

**Lower Urinary tract Contrast Studies in the Male Dog  
& Case Studies**

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## Summary

The value of combining double contrast cystography with retrograde urethrography as a single study was investigated in 20 male dogs, which had 21 studies. These animals had been presented with suspected lower urinary tract and/or prostatic disease to Glasgow University Small Animal Hospital. The most common presenting signs were haematuria (8/21) and defaecatory tenesmus (8/21). In 18/21 studies there was poor colonic preparation, but it only affected the radiological interpretation in 6 cases. Air bubbles were found to be the commonest filling defects in bladder (12/21) and urethra (12/21). The radiological abnormality most frequently encountered was prostatomegaly (9/21), though no abnormalities were detected in 8 studies. The prostatic urethra was adequately distended or just visible in the majority of the studies (18/21) regardless of the positioning of the catheter tip. It was invisible in all but one of the 5 cases where the bladder neck was intra-pelvic. Extravasation of contrast into the substance of the prostate was observed in five of the 21 studies. Disruption or a ragged appearance to the prostatic urethra was present in the one prostatic tumour, but also in two of the four cases of prostatic/para-prostatic cyst. No evidence of mineralisation of prostatic tissue was found in any of the cases. Disease associated with the prostate was the most common final clinical diagnosis (14/21). However, benign prostatic hyperplasia was diagnosed in three cases without demonstrable prostatomegaly. The conclusion reached was that the combined technique was not superior to double contrast cystography and retrograde urethrography carried out individually.

In addition, the presentation and management of ten soft tissue cases are described.

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## **Dedication**

To my parents -Salvador y Olga- and my sister Maly; without them this would not have been possible.

## **Declaration**

I, María Teresa Mato Pintané, do hereby declare that the work carried out in this thesis is original, was done by myself or with due acknowledgement, and has not been presented for the award of a degree at any other University.

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*“The best is yet to come...”*

Frank Sinatra

# **SECTION 1**

# 1. Lower Urinary Tract Contrast Studies in the Male Dog

---

## 1.1 Introduction

The urinary bladder functions both as a reservoir for urine produced by the kidneys and as an excretory organ for expulsion of urine through the urethra. Together, the bladder and urethra comprise what is referred to as the lower urinary tract (LUT).

Common clinical signs manifested by LUT disease are haematuria, dysuria and urinary incontinence. Nevertheless, systemic signs such as raised body temperature or elevated circulating white blood cell count are not usually present, and clinical examination is often unrewarding. Consequently, diagnostic imaging is one of the more helpful tools in localising the disease process in and to the LUT (Park 1981).

Survey caudal abdominal radiographs must be taken prior to any contrast study of the LUT to avoid masking radio-opaque lesions with contrast. However, the main limitation of survey films is the inability to detect intramural and intra-luminal soft tissue alterations. This is because the bladder wall and urine are of the same density, so mucosal margins cannot be identified. Further, an empty bladder and the normal urethra are not visible on survey radiographs (Mahaffey and Barber 1992, Johnston and others 1995).

Retrograde contrast radiographic studies are simple, fast and minimal risk procedures that highlight the lumen and walls, and thus alterations in the LUT (Park, 1981). A number of different single contrast studies and combination contrast studies have been advocated (Park 1981, Burk and Ackerman 1996).

The simplest study is probably the pneumocystogram, where the bladder is distended with air or a gas such as CO<sub>2</sub> or N<sub>2</sub>O, allowing the bladder wall to be evaluated. Unfortunately, insufficient distension may be easily misdiagnosed as bladder thickening (Park 1974). Although inexpensive, this method provides poor mucosal detail and small to moderate sized filling defects may not to be noticed (Johnston and others 1995).

The alternative single contrast study is positive contrast cystography, where soluble organic iodides based on sodium or meglumine salts are used to distend the bladder. It is the procedure of choice for detecting bladder tears or rupture (Mahaffey and Barber

1992). However, the main disadvantage is the masking of subtle wall thickening and small luminal filling defects (Johnston and others 1995, Park 1981).

To overcome the disadvantages of single contrast techniques, double contrast techniques have gained popularity. Double contrast cystography is performed by introducing a small volume of positive contrast followed by negative contrast injection to complete bladder distension. This technique provides the best evaluation of bladder mucosa, bladder wall thickness and, by means of the contrast puddle in the dependent portion, serves as an aid in identifying different kinds of luminal filling defects (Park 1974).

The above three studies do not really address satisfactorily the second component of the LUT, namely the urethra. Retrograde urethrography has been recommended for the evaluation of urethral disease (Ticer and others 1980), as an accurate procedure to locate and determine the extent of urethral ruptures, obstructions and congenital anomalies (Park 1981) and prostatic disease (Thrall 1994).

A technique that combined positive contrast retrograde urethrography with double contrast cystography as a means to overcome the individual disadvantages of each retrograde contrast study previously described has been proposed.

An analysis of 20 male dogs, in which this combined technique was performed (21 studies), was carried out to test the hypothesis that it is superior to other individual contrast studies.

## 1.2 Materials & Methods

An analysis of radiographs from 20 male dogs presented with suspected bladder, urethral and/or prostatic disease at Glasgow University Veterinary Hospital during the period of 1995-2000 was performed.

The cases were extracted from the hospital database by searching for dogs where combined positive contrast retrograde urethrography with double contrast cystography was done as part of the diagnostic procedure.

The case record was obtained and data was compiled concerning breed, age, neutering, presenting sign and body weight. As well as details of radiological evaluation of bladder, urethra and prostate, the degree of emptiness of colon was noted in each case to assess its influence on the observation of lower urinary tract alterations.

Combined positive contrast retrograde urethrography with double contrast cystography was carried out in all dogs under general anaesthesia and in lateral recumbence. In all cases plain films were obtained prior to the contrast study.

Lubricating jelly was used on the tip of an adequately sized polypropylene urinary catheter to ease insertion into the urethra. The bladder was drained with a 60 ml syringe connected to the catheter with a 3 way tap.

A contrast solution was prepared of 5 to 15 ml, according body size, of sodium iothalamate (420 mg/ml iodine) diluted 2:1 with sterile water for injection.

Once the bladder was emptied the urethra was occluded with a Doyen bowel clamp immediately distal to the os penis to avoid leakage. One third of the solution was introduced into the bladder, which was gently massaged to ensure good distribution. Air was insufflated to achieve adequate bladder distension, which was checked by digital palpation of the bladder through the body wall. The catheter was retracted until, by digital palpation of distal membranous urethra, the catheter tip was thought to be located immediately proximal to the os penis. The remaining solution was then injected and on completion a radiograph was taken immediately the person injecting had left the controlled area.

## **1.3 Results**

### **1.3.1 Breed distribution**

The cases analysed in this study included a wide range of breeds. Although no obvious predominance was observed, the Boxer was the breed in which the contrast procedure was performed most often (4/21). Two studies were performed on the same dog 3.6 months apart for recurrence of a prostatic cyst (study 10 and 13). Other breeds observed in this study are listed in table 1.

### **1.3.2 Age distribution**

The average age of the dogs analysed was 5.5 years with a range of 0.10-11 years. The majority of animals (61.9%) included in this study were aged between 5.5 and 11 years. Dogs less than 5.5 years were less frequent (38.1%) (Table 1).

### **1.3.3 Sex distribution**

As prostatic disease is strongly linked to testicular activity, neutering was considered a factor. Most of the dogs included in the study were entire (15/21) with only 6 neutered prior to presentation (Table 1).

