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Dynamics of female pelvic floor function using urodynamics, ultrasound and Magnetic Resonance Imaging (MRI)

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ABSTRACT

In this review the diagnostic potential of evaluating female pelvic floor muscle (PFM) function using magnetic and ultrasound imaging in the context of urodynamic observations is considered in terms of determining the mechanisms of urinary continence. A new approach is used to consider the dynamics of PFM activity by introducing new parameters derived from imaging. Novel image-processing techniques are applied to illustrate the static anatomy and dynamics of PFM function of stress incontinent women pre- and post-operatively as compared to asymptomatic subjects. Function was evaluated from the dynamics of organ displacement produced during voluntary and reflex activation. Technical innovations include the use of ultrasound analysis for movement of structures during maneuvers that are associated with external stimuli. Enabling this approach is the development of criteria and fresh and unique parameters that define the kinematics of PFM function. Principal among these parameters, are displacement, velocity, acceleration and the trajectory of pelvic floor landmarks. To accomplish this objective, movement detection, including motion tracking algorithms and segmentation algorithms were developed to derive new parameters of trajectory, displacement, velocity and acceleration, and strain of pelvic structures during different maneuvers. Results highlight the importance of timing the movement and deformation to fast and stressful maneuvers, which are important for understanding the neuromuscular control and function of PFM. Furthermore, observations suggest that timing of responses is a significant factor separating the continent from the incontinent subjects.

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1. Introduction

Anatomically, the pelvic floor (PF) contains many visceral organs having diverse functions ranging from: urination, defecation, ejaculation, orgasm, conception, labor and parturition. These multiple organ systems interact and coordinate with each other in performing their normal physiological function. Under certain conditions, these functions are subject to disruption, manifesting as incontinence: sexual dysfunction, pain or any of a spectrum of complex symptoms whose origin may or may not be readily identifiable. In this context, pelvic floor dysfunction (PFD) constitutes a global burden affecting the quality of life of the individual, their family and society in general. Clearly within the general categorization of PFD, there is broad spectrum of symptoms having diverse origins. In women, because of the magnitude of the problem, a great deal of attention has been placed on the implication of PF function as it relates to urinary incontinence (UI) and stress urinary incontinence (SUI) involving the involuntary leakage of urine on coughing, sneezing, exertion or effort to the extent that it has been termed by DeLancey to be a hidden epidemic [1]. As a consequence, diagnostic methodologies under the umbrella of urodynamic testing evolved to address the epidemic. However, as with urodynamic testing there is an analogous endeavor to formalize and systematize the evaluation of the function of PFM. Evidently the technical requirements of PF dynamics technology are complex and challenging requiring a contextual approach and the least possible invasive means. In current practice, imaging and manual muscle testing per vagina or rectum is the technique used by most clinicians to evaluate the PF muscles. Unfortunately due to the location of the PF muscles defining its normal function in a non-invasive way is clinically and technically challenging.

2. Basic considerations

The PF is a complex 3D arrangement of muscle and connective tissue, attached to the bony pelvis. The PF is a collective name for the levator ani and ischiococcygeus. The levator ani muscle consists of the pubococcygeus, the puborectalis, and the iliococcygeus muscles. The pubococcygeus and the puborectalis muscles form a U-shape as they originate from the pubic bone on either side

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of the midline and pass behind the rectum to form a sling. The iliococcygeus muscle arises laterally from the arcus tendineus levator ani and forms a horizontal sheet that spans the opening in the posterior region of the pelvis, thereby providing a "shelf" upon which the pelvic organs rest [2]. The muscles and fascias of the pelvic diaphragm are inserted on the ischial spines either directly or indirectly through the sacrospinous ligament and the tendinous arch of the pelvic fascia. The result of a PFM contraction is a medial pull on the ischial spines to produce a more rigid and narrower pelvic floor [3].

Various diagnostic approaches have been applied to evaluate PFM function directly or indirectly and assess their dynamic properties, contractility and tissue quality and strength using palpation, visual observation, electromyography, dynamometers, ultrasound and MRI. Compared collectively, each tool has its own qualities and limitations [4]. Most recently, using a reliable instrumented speculum, incontinent women demonstrated lower values in passive force, endurance and speed of contraction than continent women, however, differences between the two groups for maximal force reached the statistically significant level only in the endurance parameter [5]. PFM strengthening exercises do diminish the symptoms of SUI [6,7]. Little research has focused upon the mechanisms of therapeutic change to help identify the specific critical muscle components of manipulation [8] so it is unknown whether PFM manipulation mimics the normal physiological behavior of the PFM or is an compensation strategy, nor whether program awareness is indeed the most efficient method of conservative rehabilitation. It seems appropriate to determine whether other properties of muscle function generated from imaging are also important in defining PFM function and dysfunction, as well as gaining a greater understanding of why PFM manipulation is effective in some cases and not in others. A useful approach to understand the mechanisms involved can be made by considering the relative effect of PFM activation on the urethra under involuntary reflex conditions as well as during volitional or anticipatory contractions. Fig. 1 illustrates the anatomical as well as functional changes taking place during PFM contraction.

