will stretch the PF. (Fig 2) This will inevitably affect the surrounding of the sphincter, and thus lead to weakening of closure function. There can be no doubt that such stretch and distension of the PF will weaken sphincter function and can reduce urethral pressure. This is the critical load/stress which will lead to SUI.

What Can We Learn From Such a Simple Model?

This simple mechanical model also allows making some suggestions how this critical stress and deformation can be best reduced or even prevented. As the key is the weakness of the PF we have to provide additional mechanical support to the PF and from our simple model we can derive some b**iomechanical** criteria for optimal support and protection of sphincter function. (Fig 3)

1) The suspension should act at/under sphincter level. Definitely it should not support at the bladder neck. We have to support at the level of critical deformation to reduce downwards movement, i.e. under the PF.

2) The suspension should be tension free at rest because at rest no suspension is needed. The suspension should not cause any deformation or local pressure at rest because tissue will yield to any long term load. Only without a permanent tension it will provide long-term stability and support under stress. And only then we can avoid bladder outflow obstruction by urethral compression.

3) The suspension should be elastic, - to avoid high peak loads which are always destructive in soft tissue. Elasticity is most important for any long-term support in a highly elastic environment.

4) Such a suspension will not increase urethral closure pressure. This is not needed as the urethral closure is competent=continent at rest. We only have to prevent the impact of the stress, the mechanical stress on the sphincter. Any increase of urethral pressure by external compression would be obstructive.

Summary

This is a provocative attempt to stimulate critical thinking, the kind Gunnar Lose likes. We must face the reality of the many limitations of current practice of urodynamic measurements. Also we should avoid impressive complicated models which do not obey simple laws of physics and mechanics and are only impressive but not helpful. My simple model is not impressive but hopefully helpful in reorganizing some ideas and concepts.

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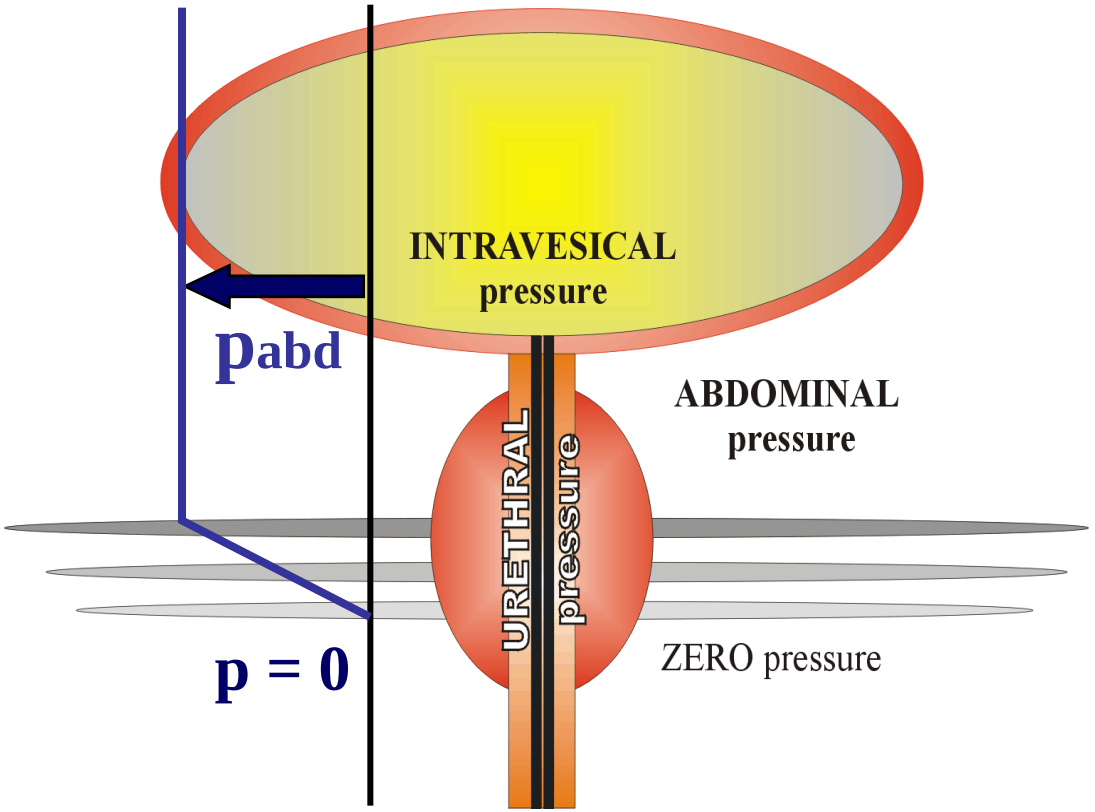
INTRAVESICAL
pressure
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pressure
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pressure
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Figure 1