### **1.3.4 Weight distribution**

The majority of the dogs (80.9%) had a body weight ranging between twenty and forty kilograms (Table 1). The average body weight of the cases was found to be 23.75 kg.

| Study No | Breed                    | Age (years) | Neutered | Weight (kg) |
|----------|--------------------------|-------------|----------|-------------|
| 1        | German Shepherd Dog      | 3           | Yes      | 38          |
| 2        | Basset Hound             | 0.10        | No       | 23          |
| 3        | Springer Spaniel         | 11          | No       | 21.5        |
| 4        | Irish Setter             | 5           | No       | 36          |
| 5        | Dachshund                | 8           | Yes      | 13          |
| 6        | Basset Hound             | 9           | Yes      | 21          |
| 7        | Belgium Shepherd Dog     | 10          | No       | 35          |
| 8        | Old English Shepherd Dog | 2           | No       | 28          |
| 9        | Labrador Retriever       | 2.3         | No       | 30          |
| 10       | Boxer                    | 7.6         | Yes      | 29          |
| 11       | Border Collie            | 7.8         | No       | 21.7        |
| 12       | Irish Setter             | 4.4         | No       | 37          |
| 13       | Boxer                    | 8.1         | Yes      | 29          |
| 14       | Jack Russell Terrier     | 11          | No       | 9.3         |
| 15       | Crossbred                | 7           | No       | 26          |
| 16       | Springer Spaniel         | 8           | No       | 22.5        |
| 17       | Boxer                    | 5           | No       | 31          |
| 18       | Boxer                    | 7           | No       | 31          |
| 19       | Border Terrier           | 9           | Yes      | 9           |
| 20       | Golden Retriever         | 9           | No       | 39          |
| 21       | Border Terrier           | 1           | No       | 8.5         |

**Table 1. Breed, age, sex and weight of 20 dogs undergoing 21 combined lower urinary tract contrast studies.**

### 1.3.5 1.3.5 Presenting signs

The major presenting features recorded were haematuria (8/21) and defaecatory tenesmus (8/21). Less frequent were dysuria (2/21), urine dripping (2/21) and penile bleeding (2/21). In some cases more than one feature was reported as a presenting sign (Table 2).

### **1.3.6 Radiological diagnoses**

The radiographic finding most frequently identified after subjective evaluation of the 21 studies was prostatomegaly (9/21). No significant abnormalities were observed in 8 of the 21 studies. In the remaining four, diagnoses of different lower urinary alterations, such as ectopic ureter, bladder mass, urethral diverticulum and hypospadiasis were made (Table 2).

### **1.3.7 Clinical diagnoses**

Radiographic observations in conjunction with other diagnostic techniques performed in each case led to a final clinical diagnoses. Prostatic abnormalities were found to be the cause of the presentation in most of the cases (14/21); benign prostatic hyperplasia (BPH) (5/14), prostatic or para-prostatic cyst (Pr. Cyst or PPr. Cyst) (4/14), idiopathic prostatic haemorrhage (IPH) (3/14), prostatitis (1/14) and prostatic tumour (1/14) respectively. The urethra was also affected (3/21) with diagnoses of urethral sphincter mechanism incompetence (USMI), urethral sphincter spasm (USS) and hypospadiasis. The urinary bladder was involved in two cases, with bladder entrapment in a perineal hernia and intraluminal polyp respectively. The two remaining cases presented penile self mutilation and ectopic ureter.

| Study No | Clinical sign                     | Diagnoses            |                   |
|----------|-----------------------------------|----------------------|-------------------|
|          |                                   | Radiological         | Clinical          |
| 1        | Urinary Tenesmus                  | None                 | USMI              |
| 2        | Perineal stoma                    | Hypospadiasis        | Hypospadiasis     |
| 3        | Haematuria                        | Prostatomegaly       | Pr. Cyst          |
| 4        | Defaecatory tenesmus              | None                 | BPH               |
| 5        | Haematuria and dysuria            | Prostatomegaly       | Prostatic tumour  |
| 6        | Defaecatory tenesmus & haematuria | None                 | BPH               |
| 7        | Penile bleeding                   | Prostatomegaly       | IPH               |
| 8        | Penile bleeding                   | Prostatomegaly       | Penile mutilation |
| 9        | Defaecatory tenesmus              | None                 | Prostatitis       |
| 10       | Defaecatory tenesmus              | Prostatomegaly       | Pr. Cyst          |
| 11       | Defaecatory tenesmus              | Prostatomegaly       | BPH               |
| 12       | Dysuria                           | Urethral diverticuli | USS               |
| 13       | Defaecatory tenesmus              | Prostatomegaly       | Pr. Cyst          |
| 14       | Defaecatory tenesmus              | None                 | BPH               |
| 15       | Defaecatory tenesmus              | None                 | Perineal hernia   |
| 16       | Haematuria and urine dripping     | None                 | IPH               |
| 17       | Haematuria                        | Prostatomegaly       | PPr. Cyst         |
| 18       | Haematuria                        | None                 | IPH               |
| 19       | Haematuria                        | Bladder mass         | Bladder polyp     |
| 20       | Haematuria                        | Prostatomegaly       | BPH               |
| 21       | Urine dripping                    | Ectopic ureter       | Ectopic ureter    |

*BPH = benign prostatic hyperplasia, Pr. Cyst or PPr. Cyst. prostatic or para-prostatic cyst, IPH = idiopathic prostatic haemorrhage, USMI= urethral sphincter mechanism incompetence, USS= urethral sphincter spasm*

**Table 2. Clinical signs and diagnoses made in 20 dogs by 21 combined lower urinary tract contrast studies.**

### **1.3.8 Colon preparation and interference in the radiological interpretation**

Enemas were performed prior to the contrast studies and their effectiveness in clearing the distal portion was assessed. Most of cases (18/21) had varying degrees of faecal content still filling the colon (Figure 1). In only 3 cases was the distal colon empty of faecal content. However, it was noted that in most cases interpretation was not affected by the presence of faeces (15/21). The colonic content had a negative effect on the evaluation of the radiographs in 6 studies (Figure 2).

### **1.3.9 Urethral location of catheter tip**

As part of the study, the catheter was withdrawn to a level which was thought correspond to just proximal to the os penis. The actual position was recorded and its effect on interpretation noted. In all studies the catheter had been withdrawn distal to the prostate (Figure 3 & Figure 4). In 4/21 studies the catheter was present in the membranous urethra (Table 3). In none of these cases was interpretation compromised by the catheter.

### **1.3.10 Degree of bladder distension**

Subjective assessment of *satisfactory* bladder distension was made on the basis of the observation of a normal “pear shaped” bladder (Figure 3 & Figure 4). In only studies 1 and 3 was this feature absent.

### **1.3.11 Puddle of contrast**

Following contrast cystography the puddle of positive contrast material remained in the dependent portion of the bladder, and this was noticed in 19 of the 21 studies. In study 3 the puddle, predominantly dorso-caudal, almost completely filled the very small bladder. In study 17 the contrast media was forced caudally by the presence of a soft tissue density mass ventral to the bladder (Figure 5).

### **1.3.12 Bladder wall thickness and severity**

Bladder wall thickness was a common finding in the studies, where (47.6%) had some degree of thickening (Figure 6). The increased thickness could be classified as *focal* (4/21) and *generalised* (6/21). In all the cases where wall abnormalities were observed, the degree of severity was classified subjectively as *mild*, compared to normal thickness.

### 1.3.13 Bladder filling defects

After injection of both contrast agents, the radiographic image showed no filling defects related to the puddle and its periphery in 7 of the studies . Air bubbles (12/21), blood clots or cellular debris (2/21), calculi (2/21) and intra-luminal soft tissue structures attached to the mucosal surface were found (Figure 3, Figure 4 & Figure 9). In some of the cystograms more than a type of filling defect was recorded in studies 5, 7 and 13 (Table 3).

| Study | Catheter tip placement | Bladder filling defects  |
|-------|------------------------|--------------------------|
| 1     | Penile                 | None                     |
| 2     | Membranous             | None                     |
| 3     | Penile                 | Air bubbles              |
| 4     | Penile                 | None                     |
| 5     | Penile                 | Air bubbles, blood clots |
| 6     | Penile                 | None                     |
| 7     | Penile                 | Air bubbles, calculi     |
| 8     | Penile                 | Air bubbles              |
| 9     | Penile                 | Air bubbles              |
| 10    | Penile                 | Air bubbles              |
| 11    | Membranous             | Air bubbles              |
| 12    | Penile                 | None                     |
| 13    | Penile                 | Air bubbles, calculi     |
| 14    | Membranous             | Blood clots              |
| 15    | Penile                 | Air bubbles              |
| 16    | Penile                 | Air bubbles              |
| 17    | Penile                 | Air bubbles              |
| 18    | Penile                 | None                     |
| 19    | Penile                 | Intra-luminal growth     |
| 20    | Penile                 | None                     |
| 21    | Membranous             | Air bubbles              |

**Table 3. Catheter tip location and filling defects in 20 dogs following 21 combined lower urinary tract contrast studies.**

#### **1.3.14 Position bladder neck/prostate**

In the majority of the studies (12/21) bladder neck/ prostate was found at the pelvis inlet as is considered normal (Table 4). An intra-pelvic location was found in 5/21 (Figure 6) and a more cranial intra-abdominal position in 4/21 (Figure 10).

