Urinary incontinence (UI) is the involuntary loss of urine. UI rarely occurs in sexually intact bitches (0-1%)\(^1\), whereas in spayed bitches the incidence is up to 20%\(^2\). The underlying pathophysiological mechanism is a reduced closure pressure of the urethra after spaying\(^3\).

The causal relationship between the removal of the ovaries and UI has been clearly demonstrated\(^4\). However, it is still unclear what mechanism triggers UI after spaying. An oestrogen deficiency was initially considered to be the underlying cause\(^5\). This hypothesis is however contradicted by several observations. For example, bitches treated with depot preparations of gestagens, to suppress oestrus, do not have an increased risk of UI, even though the treatment results in ovarian atrophy\(^6\) and the oestrogen remains in a basal level\(^7\).

Another side effect of spaying is the increase in plasma gonadotropins, due to the lack of the ovarian negative feedback\(^8\). About 42 weeks after ovarectomy the gonadotropin levels reach a plateau, when the plasma FSH is 17 times and the plasma LH is 8 times the initial concentration\(^9\). One could therefore ask if it is the elevated plasma level of FSH and LH that are responsible for the increased risk of UI in spayed bitches. If this were correct, then affected bitches could be successfully treated with depot preparations of GnRH-analogues, through down-regulation of GnRH-receptors in the pituitary and this in turn will decrease the plasma gonadotropin concentrations. Indeed, 7 of 13 bitches affected by UI were successfully treated with an injection of depot preparations of GnRH-analogues and remained continent for an average of 247 days\(^10\). However, it is questionable whether the success of this treatment is due to a decrease in gonadotropins since their blood levels in responders and non-responders are not different\(^11\). It is possible that GnRH has a direct effect on the lower urinary tract, but the success of the therapy is not based on a normalisation of the urethral sphincter incompetence after spaying\(^11\). Recent studies in beagle bitches have given rise to the assumption that GnRH modulates the function of the bladder\(^12\).

The treatment of incontinent bitches with GnRH-analogues is mainly interesting for the clarification of the pathophysiological mechanism. For patients affected by UI, the therapy of first choice is with alpha-adrenergica (Phenylpropanolamine / Ephedrine). This results in an increased urethral closure pressure and continence in more than 90% of cases.

If the therapeutic effect is insufficient, then alpha-adrenergica may be combined with oestrogen or Flavoxatum. In refractory cases, several surgical methods are described of
which colposuspension\textsuperscript{13}, urethropexy\textsuperscript{14} and the endoscopic injection of collagen\textsuperscript{15} are most common and have a success rate of 50 - 75%.

References

Urinary Incontinence (UI) in spayed bitches: frequency, causes, therapy

Iris Reichler, Madeleine Hubler, Susi Arnold
Vetsuisse Faculty, University Zurich, Switzerland

UI in adult spayed bitches

• Neurogenic
• Non-neurogenic
  – USMI
  – Ureterovaginal fistula
  – Urovagina
  – Ectopic ureter
  – Tumor
  – Cystitis
Session I: Non-reproductive Effects of Spaying and Neutering
Incontinence: Frequency, Causes and Therapy
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UI in adult spayed bitches

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Spaying - UI

• Interval between spaying and UI
  – Immediately: 10 years
  – Mean 2.9 years post-op
  – 75 % of the cases within 3 years

Spaying - UI

- Interval:
  - Immediately: 10 years

- Incidence:
  - Spayed bitches 3% – 21%
  - Intact bitches 0.2 – 2.1%
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Risk factors in spayed bitches

- Body weight
- Breed
  - Boxer
  - Doberman
  - Rottweiler
  - Giant Schnauzer
  - Old English Sheepdog

Arnold 1989

Reichler 2005
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Prepubertal spay: risk 50%

Urethral closure pressure

Continent 18 cm H₂O
Incontinent 4 cm H₂O
Critical limit 7.5 cm H₂O

Arnold 1997
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**Urethral pressure profile**

- **Intact, continent**
  - MUCP = 35 cm H₂O
- **Spayed, incontinent**
  - MUCP = 5 cm H₂O

**Factors contributing to urethral closure**

- Neuromuscular components 60%
  - Somatic 0%
  - Sympathetic 50%
  - Parasympathetic 10%
- Non-neuromuscular components 40%
  - Venous plexus 20%
  - Connective tissue 20%

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**Therapy USMI**

- **α-adrenergic substances**
  - Phenylpropanolamine
    - 1.5mg/kg bid, tid PO
  - Ephedrine
    - (1-2mg/kg bid PO)
  - Continence: 85-98%


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**Therapy USMI**

- **α-adrenergic substances**
- **Oestrogens:**
  - Increased responsiveness to alpha-agonists

Schreiter 1976
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Therapy USMI

- α-adrenergic substances
- Oestrogens:
  - Increased responsiveness to alpha-agonists
  - Cell growth and proliferation
  - Increase of bladder threshold
  - Continence: 60-65%
  - Estriol 1mg /dog /day


Forms of UI in spayed bitches

- USMI
- Combined form
- Detrusor instability
- Normal UPP

Nickel 1997
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Combined Therapy

α-adrenergic substances
&
drugs for detrusor instability

– Anticholinergic agent (propantheline)
– Antispasmodic medications (oxybutynine, tolteridine, flavoxate, diphenpyraline)
– Tricyclic antidepressants (imipramine, doxepine)
– Beta agonist (terbutaline)

Combined Therapy

Phenylpropanolamine 1.5mg/kg bid-tid
&
Flavoxate 10mg/kg bid
Effect of treatment

• Urethral closure↑
• Relaxation of the bladder
• Compensation of the “oestrogen deficit“

Removal of the ovaries → endocrine consequences?
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Feedback

Hypothalamus

GnRH

Pituitary gland

Estrogens

Changes in gonadotropins following spaying

Reichler 2004
Disturbed Feedback

Hypothalamus

GnRH

Pituitary gland

FSH, LH

? Urinary incontinence?

FSH-, LH- and GnRH-receptors in the urinary tract

Welle 2006
GnRH treatment

35 incontinent bitches
- 18 continent
- 13 improved
- 4 unchanged

Reichler 2006

UPP after GnRH treatment:

Spayed
incontinent

After GnRH-treatment,
continent

Reichler 2006
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Cystometry

before treatment

post GnRH-treatment

Endoscopic injection of collagen
Submucosal injection of collagen
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End of procedure

Long-term success of collagen injection

Barth 2002

Continent  Improved  Incontinent
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