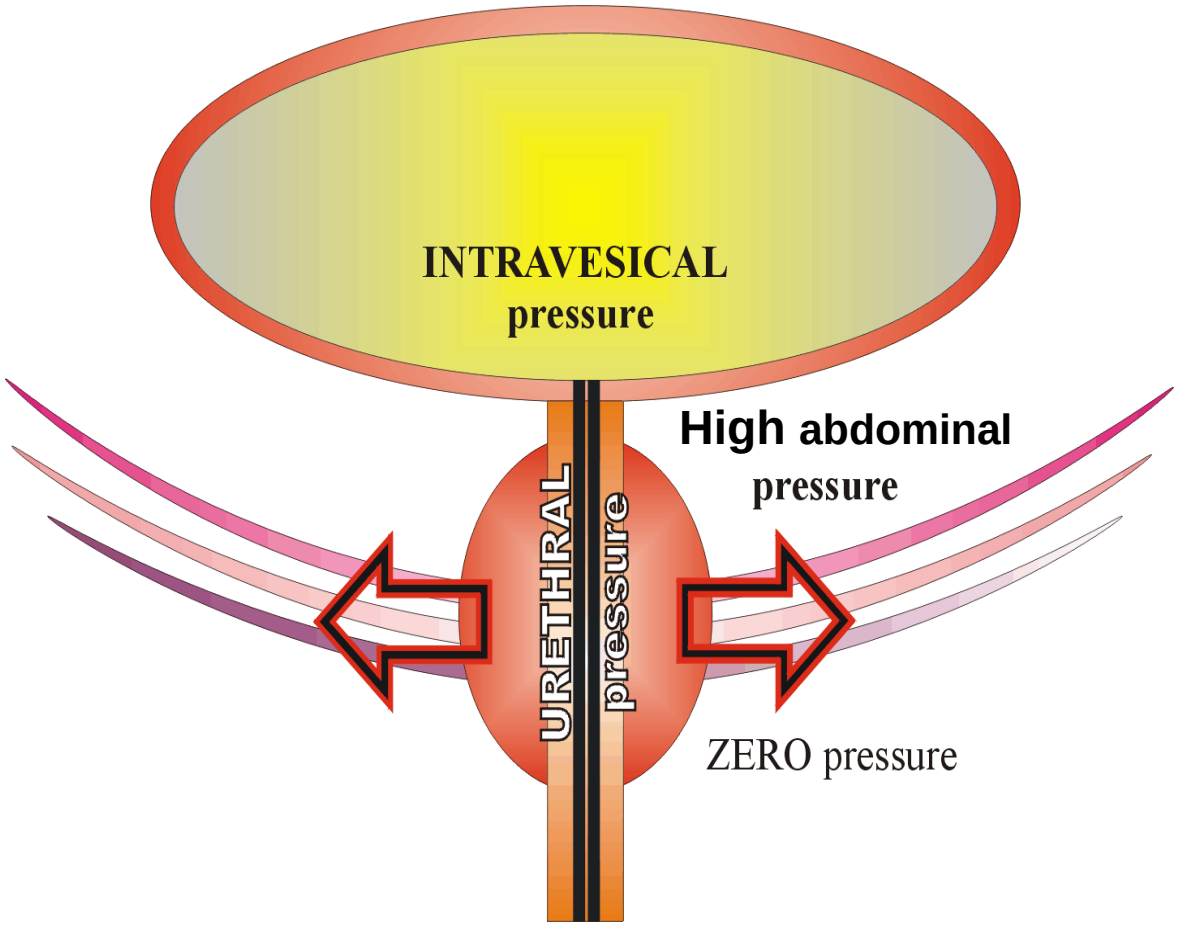
INTRAVESICAL
pressure
High abdominal
pressure
URETHRAL
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Figure 2

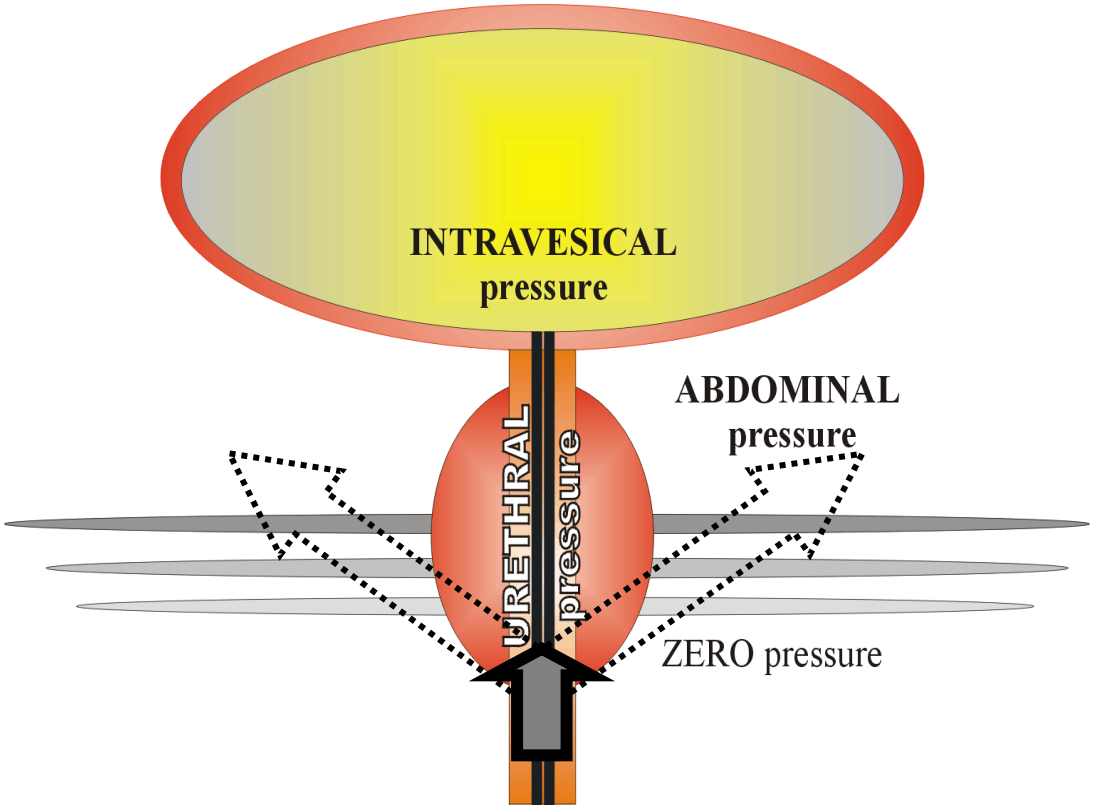
INTRAVESICAL
pressure
ABDOMINAL
pressure
URETHRAL
pressure
ZERO pressure

Figure 3

Pressure Measurement – The Details

MA Cooper, H Awada, PC Fletter, PJ Zaszczurynski, MS Damaser

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Introduction

Female urinary incontinence accounts for 12 billion dollars of health care treatment costs yearly in the United States (4). Incontinence is clinically assessed using urodynamic testing, including measurement of lower urinary tract pressures using catheter-based manometer systems (8). Urodynamic pressure data are comprised of a range of frequency components ranging as high as 15 Hz (7). Therefore, it is important to characterize how different catheter systems respond to a changing pressure input to determine if they adequately reproduce clinically relevant signals.

Four catheter-based manometer systems are presently used clinically. Water-filled catheters use water as a transduction medium; air-charged catheters use air as a transduction medium; fiber-optic and microtip transducers use electrical transduction mediums (10). Of these, only water-filled and air-charged catheters are disposable, one time use systems. The frequency response of water-filled pressure transducing catheters has been extensively characterized (1; 3). In contrast, air-charged catheters, a newer technology, have not been thoroughly characterized, but are gaining widespread clinical popularity and have an increasing market share.

Although clinical comparison studies between different transducing catheters have been performed (5; 10), simultaneous pressure measurements via two different systems is not feasible in a clinical setting. The International Consultation on Incontinence Committee on Dynamic

Testing recently reported that, while “air-charged catheters may provide an acceptable alternative to other techniques for measuring the pressure closing the female urethra, there have been no studies to show whether these catheters provide an acceptable alternative to fluid-filled lines . . . in urodynamics” (6).