#### **1.3.15 Prostatic urethra visualisation and evaluation**

The prostatic urethra could not be visualised in only three of the cases, whereas in the rest of the studies, this urethral segment was adequately *distended* (11/21) or just *visible* (7/21) (Figure 1 & Figure 3). This allowed full evaluation and detection of abnormalities in studies 3, 5 and 13 (Table 4).

#### **1.3.16 Prostatic urethra symmetry**

In 7 of the studies this evaluation could not be performed due to failure of the contrast to show the prostatic urethra and/or prostate gland. In 7 studies the position and appearance of the prostate was symmetrical. In the remaining 7, the urethra appeared asymmetric through its course within the prostate (Table 4) (Figure 10).

| <b>Study</b> | <b>Bladder neck/-prostate position</b> | <b>Prostatic urethra</b> | <b>Prostatic urethra symmetry</b> |
|--------------|--|--------------------------|-----------------------------------|
| 1            | Intra-pelvic                           | Invisible                | ?                                 |
| 2            | Intra-pelvic                           | Distended                | ?                                 |
| 3            | Intra-abdominal                        | Visible                  | No                                |
| 4            | Intra-pelvic                           | Invisible                | ?                                 |
| 5            | Intra-abdominal                        | Visible                  | Yes                               |
| 6            | Normal                                 | Distended                | No                                |
| 7            | Normal                                 | Visible                  | Yes                               |
| 8            | Normal                                 | Visible                  | Yes                               |
| 9            | Normal                                 | Distended                | Yes                               |
| 10           | Normal                                 | Distended                | No                                |
| 11           | Normal                                 | Distended                | No                                |
| 12           | Normal                                 | Visible                  | Yes                               |
| 13           | Intra-abdominal                        | Distended                | No                                |
| 14           | Normal                                 | Distended                | Yes                               |
| 15           | Intra-pelvic                           | Distended                | ?                                 |
| 16           | Normal                                 | Distended                | Yes                               |
| 17           | Intra-abdominal                        | Visible                  | No                                |
| 18           | Intra-pelvic                           | Invisible                | ?                                 |
| 19           | Normal                                 | Distended                | ?                                 |
| 20           | Normal                                 | Visible                  | No                                |
| 21           | Normal                                 | Distended                | ?                                 |

**Table 4. Prostatic location and symmetry in 20 dogs obtained from 21 combined lower urinary tract contrast studies.**

### **1.3.17 Urethral filling defects**

Air bubbles (12/21) and a calculus (1/21) were the only urethral filling defects encountered (Figure 4) (Table 5).

### **1.3.18 Prostate size**

A subjective assessment of the prostatic volume was done. A small to moderate enlargement was labelled as *BPH-type* (8/21), whereas a pronounced enlargement with obvious displacement of peri-prostatic structures was defined as *Cyst-type* (3/21). The size was considered to be normal in 4 studies and in the remaining 6 it was not possible to evaluate size, due to a failure to adequately visualise the prostate during the contrast study (Table 5).

### **1.3.19 Extravasation of contrast**

Presence of contrast within the prostatic substance was noticed in a minority of cases (5/21). Four of these cases presented a very subtle localised extravasation (*Blush-type*) (Figure 7), whereas in case number 5, the positive material formed a more massive and irregular shape (Figure 9) within the prostatic parenchyma (*ragged-type*) (Table 5).

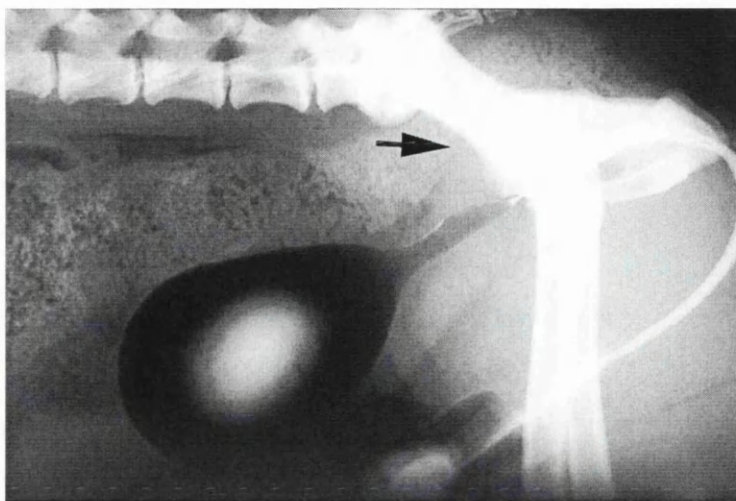
### **1.3.20 Prostatic mineralisation**

Although considered as a parameter in the assessment of the radiological findings, none of the cases presented signs of mineralisation of the prostatic tissue.

| Study | Urethral filling defects | Prostate size | Contrast extravasation |
|-------|--------------------------|---------------|------------------------|
| 1     | None                     | ?             | No                     |
| 2     | None                     | ?             | No                     |
| 3     | Air bubbles              | Cyst-type     | Blush                  |
| 4     | None                     | ?             | No                     |
| 5     | Air bubbles              | BPH-type      | Ragged                 |
| 6     | None                     | Normal        | No                     |
| 7     | Air bubbles              | BPH-type      | Blush                  |
| 8     | Air bubbles              | BPH-type      | Blush                  |
| 9     | None                     | Normal        | No                     |
| 10    | Air bubbles              | BPH-type      | No                     |
| 11    | Air Bubbles              | BPH-type      | No                     |
| 12    | Air bubbles              | Normal        | No                     |
| 13    | None                     | Cyst-type     | No                     |
| 14    | Air Bubbles              | BPH-type      | Blush                  |
| 15    | Air bubbles              | BPH-type      | No                     |
| 16    | Air bubbles              | Normal        | No                     |
| 17    | Air Bubbles              | Cyst-type     | No                     |
| 18    | None                     | ?             | No                     |
| 19    | Calculus                 | ?             | No                     |
| 20    | None                     | BPH-type      | No                     |
| 21    | Air bubbles              | ?             | No                     |

*BPH=benign prostatic hyperplasia, ?= not definable*

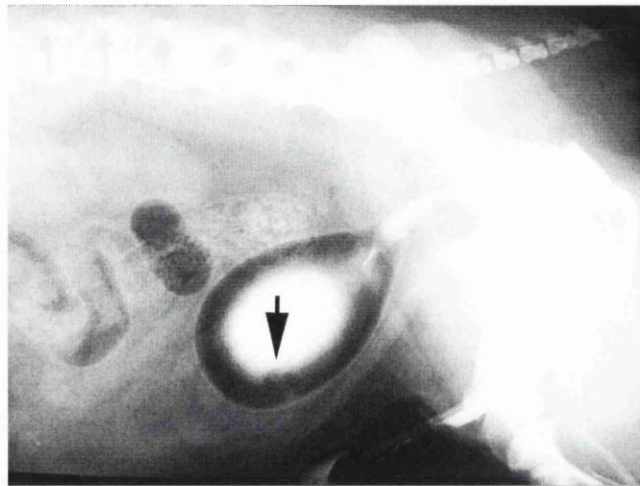
**Table 5. Details of prostatic urethra, prostatic size and contrast leakage in 20 dogs from 21 combined lower urinary tract contrast studies.**



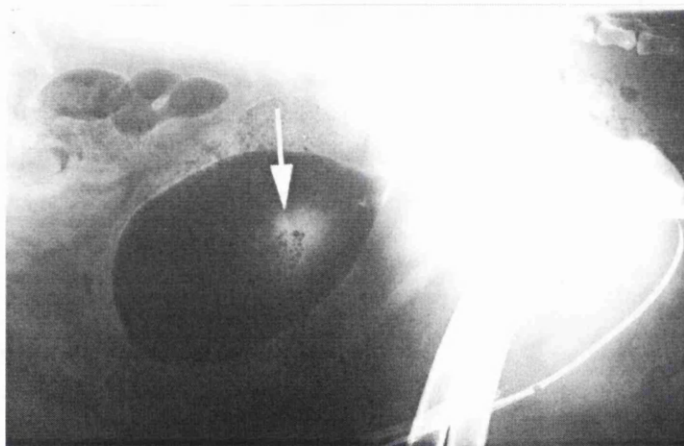
**Figure 1.** Radiograph of a combined contrast study. The faeces-filled distal colon/rectum highlights the degree of intestinal compression caused by a prostatic cyst (arrow). Peri-urethral asymmetry is also evident.