As indicated by Fig. 1, voluntary PFM contraction elevates the bladder and acts upon the urethra U thereby generating an increase in closure pressures above the resting pressure. The increase in pressure is not uniform and depends on the position along the length of the urethra producing a pressure gradient and closure. Clinically while the increase in the closure pressures produced can be felt during pelvic examination through the vagina Fig. 1b shows that the urethra is also squeezed synchronously with the PFM contraction. Clearly active PFM contraction pressures are superimposed above the resting vaginal and urethral closure given that neither structure can be considered as passive. Distinction between the relative influence of vagina and urethral can only be made if vaginal closure is measured at rest as well as during PFM contraction. Enabled by a probe system [12], the closure forces at various locations in the vagina were measured. Using this probe, a resting vaginal pressure profile VPP was characterized and Fig. 2 illustrates the distribution of closure along the length of the vagina.

In measuring the vaginal pressures with a probe, it is appropriate to consider that there is mechanical deformation of the tissues involved and consequently values obtained in the anterior as compared to the posterior require adjustment [13]. Nonetheless the VPP has provided clinically useful information in delineating the distribution of forces along the length of the vagina in continent as well as in SUI subjects [14]. Indeed the VPP, as illustrated by Fig. 2, may be considered analogous to the urethral pressure profile UPP given that the urethra is cradled by the vaginal for a considerable region of its length.

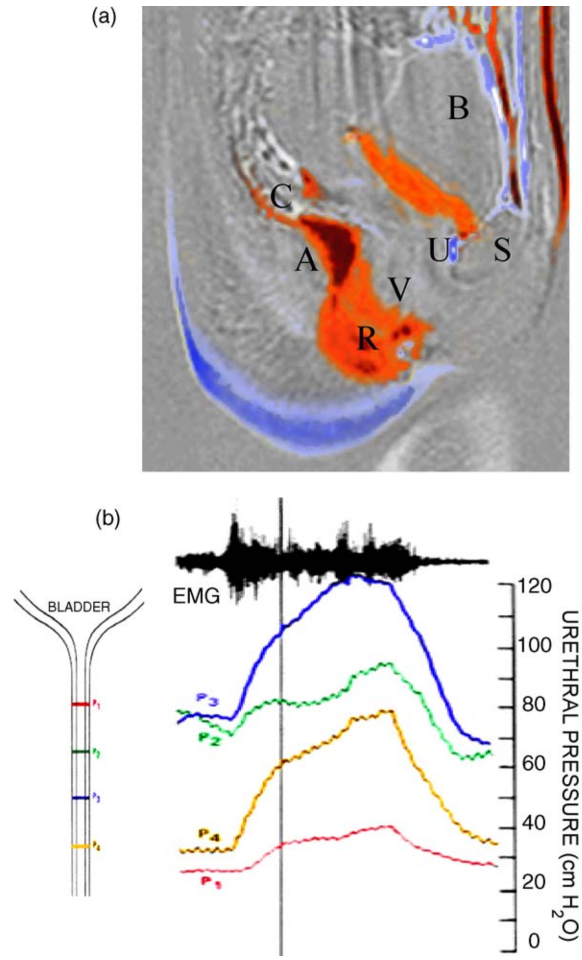

Fig. 1. Subtraction MRI image showing (a) the differences between resting and contracting pelvic floor muscles [9,10] where: A: anorectal junction, B: bladder, S: symphysis pubis, R: rectum, U: urethra and V: vagina. Red denotes region where contraction compresses while blue where it is vacated. Fig. (b) illustrates the changes in urethra pressures all measured simultaneously at different regions using the approach described in [11]. Urethral pressure rise U is shown by Fig. 1b to be generated over and above the resting pressures P1–P4. As shown by the schematic diagram associated with Fig. 1b P1 defines the pressure at the base of the bladder, having the lowest baseline pressure while P2 P3 and P4 are located 7 mm apart towards the urethral sphincter and meatus. Fig. 1b clearly shows that the maximum increase in urethral pressure from baseline can be localized at a region distal sphincter. The electromyogram shown at the top obtained with co-axial needle electrodes of the anal sphincter, is characteristic of a sustained voluntary contraction. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article).

3. Imaging/urodynamics studies

As demonstrated by Fig. 1, voluntary PFM contractions can be readily viewed using MRI primarily because the contraction can be sustained for a sufficiently long time, 10–15 s to acquire the image. Thus as indicated by imaging studies using ultrasound or MRI a voluntary contraction of the PFM changes the anorectal angle (ARA) [15] and can displace the urethra in a direction towards the pubic symphysis [11,16–19]. Yet PFM contraction in some women increase the intra-urethral pressure, but not in others [20]. It is known that in women there is recruitment of PFM motor units [21,22] and an increase in intra-urethral pressure [23] prior to an increase in intra-abdominal pressure during a stress. Two hypothetical questions then arise: Is the contractile force of the PFM as applied to the urethra diminished or the timing of urethra closure in SUI slow in responding? To clarify these questions, it is appropriate to consider the available evidence, in terms of clinical

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