To better understand the advantages and disadvantages of the newer air-charged technology, we subjected an air-charged catheter system and a water-filled catheter system to simultaneous pressure signals, including a transient step test and a frequency sweep, to characterize and compare the frequency response of each system. In addition, we evaluated benchtop re-creations of common clinical practices that can cause a loss of quality in the data: hydrostatic pressure changes and motion artifacts.

Water-filled catheter systems have been well characterized in previous experiments and have been demonstrated to function as second-order underdamped systems (1; 9), as we also observed. Shapiro and Krovetz reported that catheter length is inversely related to the frequency response of a water-filled catheter system and that with increasing catheter inner diameter there is an increase in the damped natural frequency of the catheter system (9). The addition of an air bubble to a water-filled system increases compliance of the system, and therefore decreases the damped natural frequency (9).

We utilized similar tests to characterize and compare the newer technology of air-charged catheters using catheter lengths and diameters typical of those used clinically for urodynamics. Water is an incompressible medium and therefore transmits a pressure wave without attenuating it. However, water can resonate a tube at some of the frequencies tested, amplifying the peak pressure and explaining why water-filled systems react to pressure signals as second-order underdamped systems, amplifying lower frequencies and attenuating higher frequencies. Air, in

contrast, is a compressible medium and attenuates more rapidly changing pressure waves while transmitting them. Therefore, air-charged systems attenuate even low frequencies, acting as an overdamped system similar to an analog low-pass filter.

If urodynamic signals occur mostly below 3 Hz, then the air-charged system could be beneficial since, as we demonstrated, most of the higher frequency noise is dampened by these catheters. Likewise, Rowan, et al. determined that it is beneficial to filter data during collection, by limiting the frequency range of the recording system, to remove high frequency noise that may occur (7). However, urodynamic signals can have frequency components greater than 3 Hz, particularly when utilizing rapidly changing signals, such as coughs (2).

We demonstrated significant differences in pressure transmission properties between water-filled and air-charged catheters. Knowledge about the response of each pressure transduction media ought to be considered when selecting equipment for a specific application.

Conflict of Interest Statement

Dr. Damaser acted as a consultant to T-Doc, LLC in 2007 & 2008.

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EDITORIAL COMMENT

It is not uncommon in today's urodynamics to investigate the clinical significance of a recorded signal without analysis of the underlying physical and physiological informational content of the recorded signal itself. Here it would be very unfair to blame the author for neglecting the basic scientific aspect of his measurements because he gives references to the literature. In addition, his conclusion that the clinical significance is poor with respect to stress incontinence but high when correlated with lack of anatomical support indicates that this study was done carefully. It is important to have such clinical data which show that the basic biomechanical objections against the quality of the recorded signal are correct. This result may help to direct attention towards analysing the factual informational content of the recorded signal before investigating artifactual clinical significance.

Because of the continuous discussion of urethral pressure profiles [e.g., Cadogan et al., 1988, with Editorial Comments by Constantinou and Hilton], I would like to discuss some comments at length and focus on the statement made in the introductory sentence of the Discussion: "Urethral pressure profiles performed with microtip transducers show directional differences which are *not* artefactual but are anatomically based."

This obviously expresses a general opinion of those believing in pressure profiles. I conclude that these directional differences must have some magical aspects, because they definitely do not have any serious scientific basis except that they are a rather accurately reproducible artifact, but not a closure pressure. This has been pointed out many times at a number of meetings and even in this journal [Plevnik et al., 1985; Schäfer, 1986; McNally et al., 1987]. It is not clear to me why all the serious limitations for meaningful *closure pressure* recordings in the urethra are completely ignored, or actually (which is even worse), why these limitations are mentioned, seemingly included, and respected in the discussion, but the necessary and important consequences are ignored. Maybe it is a question of unclear terminology.

WHAT IS AN ARTIFACT?

Terminology

If the attempt is made to measure a closure pressure—which, even for the urethra is a quite clearly defined physical parameter (and remember, pressure is a scalar, i.e., has no direction)—by an instrument designed and capable of measuring this pressure, and if then a signal is recorded which shows directional differences as reported, then this by definition cannot be a "closure pressure." This point cannot be open for discussion, because we cannot be more accurate than our terminology. (Alternatively one should define a nonphysical directional pressure which could be called "medical [magical] pressure".)

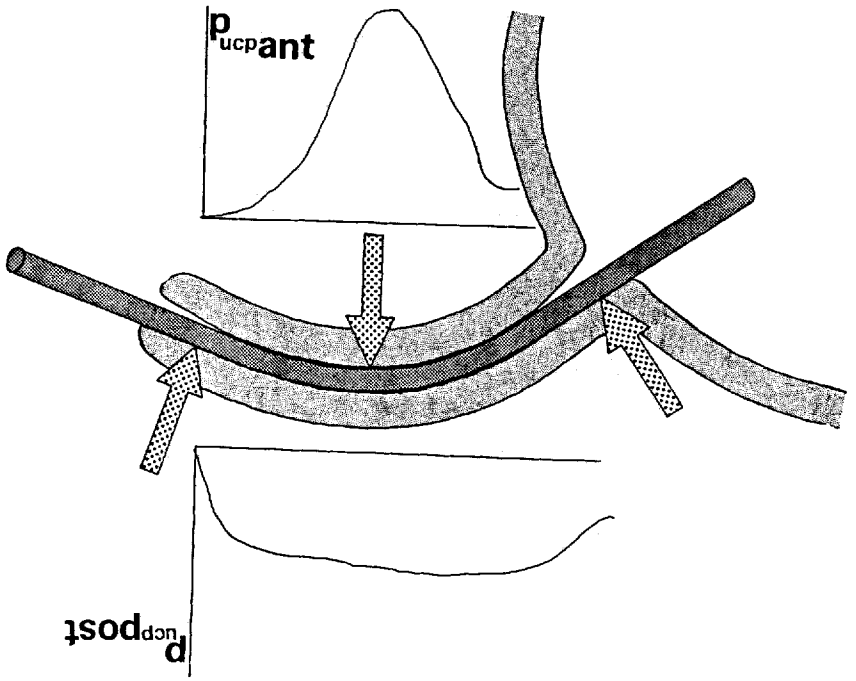

Fig. 1. Exaggerated schematic drawing of a stiff catheter in a curved urethra. The recorded directional signal results from superposition of urethral closure pressure and catheter bending forces (dotted arrows). Therefore the resultant urethral closure pressure profiles with anterior ($p_{ucp}ant$) and posterior ($p_{ucp}post$) orientation of the microtransducer show pronounced differences. It must be kept in mind that a minute force equivalent to the weight of 1 g on a surface of 1 mm^2 yields a pressure of 100 cmH_2O.