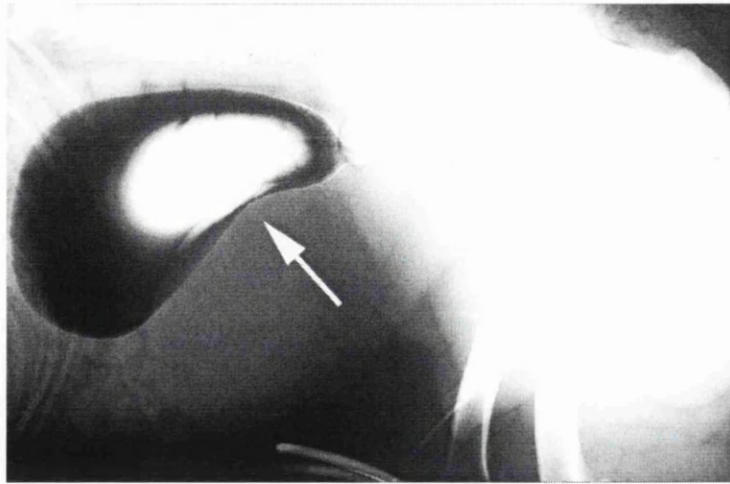
**Figure 2.** Radiograph of a combined contrast study. The presence of excessive gas generated by the enemas interferes with the radiological evaluation in this case.



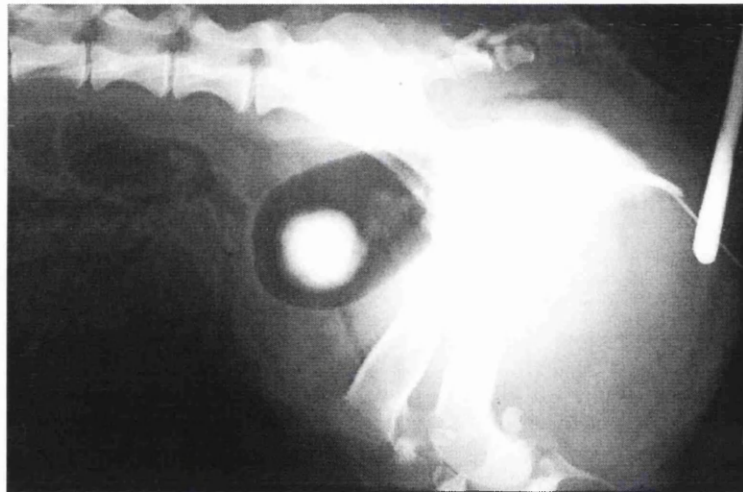
**Figure 3.** Radiograph of a combined contrast study. The prostatic urethra is widely distended following penile urethral injection of contrast. There is a generalised increase in bladder wall thickness. A filling defect created by a bladder polyp has caused distortion of the contrast puddle cranio-ventrally (arrow).



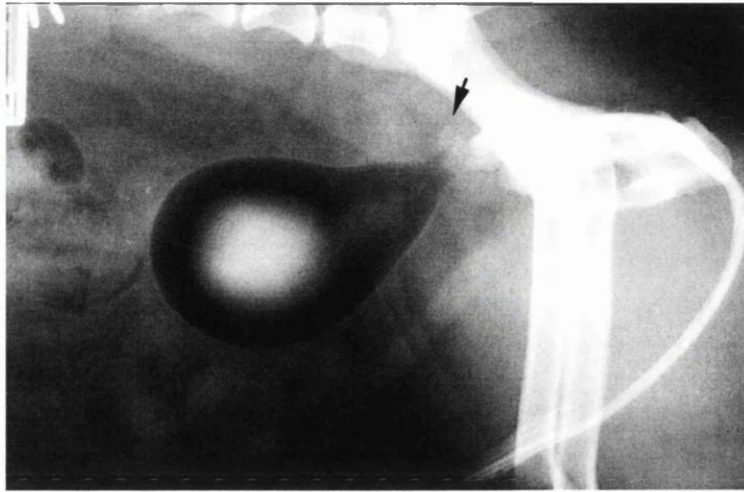
**Figure 4.** Radiograph of a combined contrast study. There is a generalised increase in bladder wall thickness. Radiolucent cystic calculi are located in the centre of the contrast puddle (arrow). At least one air bubble is present in the distal urethra.



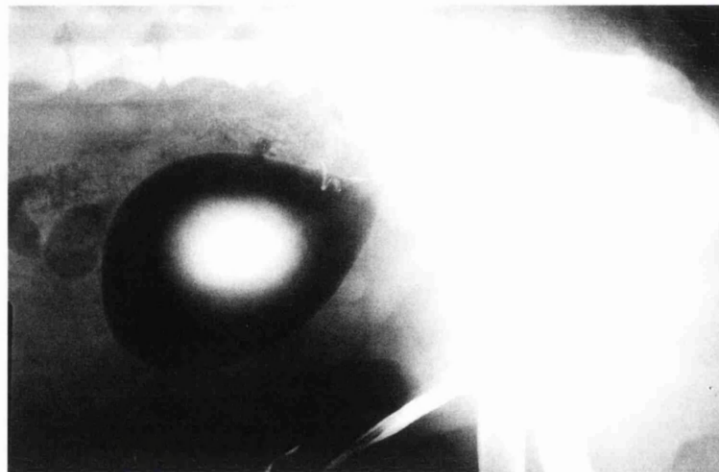
**Figure 5** Radiograph of a combined contrast study. There is an intra-abdominal bladder with compression and displacement due to the presence of a prostatic cyst (arrow).



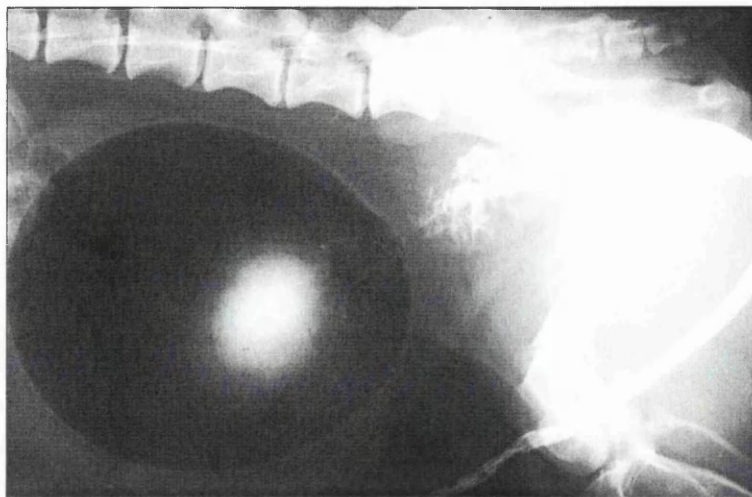
**Figure 6** Radiograph of a combined contrast study. There is an intra-pelvic bladder present in this case of hypospadias. The prostatic urethra is distended and there is a generalised increase in bladder wall thickness.



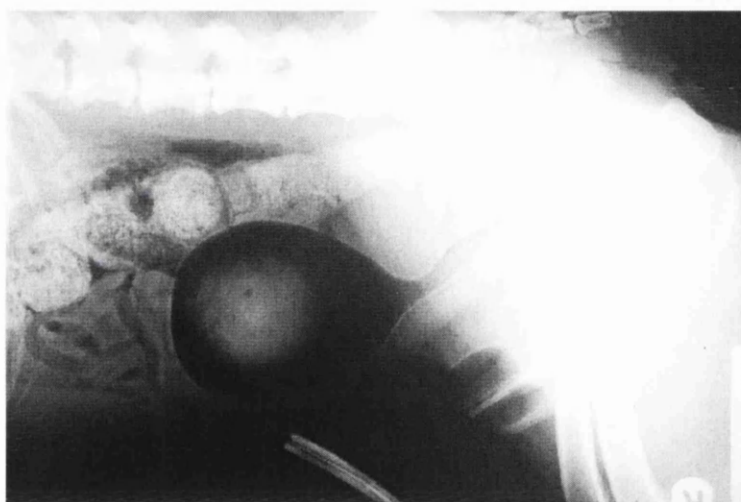
**Figure 7.** Radiograph of a combined contrast study. The urethra is widely distended throughout its length following injection from a catheter located in the membranous portion of the urethra. Contrast extravasation (*blush*) is evident in a dog with a clinical diagnosis of benign prostatic hypertrophy (arrow).



