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Review

Acquired urinary incontinence in the bitch: Update and perspectives from human medicine. Part 2: The urethral component, pathophysiology and medical treatment

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ABSTRACT

Various pathologies can affect the bladder and/or urethral contractility causing signs of urinary incontinence. In this second part of a three-part review, the pathophysiology of impaired urethral contractility (including urethral hyper- and hypotonicity) in the bitch and in women is discussed. Urethral sphincter mechanism incompetence (USMI) is the most common form of acquired urinary incontinence in bitches and is characterized by a decreased urethral tone. The pathophysiology and current recommended medical treatment options for USMI and cases of modified urethral tonicity due to a neurological disorder or functional outlet obstruction are discussed. Treatment options in human medicine in cases of impaired urethral contractility are described.

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Introduction

Acquired urinary incontinence is a common problem in dogs, particularly in spayed bitches (Holt, 1985a). This is the second part of a three-part review; in Part 1, we reviewed current knowledge on the pathophysiology and medical treatment for micturition disorders due to impaired bladder contractility in bitches and in women (Noël et al., 2010a). Here, the pathophysiology and medical treatment of micturition disorders due to modified urethral contractility is discussed.

Urethral sphincter mechanism incompetence (USMI) is the most common cause of urinary incontinence in the adult spayed bitch (Holt, 1985a; Krawiec, 1989), with an incidence ranging from 4.5% (Thrusfield, 1985) to 18–20% (Arnold et al., 1989). Neurogenic urethral dysfunction includes lower motor neuron (LMN) lesions associated with urethral hypotonicity and upper motor neuron (UMN) lesions associated with urethral hypertonicity. Detrusorurethral dyssynergia is due to partial suprasacral lesions which may result in inappropriate contraction or relaxation failure of the urethral muscle, associated with detrusor muscle contraction (Hosgood and Hedlund, 1993). Non-neurogenic functional outlet obstruction can develop after urethral urolithiasis and is due to an increased sympathetic stimulation of the urethral musculature

leading to an inappropriate ure thral hypertonicity (Lane, 2001; Fischer and Lane, 2007).

Pathophysiology

Urethral hypotonicity in women

Stress urinary incontinence (SUI) is the most common type of urinary incontinence in women and is characterized by involuntary urine leakage during effort, sneezing or coughing (Abrams et al., 2003). Urine leakage occurs when the intravesical pressure exceeds the maximal urethral closure pressure (MUCP) in the absence of involuntary detrusor contractions, implying a failure in the urethral closure capacity (Ortiz, 2004). This may be due to an 'intrinsic sphincter deficiency' related to alteration of the urethral wall components, or a 'hypermobile bladder neck or urethra' which may be displaced during a sudden increase in abdominal pressure due to weakness to the pelvic floor (Van der Vaart et al., 2002). The role of age, pregnancy, childbirth and obesity has been established in the development of SUI, but the role of hysterectomy and menopause remains controversial (Van der Vaart et al., 2002; Ortiz, 2004)

Urethral hypotonicity in bitches

USMI is a multifactorial condition associated with decreased urethral resistance. Urine leakage occurs when the intra-abdominal

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pressure rises, for example during recumbency or barking (Holt, 2008). Predisposing factors include urethral tone, bladder neck position, urethral length, neutering, body size, breed, docked tail, and obesity. The urethral tone is maintained by a complex interaction of neuromuscular, vascular and passive elastic components (Holt, 2008). A significant decrease in MUCP and in functional profile length (FPL) is observed in dogs with USMI compared to continent dogs (Rosin and Barsanti, 1981; Richter and Ling, 1985; Holt, 1988) (Figs. 1 and 2).

Bladder neck position and urethral length may play a role in the development of USMI. Pelvic bladder, defined as more than 5% of the bladder length located inside the pelvis (DiBartola and Adams, 1983), is a frequent radiographic finding in incontinent bitches and is generally associated with a shorter urethra (Holt et al., 1984; Holt, 1985a,b). The pelvic position of the bladder neck could alter the pressure transmission between bladder and urethra (DiBartola and Adams, 1983; Holt, 1985b) (Fig. 3).