Physical Evidence

Obvious and rather simple theoretical considerations make it easy to explain, and it has been demonstrated convincingly from experiments, that this directional component of the signal is caused by catheter bending in the curved urethra; i.e., this recorded directional difference originates from direct and necessarily directional urethra/transducer interaction, because bending is a directional deformation which needs a force (force is a vector, i.e., has a direction) (Fig. 1).

Physiological Relevance

This signal recorded with a microtip transducer from the urethra, then, contains information about two entities: urethral closure and urethral shape, or better, deformation. Theoretically, it would be possible to separate these entities. But I do not think that urethral axial configuration is best determined by microtip transducers. The most important physiological consideration, however, which must be understood is the fact that *this directional component of the recorded signal does not exist in the urethra without the catheter* in place. Thus, here we do not record a genuine physiological parameter with an inevitable minimum disturbance (i.e., common artifact), as we do with suitable closure pressure recording technique (e.g., try to approximate with lateral transducer orientation of microtips). The directional com-

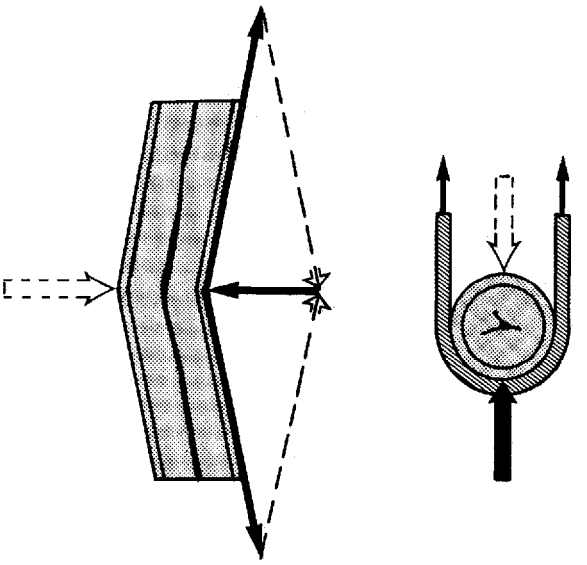

Fig. 2. A force such as that created by a sling acts radially on a tubelike structure. The balance of forces is shown schematically in a lateral view on the **left** and a cross-sectional view on the **right**. The sling force will lead to deformation and the direct balancing forces will be longitudinal (axial) forces in the wall. Strength and direction of forces (vectors) can be determined following the parallelogram law (left). This balance between axial and radial forces does not directly and effectively enhance closure pressure (except indirectly, e.g., by kinking). The sling needs an opposite (radial) balancing force (broken-line arrow) to compress the urethra effectively.

ponent is not a physical/physiological parameter of urethral closure function, but is only related to the catheter. The directional component is a genuine artifact from which we could try to abstract physical/physiological information. In theory we could investigate the relationship between catheter mechanics and urethral properties and deformations, and then we could standardize the catheter properties to the extent that we could draw conclusions from the directional component. But these conclusions would be very different from previous speculations.

In summary, this directional signal allows an ideal description of an artifact. If a recorded signal 1) is not what we try to measure (not a pressure); 2) is not clearly and definitely related to what we want to investigate (not to closure function but shape); 3a) only exists because of undefined properties of the catheter (stiffness, weight, shape, etc.); and 3b) only exists with the catheter in place during the recording (because of deformation, acceleration, etc.), then this I call a genuine measurement artifact.

While it may require some physics to understand that such directional differences in recorded urethral closure pressure values prove to be artifacts, it only needs common sense to understand that a urethra with a longer posterior than anterior functional (closure) length must be a magical structure, because against what should the "longer" part close? The recorded signal originates from the "closure" between tissue and catheter, again a simple artifact (see again Fig. 1).

To make this quite clear: these directional differences exist in the recordings, they are reproducible, they are anatomically based, but they are not what we want to

measure, and their anatomical basis is different from what the author seems to point out. The references to the anatomical studies of John DeLancey [1986] are misleading because these structures in the way they are discussed here and elsewhere [Constantinou, 1988] are contributing to the artifacts but not to the closure pressure. Physically, there are no structures just on one side of the urethra without having a balance on the other side which could exert a closure pressure on the urethra (Fig. 2).

The "experimental" proof is well known: all the effective surgical procedures for curing incontinence create a more-or-less slinglike supporting structure dorsally (posteriorly) around the urethra, i.e., act from one side onto the urethra. It is well known that none of these procedures enhances closure pressure (except in nonphysiological conditions when severely obstructing the urethral lumen).

Maybe it is helpful to illustrate the mechanics of closure by a very simple example (slightly modified from John DeLancey's presentation at the ICS, Oslo, 1988): have the end of a garden hose lying on your open hand (i.e., single-sided (directional) *force* on the hose from your hand below) in front of your stomach (of course with its open end directed towards you); ask somebody to turn the water on (slowly), and just try to close the hose by increasing the force with your hand from below. You will splash your face and learn that a single(-sided) force can change the direction of the stream, but it needs an opposite balancing force to compress and close the hose (see again Fig. 2).

(No, I do not think that this example is more lunatic than the urodynamic concepts of directional single-sided urethral closure pressure [i.e., "medical pressure"].) The true anatomical basis of the directional signal is shown clearly here by the data but is not presented as clearly in the Discussion in question. The key is the excellent correlation of the directional differences and other parameters, indicating loss of urethral vesical support. It is very easy to see that this loss of support ultimately enhances axial urethral deformation and bending, and thus, enhances the directional artifacts in the attempted recording of closure pressure.

We make measurements in urodynamics because we cannot see directly what is happening. Measurement means more than recording a signal, looking at it, speculating about the meaning, and believing in it. We must be much more critical about the techniques of measurement, and we must first understand the origin and (bio-)physical nature of the signal; then we can study the basic pathophysiological information content of the signal, and only thereafter can we learn about its clinical meaning and significance.

Nevertheless, the urodynamic literature is full of a colorful variety of urethral pressure profiles that have been produced and interpreted without much regard to the nature and quality of the recorded signals. The need for an accurate tool for analysis of urethral closure function, for reliable diagnosis of incontinence and guidance in therapy, does not justify a shortcut in the path from recording a signal to its clinical interpretation—bypassing basic research. This only leads to scientific short-circuits.