**Figure 8.** Radiograph of a combined contrast study. The urethra is extremely well distended with contrast and one of the ureters is filled with positive contrast.



**Figure 9.** Radiograph of a combined contrast study. A ragged prostatic urethra and massive peri-urethral extravasation of contrast is evident in this case of prostatic carcinoma. There are air bubbles/blood clots and debris present in the contrast puddle.



**Figure 10.** Radiograph of a combined contrast study. There is prostatic asymmetry, urethral displacement and compression caused by a prostatic cyst.

## 1.4 Discussion

The urinary bladder is a hollow musculo-membranous organ that varies in form, size and position, depending on the amount of urine it contains. The prostate, the only accessory sex gland in the male dog, completely envelops the proximal portion of the urethra at the neck of the bladder (Evans and Christensen 1993).

Such close relationship between the components of the lower urinary tract and the prostate is not only merely a matter of boundaries but of function. Prostatic fluid, acts as a transport and support medium for sperm during ejaculation, but basal secretion is constantly entering the prostatic excretory ducts and urethra. When neither micturition nor ejaculation is occurring, urethral pressure moves this basally secreted fluid cranially into the bladder (Barsanti and Finco 1986).

The prostate, as well as the other components of the lower urinary tract, is radiographically a soft tissue dense structure, and in many instances, clear visualisation of the prostatic shadow requires the administration of contrast material to outline the lumen and identify the gland (Bartels 1981, Barsanti and Finco 1986).

Johnston and others (1991) recommended that identification of prostatic urethra and urinary bladder by positive contrast retrograde urethrography should be performed for evaluation of prostatic disease. Positive retrograde urethrography is extremely useful for assessment of urethral and urinary bladder integrity after caudal abdominal or pelvic trauma and for investigation of stranguria, dysuria, incontinence or haematuria (Ticer and others 1980). Performing this technique with an incompletely distended urinary bladder causes luminal narrowing of the cranial and mid-regions of the prostatic and pelvic urethra on radiographs (Johnston and others 1983a, Poogird and Wood 1986). This is probably due to the longitudinal mucosal folds that project into the urethral lumen (Evans and Christensen 1993).

Double contrast cystography has been found to be the most complete contrast technique to assess possible pathologic conditions of the bladder (Park 1974). It is recommended that pneumocystography routinely follow positive contrast cystography, since the mucosal surface of the urinary bladder is best appreciated when the bladder is opacified and the thickness of its wall is best seen when contrasted with air (Root 1984).

actually presented interpretation problems due to gas or faecal content within the intestinal lumen, which implies that colonic evacuation is not critical. Indeed Lattimer (1998) postulated that colon contents often improve the identification of the prostate, delineating more clearly the degree of colonic compression by an enlarged prostate or other mass.

Tri-iodinated ionic compounds are recognised as contrast agents of choice (Holland 1993). Although residual urine and continued excretion will decrease the concentration of contrast where small volumes (1-4ml) are used (Feeney and others 1999), we considered that dilution of 1 volume of contrast to 2 volumes of water would give satisfactory opacification and reduce the risk of mucosal irritation (100mgI/ml).

In an attempt to maintain contrast in the urethra whilst the exposure is being made Park (1974) suggested a mixture of aqueous lubricant and contrast agent as a mean of increasing viscosity. However, Johnston and others (1983a,c) found that not only did this type of mixture fail to distend all areas of the urethra but also caused lesions in the bladder. Earlier Ticer and others (1980) obtained satisfactory results with a simple aqueous solution of contrast medium when the radiographic exposures was made at or near the end of injection.

Ticer and others (1980) felt that naturally occurring luminal closure of the membranous and penile urethra around the catheter prevented significant contrast leakage. Indeed several reports have suggested that balloon-tipped catheters may cause over-distension of the bladder and consequently acute and severe reactions in bladder and urethra (Barsanti and others 1981, Johnston and others 1983abc, Feeney and others 1984a). Digital palpation of the bladder during injection was used to judge the degree of distension in this study, as it has been shown that there is no precise method of gauging volume based on surface area or body weight (Johnston and others 1985). Indeed over-distension can cause thickening due to mild or moderate cystitis to disappear (Mahaffey and others 1989). Whilst satisfactory distension was achieved in 19/21 studies, in 2 studies distension, as judged by the subsequent film, was not good enough. This was due in study 1 to overestimating the degree of bladder distension, and in study 3 by the presence of a prostatic cyst masking the bladder.

The main filling defect found in the bladder (12/21) was the presence of air in the positive contrast, seen as coalescent or solitary bubbles at the periphery of the contrast

puddle. This feature needs to be distinguished from emphysematous cystitis where multiple coalescent gas pockets are identifiable within the bladder wall (Root and Scott 1971, Sherding and Chew 1979). Radiolucent calculi will also produce filling defects in the puddle but will be constrained by gravity to the centre or dependent part of the puddle (Park 1981). Rarely, calculi attached to the wall may be encountered in non-dependent parts of the bladder (Johnston and others 1986). Lastly, blood clots may be located anywhere within the lumen, but are more irregularly shaped and with a more indistinct border than calculi or bubbles (Park 1981).

The presence of filling defects in the urethra presents more of a diagnostic challenge, and in 12/21 studies air bubbles were identified as filling defects. These are usually smooth and round compared to calculi, which are often more irregular in shape with uneven margins (Burk and Ackerman 1996).

One of the goals of this series was to assess the bladder, prostate, prostatic and membranous urethra. To achieve this the degree of urethral distension is important. Ticer and others (1980) suggested that deposition of contrast medium directly into the prostatic urethra increases the likelihood of obtaining adequate luminal distension, but this means that the catheter is in the membranous urethra. Yet, Thrall (1994) concluded that size of urethral lumen, evaluated urethrographically, is of little importance in formulating a list of differential diagnoses as an enlarged prostate may make the prostatic urethra less distensible than normal. In contrast, Feeney and others (1984c), reported that the ratio between prostatic and membranous urethral diameters is the most clinically useful parameter in the evaluation of this area.

The normal urethrogram should outline a smooth, regular tube thorough the entire urethral length with slight narrowing at the ischial arch and the pelvic brim (Burk and Ackerman 1996). Of the 17 cases, in which the catheter tip was positioned just cranial to the *os penis*, the prostatic urethra was satisfactorily distended in 7 cases and visible although not entirely distended in another 7 cases, whereas in only 3 radiographs was the prostatic urethra invisible. When the injection of contrast was made in a more proximal position along the membranous urethra (4 cases), the prostatic portion was distended with the radio-opaque material. In all the cases where there was a failure to identify the prostatic urethra the bladder neck was intrapelvic. Though the reverse was

not true as evidenced by case 15, where a prostatic urethra was identified despite an intrapelvic bladder following a penile urethral injection of contrast.

Thrall (1994) took the view that the advantage of positive contrast retrograde urethrography was that it allowed evaluation of the peri-urethral symmetry of the prostate. He and other authors (Stone and others 1978, Feeney and others 1987a) found that asymmetry of the prostate was not specific for any type of disease, but was indicative of focal or regional, rather than diffuse, parenchymal disease. These authors reported that peri-urethral asymmetry was observed only in patients with neoplasia, parenchymal cavitation (abscess or non-inflammatory non-neoplastic cavitating disease) or paraprostatic cyst. Surprisingly in this series, asymmetry found in 7 cases, was present in 3 dogs with BPH.

In 7 of the 21 studies assessment of symmetry was not possible. In five the bladder neck was intra-pelvic. In the remaining two (19 & 21), despite a normally positioned bladder neck and satisfactory urethral distension, symmetry could not be assessed. One possible explanation is poor abdominal contrast due to insufficient abdominal fat (Park 1998), and specifically the triangular area of adipose tissue located ventrally between the prostate gland and the abdominal wall (Mahaffey and Barber 1992).

Prostatic size and its relationship to disease has been a matter of debate for sometime as a normal sized prostate does not allow identification of diseases such as infection, cystic change, or early/mild neoplasia (Barsanti and Finco 1986). Feeney and others (1987a) used the distance between the sacral promontory and the pubis to measure the ventro-dorsal and cranio-caudal dimensions of the prostate, using parallel and perpendicular axis respectively. They concluded that if either prostatic dimension exceeded 70% of the pubic-promontory dimension the prostate gland was considered to be enlarged. Furthermore, any dimension larger than 90% was considered highly suggestive of neoplasia, abscess or paraprostatic cyst. Lattimer (1998) found that the prostate may be as great as 20 or more times normal size, or the degree of enlargement may be minimal. In this series all cases of prostatic/paraprostatic cyst and tumour had observable prostatomegaly. However, only 2/5 cases of BPH had demonstrable prostatomegaly.