A relationship between neutering and USMI exists, as 90% of bitches with USMI are spayed (Holt, 1985a; Krawiec, 1989) and up to 20% of spayed bitches develop urinary incontinence (Arnold et al., 1989). However, the relationship between USMI and the age of spaying is controversial. A lower rate of incontinence has been reported in bitches spayed before the first oestrus cycle (Holt, 1985a; Arnold et al., 1989; Stöcklin-Gautschi et al., 2001; de Bleser et al., 2010). USMI can also be congenital with half of the affected juvenile bitches becoming continent following their first or second oestrus (Holt, 1985a, 2008).

Sterilization is associated with urethral structural modifications. After sterilization, a decrease in smooth muscle is observed in both bladder and urethra, whilst an increase in the volume of vascular urethral plexus is observed in the first quarter of the urethra (Augsburger and Cruz-Orive, 1995; Ponglowhapan et al., 2008a). Structural modifications on collagen content are controversial (Augsburger and Cruz-Orive, 1995; Augsburger and Oswald, 2007), with recent studies reporting an increased proportion of collagen in both the bladder and the urethra in neutered dogs (Ponglowhapan et al., 2008a). Excessive collagen deposit and decreased muscle volume could impair the functional integrity of the lower urinary tract. In spayed bitches, the total number of types I and II striated fibres is decreased (Augsburger and Cruz-Orive, 1995, 1998). The decrease in type II fibres is compensated by

an increase in their volume (Augsburger and Cruz-Orive, 1998). As type I fibres contribute to the resting urethral tone, their decreased number could also be involved in the weakness of the urethral closure mechanism.

Urethral length is shorter in spayed compared to intact bitches (Wang et al., 2006). Sterilization is also associated with urethral functional modifications. Ovariectomy induces a significant decrease in MUCP (Nickel, 1998; Reichler et al., 2004), FPL and integrated pressure (IP) (Salomon et al., 2006). Different hypotheses have been proposed to explain the decreased urethral resistance observed after neutering, such as adhesions between the bladder neck and uterine stump or damage to the lower urinary tract supporting structures at the time of surgery. These hypotheses are now obsolete (Holt, 1990) and no significant difference was observed between the percentage of dogs developing USMI after ovariectomy or ovariohysterectomy (Arnold et al., 1989).

Oestrogen deficiency is the most widespread explanation for the development of USMI. However, no significant difference was observed between the endogenous oestrogen concentration of continent bitches in anoestrus and spayed incontinent bitches (Richter and Ling, 1985) so the possible correlation between low oestrogen plasma concentrations and USMI remains controversial. High affinity oestradiol receptors are located at the level of the proximal urethra (Larsson et al., 1984; Schulze and Barrack, 1987) and oestrogens are known to increase the number and the responsiveness of α-adrenergic receptors to sympathetic stimulation (Osborne et al., 1980; Creed, 1983). Nickel (1998) demonstrated that a high 17β-oestradiol plasma concentration during the late follicular phase was correlated with increased FPL whereas low 17β-oestradiol plasma concentration was associated with decreased MUCP and IP in ovariectomized bitches. Changes in urethral resistance during the oestrus cycle has been confirmed, with increasing values observed from late anoestrus to pro-oestrus and decreasing values observed in oestrus and early dioestrus (Hamaide et al., 2005).

The role of gonadotrophins in the development of USMI has been suggested as ovariectomy induces a chronic increase in follicle stimulating hormone (FSH) and luteinizing hormone (LH) (Reichler et al., 2003). However, no relationship between increased gonadotrophin concentration and decreased MUCP values has been demonstrated (Reichler et al., 2004, 2006).

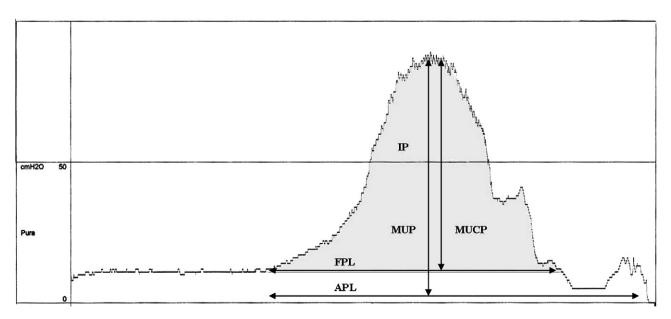


Fig. 1. Urethral pressure profile of a continent bitch. Pura, urethral pressure; MUP, maximal urethral pressure; MUCP, maximal urethral closure pressure; APL, anatomical profile length; FPL, functional profile length; IP, integrated pressure under the urethral functional profile.

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