Benson comes to the clinical result which one would expect from basic mechanical considerations of the nature of the directional signal and, therefore, this paper is a valuable contribution. Another recent carefully done clinical paper [Cadogan et al., 1988] clearly shows other limitations of various modes of urethral pressure profiles. (My disagreements with the related Editorial Comments are obvious.)

For many years, instead of slowly and steadily building up a body of knowledge

about urethral urodynamics on sound fundamentals, we have been cutting down on the promises born out of wishful thinking based on shortcut (short-circuit) urodynamics. We will not be able even to approach fine tuning of profilometry before we agree on the fundamental informational content of the recorded signal. When seriously considering the basic problems with static urethral closure pressure profiles, it is actually amazing to observe that such recordings are made while patients are moving or even coughing, and then a specific, sensitive, and significant (bio-) physical/pathophysiological interpretation of the signals is offered in form of local directional "(medical) pressure" transmission. The only thing clear to me is that coughing provides new opportunities for exciting and almost perfect artifacts in

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ADDENDUM

The recent Special Issue, "Female Urinary Incontinence" (Vol. 7, No. 6), clearly shows the urgent need for a comprehensive discussion of the scientific basis, the physical and physiological meaning, and the clinical usefulness of urethral measurements. At least the last point seems to be quite clear, in so far as none of the clinical-surgical papers [McGuire, 1988; Siegel and Raz, 1988] even refers to urethral measurements. The research papers [DeLancey, 1988; Constantinou, 1988], in addition, provide detailed material in support of my comments.

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Urethral Sleeve Sensor: A non-withdrawal method to measure maximum urethral pressure during dynamic conditions[1].

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Introduction.

Whenever bladder pressure exceeds urethral pressure, urinary incontinence or bladder emptying occurs. Activities that increase intra-abdominal pressure (such as coughing, straining or exercising) result in a corresponding increase in bladder pressure and if that bladder pressure exceeds urethral pressure, stress urinary incontinence (SUI) results. Urethral pressure is typically measured as a profile using a small microtip transducer, a perfused side-hole, or a small air balloon on a semi-rigid catheter which is withdrawn through the length of the urethra with a puller device at a determined rate. These techniques collect pressure measurements at discrete sites along the urethra and a graph indicating the pressure at each of these sites along the length of the urethra is known as the urethral pressure profile (UPP) [2]. Only a small portion of the urethra can be measured at any given instant and these techniques often take 30 seconds or more to obtain and therefore do not allow reliable measurements of the maximum urethral pressure during Valsalva maneuvers or pelvic floor muscle contractions which cannot be maintained for that time period without fatigue. Some investigators place the catheter in a stationary position when maximum urethral pressure is reached and attempt to measure maximum urethral pressure during dynamic conditions such as Valsalva, cough or during a pelvic floor muscle contraction (PFMC). However, these dynamic activities often produce a slight migration of the catheter sensor away from the peak of the bell-shaped maximum urethral pressure zone and if the pressure decreases, it cannot be determined if this decrease is real or artifact.

Perfused sleeve sensor technology, commonly called the Dent sleeve, is a well accepted technique that has been used for more than 25 years measuring maximal pressures in gastro-intestinal sphincters such as the esophagus or anal canal [3-5]. The unique quality of this perfused sleeve is its ability to record maximum pressure measurements anywhere along the length of the sleeve without requiring withdrawal of the catheter. If the maximum pressure zone of a sphincter resides within the sleeve, the sleeve will record the maximum pressure of the sphincter. We could not find literature to suggest that Dent sleeve technology has been used in urodynamics.

We are reporting our evaluation of the urethral sleeve sensor (USS) for urethral pressure measurements. [1] The aims of this study are four-fold; 1) to determine if there are axial variations with sleeve sensor technology in the urethra, 2) to compare maximum urethral closure pressures (MUCP) using sleeve sensor technology with maximum urethral closure pressures obtained by water perfusion UPP, 3) to determine if sleeve sensor technology measures respond appropriately during dynamic conditions such as Valsalva or PFMC in normal and stress incontinent women, and 4) to determine patient discomfort with this technology compared to water perfusion UPP.

Methods.

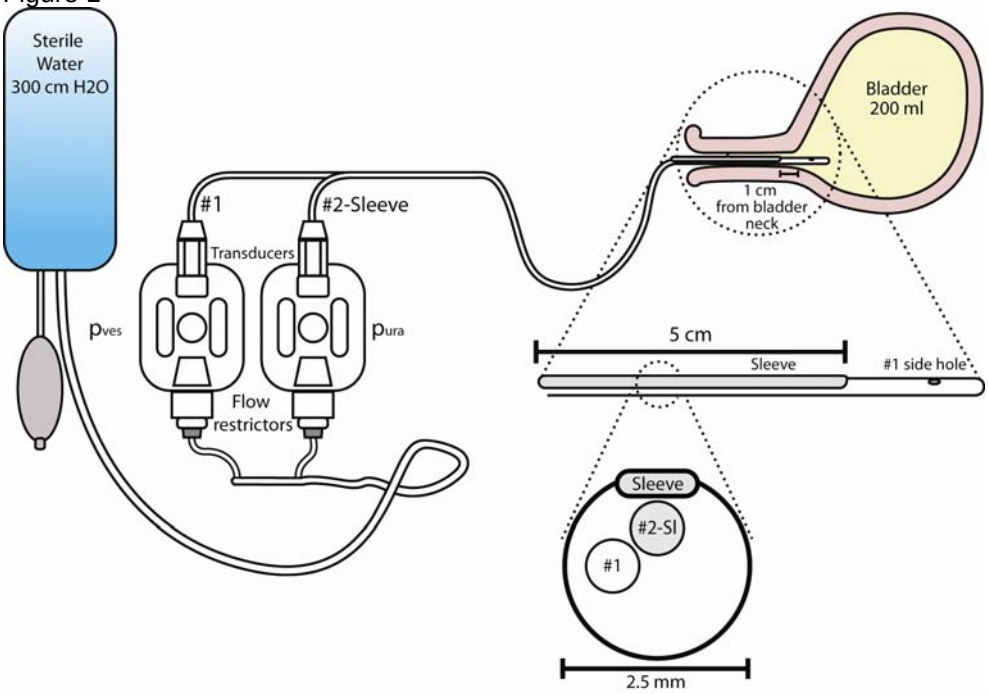
The study population consists of eighteen continent women volunteers and 7 women with stress dominant urinary incontinence scheduled for diagnostic urodynamic studies. The stress incontinent patients had demonstrable stress incontinence during office evaluation.