Reflux of contrast from the urethra into the prostate has been mooted as an indicator of disease. However, Ackerman (1983) postulated that there was no correlation between prostatic reflux and specific prostatic diseases. Feeney and others (1984b) noted

contrast reflux could be present in healthy dogs and that barium particles were observed within the secretory (acinar) portion of the prostatic parenchyma after positive retrograde urethrography, confirming that urethro-prostatic reflux (UPR) extended beyond the prostatic duct system that directly empties into the urethral lumen.

However, a later study (Feeney and others 1987a), showed that UPR, in non-neoplastic diseases was usually characterised by more rounded margins, with a tendency for the refluxed contrast medium to pool, whereas the reflux observed with neoplasia was usually characterised by an irregular (ragged) and massive coalescent appearance. Only 5 cases in this study showed UPR as a subtle cloudy blush in 4, and more irregular and massive in case 5 a prostatic tumour.

Neoplasia should be considered, regardless of prostatic size, when there is evidence of disruption or ragged appearance to the prostatic urethra (Feeney and others 1987a). However, prostatic urethral abnormalities, less marked, were also found in two cases of prostatic cyst (cases 3 & 13). On the other hand, the prostatic urethra was unremarkable in two other cases of cysts (10 & 17).

Definitive diagnosis of prostatic disease cannot be made by radiographic means. Radiographic signs must be interpreted in the correct context along with other historical, clinical and laboratory information. A definitive diagnosis of prostatic disease is frequently based upon histopathological examination (Thrall 1994). However, the introduction of ultrasound has made a major impact on clinicians ability to differentiate cystic from solid prostatic enlargement (Cartee and Rowles 1983, Feeney and others 1987b), and provides another method of prostate measurement (Atalan and others 1999).

The intention of this study was to combine two independent radiographic techniques and carry them out simultaneously in an effort to get a wide range of reliable information from a single radiographic exposure. The results from this study suggest that the information gathered concerning urethra and prostate is comparable with those achieved with individual techniques, though not obviously superior.

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# **SECTION 2**

# **1. Chronic Hypertrophic Pyloric Gastropathy**

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## **1.1 Introduction**

The pylorus/antrum is the distal functional area of the stomach. It acts like a pump to deliver digestive content, after been mixed with gastric secretions, to the duodenum (Wingfield 1981). In addition, the pylorus prevents reflux of duodenal contents (Sikes and others 1986).

Obstructive diseases of the pyloric canal may arise from intrinsic or extrinsic lesions. Intrinsic pyloric obstruction may result from a number of conditions such as muscular hypertrophy, neoplasia and eosinophilic granuloma diseases. Extrinsic causes include pancreatic or liver abscesses, neoplastic or inflammatory diseases, which may infrequently compress the pylorus causing delayed gastric emptying (De Novo 1989).

Chronic hypertrophic gastropathy has been described as a discrete syndrome because of the marked similarities in the historical, clinical, and pathological features of cases. It is considered to be the most common cause of pyloric outflow obstruction in the mature and older dog (Walter and Matthiesen 1993), being due to hypertrophy of the pyloric circular smooth muscle, hyperplasia of the pyloric mucosa or both (De Novo 1989).

Previous reports have used a variety of names for this syndrome because of uncertainty as to the cause and a lack of consensus. Names used include gastric polyps (Happe and others 1977), hypertrophic gastritis (Happe and others 1981), and chronic hypertrophic pyloric gastropathy (Walter and others 1985, Matthiesen and Walter 1986, Sikes and others 1986, Bellenger and others 1990).

Gualtieri and others (1996) described a group of young French Bulldogs, whose pedigree analysis showed that they were related, with a history of post-weaning chronic vomiting and histological changes of foveolar mucosal hyperplasia.

Most dogs affected are middle-aged or old, typically brachycephalic breeds such as Boxer or Boston Terrier where the lumen diameter of the pylorus is decreased due to circular smooth muscle hypertrophy (De Novo 1989). The male to female ratio has been reported as 2:1 (Matthiesen and Walter 1986, Bellenger and others 1990). A size predisposition - small dogs under 10kg- is recognised and breeds most likely to suffer are Lhasa Apso, Shih-Tzu, Maltese, Miniature Poodle, Pekinese or Yorkshire Terrier

(Walter and others 1985, Bellenger and others 1990, Walter and Matthiesen 1993). Although, there have been case reports in the Doberman Pinscher and Collie (Walter and others 1985), cats and horses (Walter and Matthiesen 1993).

A putative explanation for the high incidence of the syndrome in small breeds has been their excitable or vicious nature (Walter and others 1985). It has been speculated that neuro-endocrine disorders may contribute to the pathogenesis of the gastric outflow obstruction. Acute stress, inflammatory disease or trauma are believed to be able to stimulate the sympathetic tone in the stomach, causing decreased gastric motility and retention of gastric contents. When the pyloric antrum becomes distended, G cells in the antral mucosa are stimulated to produce gastrin, which in addition to stimulating the production of hydrochloric acid, has a potent trophic effect on both gastric mucosa and smooth muscle. Such effects over a prolonged period are believed to result in hypertrophy (Sikes and others 1986, De Novo 1989). Persistent gastric hyperacidity stimulates release of secretin and cholecystokinin, enteric hormones that are trophic to the pylorus and stimulate contraction contributing to muscular hypertrophy (De Novo 1989). Gualtieri and others (1996) noted that affected dogs were hypogastrinaemic and there were fewer G cells than expected in histological studies of the antrum. Another theory is that there may be decreased numbers of abnormally functioning myenteric ganglia cells and nerve fibres in the pylorus as occur in children with pyloric stenosis (Sikes and others 1986, De Novo 1989).

The types and form of inflammatory cell infiltration into the mucosa and submucosa have led some authors (Happe and others 1977, Walter and others 1985, Walter and Matthiesen 1993, Gualtieri and others 1996) to suggest that the disease may be immunomediated.

Most dogs have a history of post-prandial chronic intermittent vomiting over a period of several weeks to months (Matthiesen and Walter 1986). Abrupt projectile vomiting typical of gastric outflow obstruction does not occur commonly, and the content varies from undigested to partially digested food mixed with fluid and mucus, rarely containing bile (De Novo 1989).

Radiology is one of the most useful tools in the diagnosis of this disease (Sikes and others 1986, De Novo 1989). Plain survey radiographs may show a normal to markedly enlarged fluid or gas filled stomach. Barium studies confirm delayed emptying (Happe

and others 1981). A narrowed antrum or polypoid defects may be visible due to marked mucosal hyperplasia (De Novo 1989).

Endoscopy has proved effective in diagnosing acquired antral pyloric hypertrophy syndrome, but if contrast studies have been used, a fasting period of 24 to 48 hours before undergoing endoscopy is needed. Lesions observed include subtle circumferential narrowing of the pylorus, with large thickened mucosal folds involving 270 to 360 degrees of the pylorus and diffuse nodular masses of the distal antrum and pylorus. The mucosa is usually not ulcerated and insufflation with air fails to distend the pyloric canal (Fallin and others 1996).

Definitive diagnosis of chronic hypertrophic gastropathy is based on exploratory laparotomy and histological examination of full-thickness biopsies of affected tissue (Walter and Matthiesen 1993).

## **1.2 Case details**

### **History**

A nine year old male Staffordshire Terrier with 17.3 kilograms (kg) of body weight, was referred with a five week history of intermittent vomiting and diarrhoea. Initial treatment, by injection of penicillin/streptomycin followed with kaolin & neomycin tablets from the referring veterinarian was largely unsuccessful. However, the owners had noticed an improvement in faecal consistency.

Approximately three times daily post-prandial vomiting, of partially digested food mixed with “green bile”, occurred a few hours after feeding. Prior to vomiting, gagging and retching were observed. No other signs, except a slight degree of lethargy and increase in thirst, were reported by the owner.

The usual diet for this dog was based mainly on pork, lamb and beef, and an excess of chocolate treats.

### **Clinical examination**

The dog was bright, alert and with a nervous attitude. The only finding was a moderately tense abdomen on palpation.

## Ancillary aids

No significant abnormalities were detected in blood values, apart from a slight increase in cholesterol (7.1 mmol/l) and albumin (40 g/l) values and an equally moderate decrease in total globulin (23 g/l) and phosphate (1.05 mmol/l) values.