Subject evaluation

After voiding, all subjects had a Foley catheter inserted into the bladder and a post-void residual was obtained. The bladder was filled to 200 ml. Gentle traction was placed on the catheter to bring the balloon to the urethral-vesical junction and the urethral length was measured by marking the catheter at the level of the external meatus. The catheter was then removed and a stress test was performed with Valsalva and coughing, first in the supine position, and then standing if leakage was not observed supine.

All subjects then underwent assessments with the urethral sleeve sensor (USS) and conventional water perfusion urethral pressure profilometry (UPP). The order of the two procedures was randomized to eliminate bias attributable to tolerance or fatigue. Stress incontinent patients also underwent conventional filling cystometry and pressure-flow studies as part of their clinical evaluation.

Urethral Sleeve Sensor.
Figure 1 illustrates the sleeve catheter measuring system and the catheter positioning in the urethra and bladder. The sleeve sensor catheter is 2.5 mm in diameter (7.8 Fr) and made of flexible silicone and was specially manufactured for our purposes (MUI Scientific Inc., Mississauga, Ontario, Canada, www.dentsleeve.com). Reverse-perfused sleeves are recommended when distal catheter pressures (bladder) are likely to be higher than proximal measures (atmosphere) and a reversed-perfused sleeve was used in this study. At the distal end of the catheter, a perfused side hole measures intravesical pressure. Two centimeters proximal from the distal end, the sleeve sensor begins on the catheter and extends 5 cm in length proximally. Based on the urethral length measurements, the sleeve sensor is positioned in the urethra so that the sleeve's distal end is 1 cm from the urethral-vesical junction and the remainder of the sleeve clearly resides within the entire rest of the urethra including the midurethral high pressure zone. The sleeve sensor is connected to the external transducers and both the intravesical and intraurethral measuring systems were zeroed to atmospheric pressure with the catheter and transducers at the level of the patient's urethra. Room temperature sterile water in a pressurized bag at 300 cm H_2O was perfused through the system at 0.5 ml/min with a commercially available flow restrictor (Uniflow Flush Device, Edwards Lifesciences, Irvine, California, USA).
Figure 1



Pressure measurements were taken during 3 coughs, 3 Valsalva maneuvers and 3 pelvic floor muscle contractions with the sleeve oriented at 3, 6, 9 and 12 o'clock. The sequence of axial variation measurements was randomized. The catheter was secured in place during the various maneuvers and the urethral meatus was observed to determine leakage. For PFMC, subjects were asked to "contract their pelvic muscles as if trying to prevent urination or passing gas" and each subject was coached until they could do this maneuver properly. Intravesical pressure (p_{ves}) and urethral pressure (p_{ura}) tracings were collected continuously throughout the study and a third signal recorded urethral closure pressure (p_{clo}) by continuously subtracting p_{ves} from p_{ura}. The pressure readings were all recorded on Laborie urodynamic software (Laborie Medical Technologies, Williston, Vermont, USA).

Urethral Pressure Profilometry.
Water perfused UPP's were chosen for comparison to the sleeve catheter because the ICS has defined urethral pressure as the fluid pressure needed to just open a closed urethra [2]. For UPP measures, the same perfusion system that was used for the sleeve sensor measures was used with a 7 French Laborie triple lumen water perfusion catheter. Measurements were obtained with the catheter laterally oriented to the 9 o'clock position and withdrawn at 1 mm per second with a mechanical puller. Three separate measurements of p_{ves}, p_{ura}, and p_{clo} were obtained from three separate pulls with the patient at rest.

At the completion of their entire evaluation study, subjects were asked to complete a 0-100 mm VAS scoring their discomfort for the UPP and the USS urodynamic techniques.

Results.

Urethral Sleeve Sensor Signals
Because of frequency response time limitations with a water perfused system using a distant transducer, the urethral sleeve sensor technology does not accurately measure pressure from millisecond events such as a cough, although cough signals are observed in both the bladder and urethra measuring systems. Figure 2a is an example of a typical signal obtained from a continent subject. During Valsalva maneuvers there is minimal to no change from baseline for p_{clo}; p_{clo} remains positive and no leakage occurs. A properly performed PFMC produces an increase in p_{ura} with no increase in p_{ves}, and therefore an increase in p_{clo} above resting baseline (figures 2a and 2b). An improperly perfomed PFMC with a Valsalva contribution, produces an increase in p_{ura} and p_{ves}.

Figure 2a

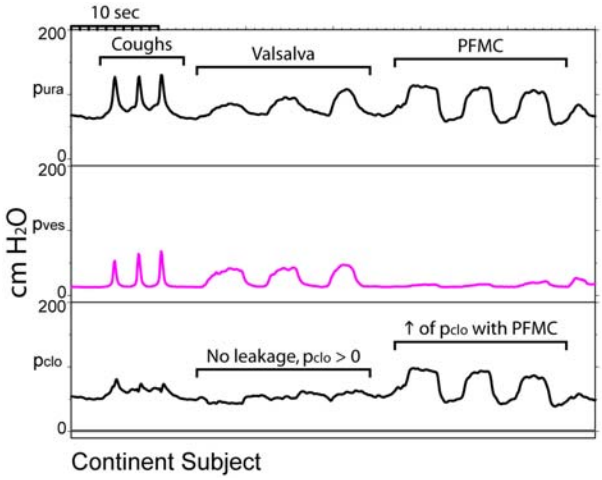


Figure 2b

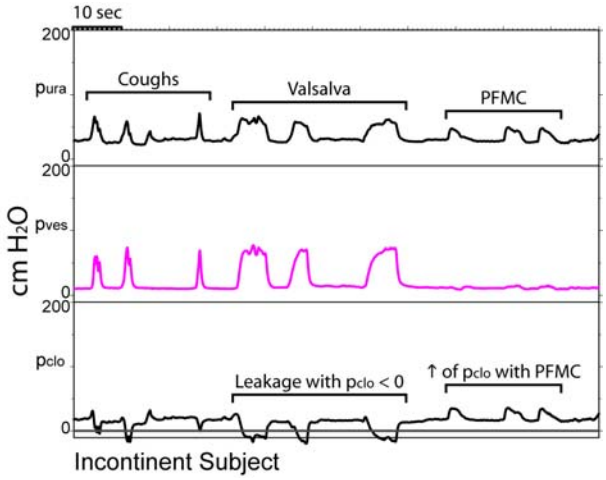
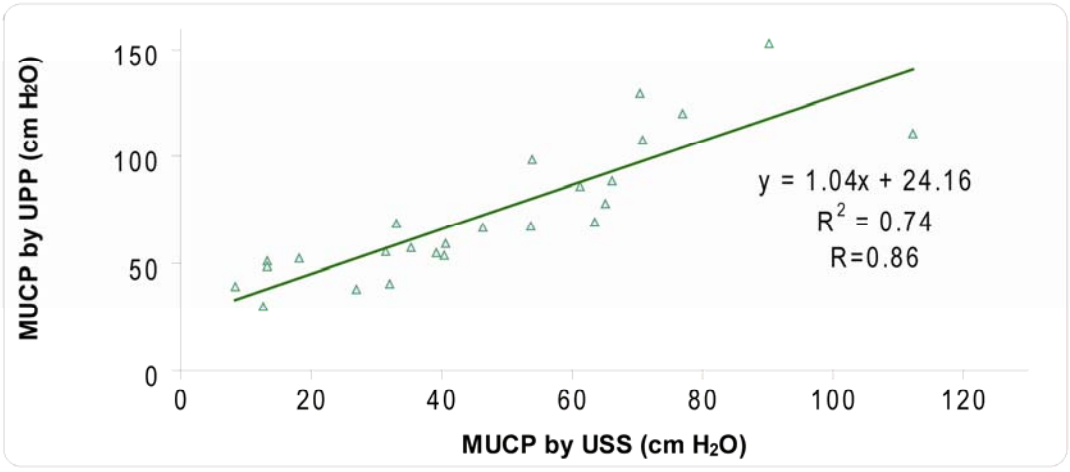


Figure 2b is an example of a typical signal from an incontinent subject. Valsalva maneuvers produce a bladder pressure that exceeds urethral pressure, p_{clo} becomes negative, and leakage is observed. During PFMC, the maximum total p_{clo} pressure is less than in the continent individual.

Laterality.
The 12 o'clock position produced different (higher) results than the 3, 6 and 9 o'clock results. Measurements were compared in a pair wise fashion. The mean differences between the other three positions was <7 cm H_2O with correlation coefficients >0.84 but the difference between the three other orientations and the 12 o'clock orientation were between 10-17 cm H_2O with correlation coefficients of <0.83. This 12 o'clock deviation in the correlation relative to the other orientations is sufficiently different enough to warrant exclusion of the 12 o'clock data from all further calculations. This exclusion of 12 o'clock data is also consistent with urodynamic literature on urethral laterality [6-9].

Comparison of Water Perfusion MUCP and Sleeve Sensor MUCP

Figure 3 is a scatterplot demonstrating the mean water perfusion MUCP on the y-axis and the mean urethral sleeve sensor MUCP on the x –axis for all 25 subjects. The correlation coefficient between these two methods of maximum urethral closure pressure measurement is high at 0.86 ($p<0.001$). The mean difference between the UPP and USS was 26.3 cm H_2O for all subjects. The 95% confident limits of agreement (-6.16 and 58.76) were based on the technique described by Bland and Altman [10].
Figure 3



Pelvic Floor Muscle Contraction.
Table I shows the mean p_{clo} pressures at baseline and with PFMC in continent and incontinent subject groups. Two continent subjects and one incontinent subject were unable to perform the PFMC despite verbal coaching and were excluded from this analysis. Continent subjects demonstrated significantly greater values of baseline p_{clo}, p_{clo} with PFMC, and change in pressure than incontinent subjects.

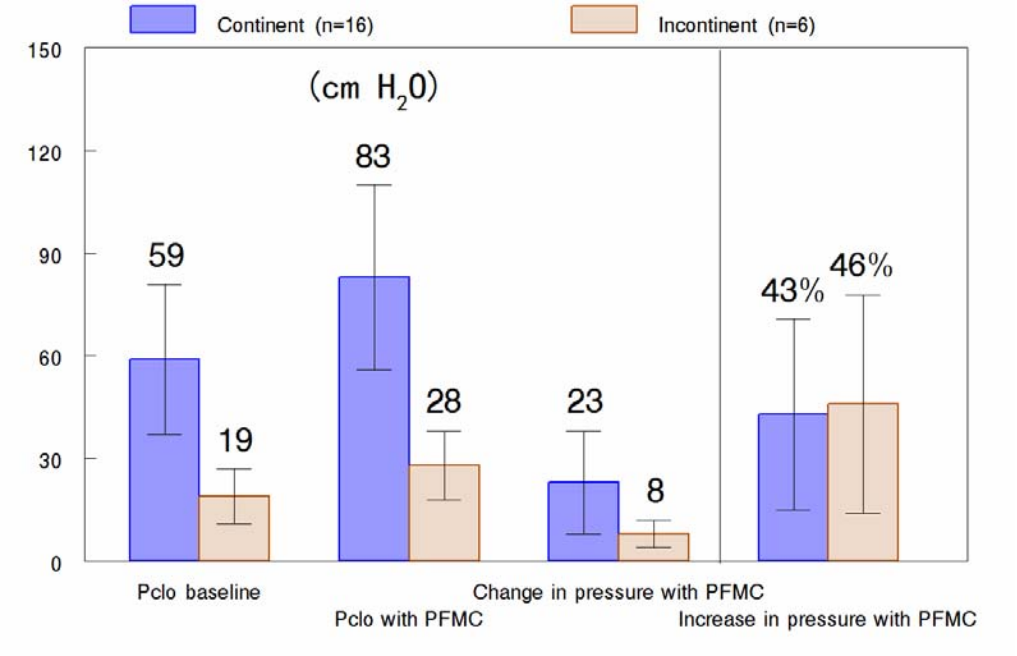
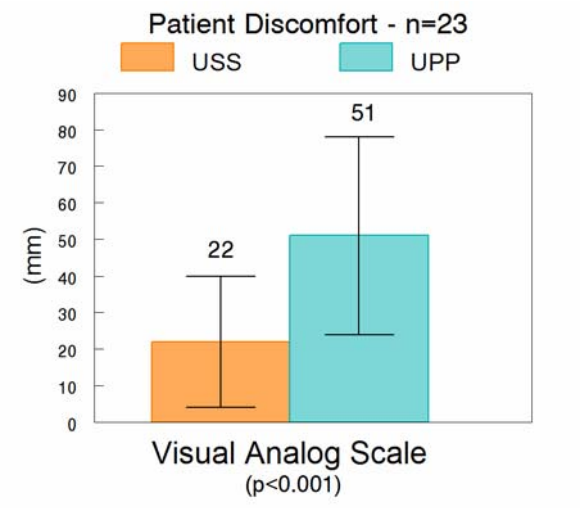


Table II demonstrates sleeve urodynamic findings of a urethral closure pressure declining to 0 cm H_2O during a Valsalva maneuver in the incontinent group. Note the high sensitivity and specificity of this urodynamic finding to clinical findings in each group.
Table II

	Clinically demonstrated leakage with valsalva.	
Sleeve urodynamic findings	Continent (no leakage with valsalva)	Incontinent (leakage with valsalva)
Valsalva MUCPuss>0	18	0
Valsalva MUCPuss<0	0	7

Subject Tolerance of Urodynamic Techniques.
Twenty-three of 25 subjects completed the VAS for discomfort. The discomfort score (mean +/- SD) for the USS (22 ± 18 mm) was significantly less than for the UPP technique (51 ± 27 mm) ($p<0.001$).