No abnormalities were found on initial plain films. Barium contrast studies showed retention of contrast in the pylorus and, although it could be seen in the small intestine after 40 minutes, there was no evidence of complete gastric emptying 2 hours post-administration. Barium was still present twenty hours later, with a mottled pattern in the fundus, probably due to feeding overnight (**Figure 1.1**). Two days post-administration, some residual contrast was still present in pyloric region.

Gastroscopy was performed under general anaesthesia. The oesophageal mucosa appeared reddened and the stomach was coated with a mixture of mucus and barium from the previous contrast study. A smooth non-ulcerated mass was observed in the pylorus (**Figure 1.2**).

## Surgery

The anaesthetic protocol consisted of intra-muscular (IM) pre-medication with 0.5 milligrams (mg) of acetylpromazine and methadone (3.5mg), intravenous (IV) induction with 200mg of thiopentone, followed by endotracheal intubation and maintenance of anaesthesia with a halothane/nitrous oxide and oxygen mixture.

The stomach was exposed via cranial midline laparotomy. The pylorus was markedly thickened circumferentially and a semi-mobile mass was palpable in the antral lumen. A *Heineke-Mikulicz pyloroplasty* with sub-mucosal resection of the mass was carried out. Using the pylorus as mid point a stab incision was made in the antrum and a 4cm longitudinal incision extended from the stab along the anti-mesenteric border of the duodenum. This incision exposed the lumen and the polypoid mass of 2x4 centimetres (cm) was resected from the mucosa (**Figure 1.3**). No attempt was made to close the mucosal defect. Two stay sutures were used to approximate the two ends of the longitudinal incision, which was closed in a transverse fashion using two layers of simple interrupted 2 metric polyglactin 910. The laparotomy was closed in a routine fashion.

Postoperative management consisted of amoxicillin/clavulanic acid 300mg three times a day (TID) IV, cimetidine 10mg/kg TID and continuation of fluid therapy during the post surgical fasting of 24 hours. Return to oral alimentation was delayed until the second day, with small soft meals four times a day for 7 days, before returning to a normal diet.

## **Follow up**

Initially the dog recovered well, but by the third day it started to vomit again. Fluid therapy was reintroduced and reinforced with metoclopramide 10mg TID subcutaneous (SC) and sucralfate 4 millilitres (ml) twice a day (BID).

Six days after surgery and due to persisting vomiting contrast studies were repeated, which showed gastric emptying but narrowing of the pyloric canal. Endoscopy revealed marked mucosal swelling, but no hindrance in passage of the endoscope into the duodenum. The dog was discharged with cimetidine 200mg four times a day (QID) and erythromycin suspension 0.7ml TID. A select protein diet, in small liquidised meals 6 times daily was also advised.

Four weeks later the owners reported an improvement in the signs, with only four episodes of vomiting. However, the dog was still on medical and dietary management. On endoscopic examination, there were several small mucosal folds at the pylorus indistinguishable from those found in normal animals.

## **Pathology**

The submitted tissue was classified as a benign lesion consisting of irregular gastric mucosal hyperplasia and dysplasia.

### **1.3 Discussion**

The history and lack of clinical features were suggestive of a pyloric outflow obstruction, though a gastric tumour could not be ruled out. However, gastric carcinoma classically presents with marked weight loss and polydipsia, in addition to vomiting on an empty stomach (Sullivan and others 1987). The lack of plain radiographic findings of pyloric distension and *gravel* stress the value of barium in determining gastric emptying. However, the failure to detect the polyp is probably

related to its small size and the ease with which barium can overwhelm small lesions on radiographs.

The post-surgical response of this case was not as good as expected and the findings on the second endoscopic examination raised some doubts over the choice of surgical technique. With appropriate surgical management, most of dogs affected should have complete resolution of their clinical signs and about of 80 per cent of cases reportedly have had an excellent clinical response on long-term follow-up (De Novo 1989). However, the endoscopic examination at 4 weeks post-surgery found what were considered normal antro-pyloric folds.

Sikes and others (1986) suggested a classification based in the histological origin (muscular or mucosal) and the exact nature of the changes (hypertrophy, hyperplasia, or aplasia) of the process taking place in the pylorus, in order to choose the most accurate surgical procedure. However, as the surgeon is not able to confirm these characteristics intra-operatively, the assessment of the outflow diameter, the amount of tissue to be excised and the perceived ability of the remaining tissue to expand should be the guide (Walter and others 1985, Matthiesen and Walter 1986).

*Heineke Mikulicz (HM) pyloroplasty* has been reported as one of the simplest and fastest gastric drainage procedures (Papageorges and others 1987a), used as treatment of choice for small isolated polyps (Walter and others 1985, Matthiesen and Walter 1986). In general all pyloroplasty procedures are designed to make the gastric outlet wider than the normal pyloric ostium (Van Sluijs 1992). Although easy to perform HM pyloroplasty may lead to failures in relieving outlet obstruction in cases with generalised mucosal lesions and/or muscular hypertrophy (Matthiesen and Walter 1986, Sikes and others 1986).

In dogs with a poor response to surgery, incorrect assessment of the degree of gastric outflow obstruction has usually been made. This indicates that a more radical procedure such as a *gastroduodenostomy (Billroth I)* should have been performed (Matthiesen and Walter 1986). This is done in the belief that radical resection of the pylorus and antrum with gastroduodenostomy is not associated with increased frequency of post-operative complications when compared with less radical procedures (Walter and others 1985).

Some authors, however, still prefer the most “*conservative*” technique and suggest that possible failures in achieving the surgical goals, can be avoided by checking the outflow diameter digitally through a separate antral stab incision (Sikes and others 1986). However, this requires a great deal of experience in this area, which is usually lacking due to the uncommon occurrence of this problem.

Taking the HM pyloroplasty as conservative and the Billroth I as a radical option, a compromise procedure has been described, the *Y-U pyloroplasty* (Bright 1998). In this procedure good exposure of mucosa occurs and a flap of antrum is advanced into the duodenum to enlarge the pylorus (Walter and Matthiesen 1993).

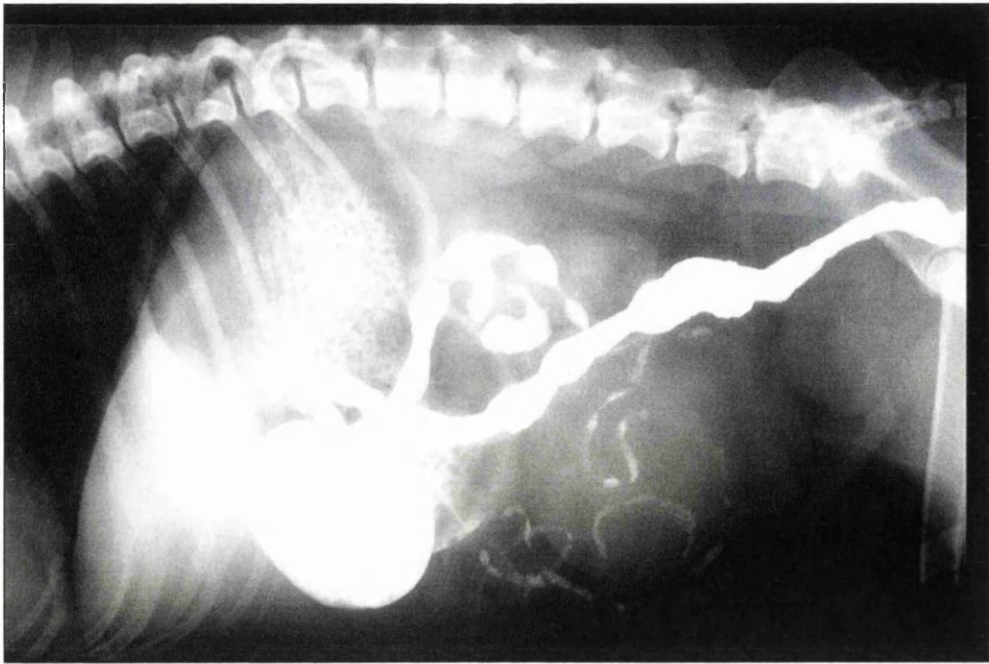
The post-operative complication that causes concern in any pyloric surgery is the stimulation of persist bilious vomiting due to alkaline reflux gastritis secondary to duodenogastric reflux. However, this usually responds to a 2 to 3 week course of metoclopramide, systemic antacids and sucralfate (Bright 1998). In studies comparing different gastric drainage procedures, a slightly increased frequency of enterogastric reflux on endoscopy was reported following HM pyloroplasty compared with normal dogs, although the volume detected was higher in other types of techniques (Papageorges and others 1987b).