Discussion.

Our study aims were to evaluate a GI manometric technique for possible use in the urethral sphincter and our results are encouraging. We found that MUCP's measured by a sleeve sensor have excellent correlation ($r= 0.86$, $p<0.01$) with MUCP's measured with a conventional profilometry technique. This suggests that both methods measure the same biological phenomena: urethral pressure. The sleeve sensor allows accurate recording of maximal urethral pressure measures under dynamic conditions like Valsalva and pelvic floor muscle contraction when catheter migration with conventional systems can

produce false decreases in maximum pressure recordings. We have also shown that the USS has very good sensitivity and specificity for detecting incontinence based on MUCP<0 during Valsalva maneuver. This sleeve sensor does not require withdrawals and is better tolerated by patients than profilometry techniques. We also found that similar to other studies of urethral pressure, the urethral sleeve sensor is most consistent when the catheter is oriented to the 3, 6, or 9 o'clock position. Finally, this technique is well-tolerated; subjects report that the conventionally performed UPP using a puller was at least twice as uncomfortable as the sleeve sensor.

The sleeve sensor was specifically designed for the evaluation of dynamic sphincters within a biological system. Dent originally introduced the sleeve-catheter device for obtaining a continuous recording of lower esophageal sphincter pressures [5]. It was devised for measuring the serial change of maximal lower esophageal sphincter pressure without the need for catheter manipulation. The sleeve is a collapsible membrane that measures the maximal sphincter pressure at any point along the sleeve and the sensor is suitable for monitoring continuous pressures of biological sphincters [3, 5]. The sleeve has been validated in human models in the gastroenterology literature [3, 4]. It can be considered a Starling resistor. As long as any part of the sleeve remains within the urethral high pressure zone, then minor movements of the catheter do not effect measurement results and maximal pressure is measured. As a result, the sleeve catheter is not prone to slippage artifact commonly encountered with methods which measure a discrete point along the urethra, it does not require a puller, and it is able to measure a pressure over a prolonged amount of time.

Similar to other reports in the literature, we found that the accuracy of the urethral pressure readings deviated most when the sleeve catheter was placed in the 12 o'clock position. Several other studies evaluating axial variations of the UPP in continent and incontinent females also found that MUCP was always higher in the anterior direction (12 o'clock) [6-9]. A possible biological explanation for this phenomenon may be that the urethral walls at the 12 o'clock position are adjacent to the pubic bone which produces less elasticity of the urethral walls. We concur with the stated literature and recommend that the urethral sleeve sensor be oriented in the lateral position for all evaluations.

Although an excellent correlation between the water perfused UPP and the USS is demonstrated, pressures averaged 26 cm H_2O higher with the UPP. Water–perfused systems do seem to produce higher urethral pressures than microtip catheter systems. In a study by Wang and Chen the average pressure obtained from the double-lumen water perfusion catheter was 24.5 cm H_2O higher than that from the microtip catheter in all age groups [11]. Another likely reason for higher pressures during the UPP withdrawal technique compared to the sleeve sensor is because the urethra probably does not relax during withdrawal techniques; the withdrawal technique produces an involuntary reflex or voluntary contraction of the urethra. This phenomenom is well known in anal manometry literature; when a catheter is continuously withdrawn through the anal canal the moving catheter produces a reflex or voluntary contraction of the anal sphincter and the resting anal canal pressures are inaccurately high. For this reason, most GI manometrists use sleeve sensor or interrupted "station" measures taken at finite intervals for true anal resting pressures. We think the sleeve technology is the first technology to truly measure maximum urethral pressure along the length of the urethra at rest.

The sleeve sensor has potential for pelvic floor muscle training because it can provide direct urethral pressure measures during biofeedback. Conventional biofeedback equipment utilizes probes with surface EMG sensors or balloons in the vagina and rely on measuring vaginal pressure as a surrogate for urethral closure pressure [12]. Valsalva maneuvers can give false results [13]. With the sleeve sensor, if a patient improperly performs a Valsalva maneuver instead of a PFMC the p_{ves} pressures increases and a patient can be instructed that she is not contracting the correct muscles.

Limitations of the sleeve catheter (as with all water-perfused systems that utilize a distant transducer) are its inability to accurately measure millisecond events such as a cough, or sneeze, The sleeve sensor may not discriminate between continent and incontinent subjects if the patient only leaks with cough and not with Valsalva. A technical limitation is that the sleeve needs to be properly positioned so that the sleeve is always in the maximum urethral pressure zone, but not in the bladder, where it would measure bladder pressures rather than urethral pressure if bladder pressure were higher. Since maximum pressure is

usually at midurethra and the typical urethra is 4 cm in length, by positioning the upper end of the sleeve 1 cm from the urethra-vesical junction we are capturing the high pressure zone with our measures. Current studies are underway with a catheter that has an inflatable balloon 0.5 cm above the distal end of the sleeve to insure proper positioning of the sleeve with a single catheter insertion. We are currently using the sleeve catheter during filling cystometry and pressure flow studies. Preliminary observations confirm urethral relaxation during voiding and flow when p_{clo}=0.

Conclusions.

The urethral sleeve sensor measuring system is a technique used in GI manometry that is well suited for measuring maximum sphincter pressures. This study demonstrates that it can be used in the urethral sphincter to measure maximum urethral pressure without the limitations of catheter withdrawal systems. It correlates well with the current UPP methods and functions as expected in continent and incontinent subjects. The sleeve sensor has potential to allow direct biofeedback measures of the urethra during pelvic floor muscle contractions. Further studies are underway to evaluate its use during filling and emptying studies.

Acknowledgement.

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