The presence of marked muscle hypertrophy and post-operative intra-luminal swelling suggests that the most suitable technique was not chosen. However, despite the relative return to normality four weeks after surgery, a more radical surgical procedure such as a Billroth 1 or II should be carried out if the animal fails to thrive on a diet of normal consistency.

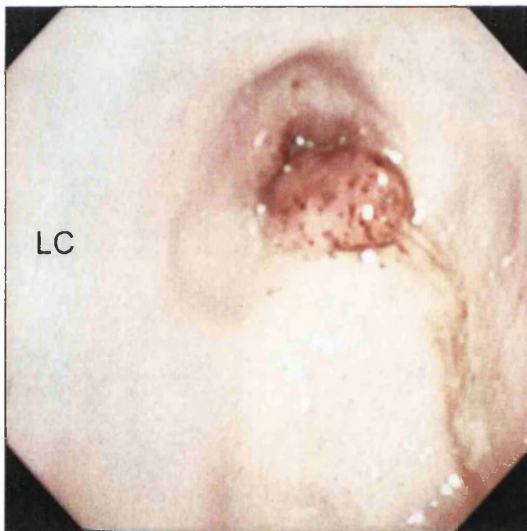
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**Figure 1.1** Chronic hypertrophic pyloric gastropathy, lateral abdominal radiograph. Twenty hours following barium administration there is still retention of contrast in the pylorus and antrum, which are dilated.



**Figure 1.2** Chronic hypertrophic pyloric gastropathy, endoscopy. The somewhat pedunculated polyp can be seen dangling from the pyloric mucosa. On the right is the lesser curvature (LC).



**Figure 1.3** Chronic hypertrophic pyloric gastropathy. The resected hypertrophied area measures 2x4cm.

## 2. Canine Inguinal Hernia

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### 2.1 Introduction

Herniation is the abnormal protrusion of an organ or tissue from the cavity where it normally lies (Read and Bellenger 1993). The exit may be a normal body opening (Dean and others 1998) or a defect in the body wall (Read and Bellenger 1993), thus defining *false* and *true* hernias respectively (Smeak 1993). True hernias have a peritoneal sac, whereas false ones do not (Parks 1981).

In inguinal hernias the protrusion occurs through the inguinal canal. This is a passage between the internal inguinal ring - limits of which are the caudal edge of the internal oblique, rectus abdominis and the pre-pubic tendon, and the external inguinal ring - a slit between pelvic and abdominal tendon portions of external oblique muscle (Ashdown 1963).

During the embryological stage of development the vaginal process, an evagination of peritoneum, passes through the inguinal canal. The vaginal ring, which permits communication between the lumen of the process and the peritoneal cavity, does not become obliterated, and persists throughout life in domestic animals (Ashdown 1963). Inguinal hernia may be congenital, though clinical signs may not develop until later in life (Read and Bellenger 1993).

Inguinal hernia may be considered indirect when abdominal contents enter the vaginal process, and less commonly direct when passage through the inguinal canal but adjacent to the vaginal process (Smeak 1993).

Hayes (1974) noted certain breeds, such as Pekinese, Cairn Terrier, Basset Hound and West Highland White Terrier, have a significantly increased risk of inguinal herniation. The increased prevalence in some breeds suggests a pattern of familiar inheritance, but it should not be assumed that all congenital hernias are heritable defects (Read and Bellenger 1993).

Males are more likely to present with congenital inguinal hernias than females, possibly because of delayed inguinal ring narrowing from late testicular descent (Fox 1963). Acquired inguinal hernias are seen primarily in intact females and often in association with oestrus or pregnancy, suggesting a hormonal factor (Parks 1981). It has been

suggested that sex hormones may change the strength and character of the connective tissue, enlarging the diameter of the inguinal rings (Smeak 1993). Thus during oestrus and pregnancy, the broad ligament and uterus are able to migrate into the inguinal canal (Parks 1981). Nevertheless, Waters and others (1993) described this disease in six spayed females, so the role of hormones remains unclear.

Parks (1981) suggested changes in intra-abdominal pressure (coughing, obstipation), metabolic disease (diabetes mellitus, hyperadrenocorticism) or nutritional muscular-aponeurotic deficiency of the abdominal wall leading to its weakening, as causative factors in herniation. In addition, obesity is a predisposing factor. Accumulation of fat encircling the round ligament causes a dilatation in the vaginal process and even of the inguinal canal itself (Ashdown 1963).

In general, when the protruding contents of the hernia can be easily returned to the cavity, the hernia is classified as *reducible*. If this is not possible, as a result of adhesion of the contents in their new location, it is *irreducible*. Irreducible hernias of hollow organs can be *strangulated*, which implies obstruction to blood flow through the organ (Read and Bellenger 1993).

Incarceration may also obstruct vascular supply to the herniated tissue, usually at the edge of the defect, and eventually result in necrosis due to strangulation. *Strangulated* hernias usually lead to engorgement, fluid and blood loss from the organ affected. Bacterial overgrowth, toxin production and a decrease in the effectiveness of tissue barriers to prevent bacterial invasion are common features in poorly perfused organs, leading to endo and/or exotoxaemia and septicæmia (Parks, 1981).

The hernia can contain more than one organ, and the contents may vary from time to time; most often organs in the immediate vicinity are found. However, those organs that are freely movable on long vascular pedicles can travel to fill the hernia and are prone to torsion (Parks 1981). Hernial contents include fat, omentum, intestine, peri-prostatic fat and uterus; also bladder and spleen have been reported (Dean and others 1998). However, fat and omentum are reported as the most common contents in canine inguinal hernia (Grier and others 1971). This may explain the usual asymptomatic nature and benign course, although omentum may become incarcerated, resulting in pain and depression, having been described some cases in males in which concomitant hydrocoele was present (Waters and others 1993).

The main clinical sign is the presence of a painless mass with soft doughy consistency, which may be bilateral but is more often left sided (Smeak 1993).

In 12 cases analysed by Waters and others (1993), in which hernias contained both small intestine and uterus, none became incarcerated. The presence of uterus within a hernia limits herniation of small intestine.

Incarcerated hernias have to be differentiated from other inguinal masses such as mammary tumours and cysts, lipomas, enlarged lymph nodes, abscesses and haematomas. Femoral hernias, due to their vicinity, are usually misdiagnosed as inguinal hernias. Femoral hernias are palpated caudal to the inguinal ligament and ventro-lateral to the pelvic brim, whereas inguinal hernias are cranial and medial to the pelvic brim (Smeak 1993).

## **Case Details**

### **History**

A five month old female English Cocker Spaniel (10kg body weight) was referred with a four week history of a right sided inguinal swelling. The swelling was intermittent initially, but had become permanent without causing any apparent signs of discomfort. The dog also presented with a persistent ocular watery discharge due to bilateral entropion that had been treated unsuccessfully with temporary Lembert sutures one month previously.

### **Clinical examination**

The dog was alert and responsive. On examination, a round, soft, fluctuant mass (approximate size 3x3x4cm) could be palpated in the right inguinal area (**Figure 2.1**). The consistency of the content was fatty, and attempts to reduce the hernia were unsuccessful. The left inguinal area appeared normal. A diagnosis of right unilateral inguinal hernia was made.

Ophthalmologic examination confirmed bilateral entropion causing corneal ulceration and watery discharge. Imperforate right nasolacrimal puncta and left micropuncta were also noted.

## Surgery

The anaesthetic protocol consisted of IM pre-medication with acetylpromazine (0.3mg) and methadone (2mg), IV induction with 137.5mg of thiopentone, followed by endotracheal intubation and maintenance with a halothane/nitrous oxide and oxygen mixture.

The dog was placed in dorsal recumbence and the herniated sac was exposed via a caudal midline skin incision. As the mass was dissected free of its adhesions the sac was inadvertently breached. The hernial content, mainly fat, was pushed back into the abdominal cavity.

The external inguinal ring was closed with pre-placed 2 metric nylon simple interrupted sutures, except for the most caudal aspect (**Figure 2.2**). The patency of the external pudendal vessels was checked.

The left side was examined and the ring was noticed to be slightly enlarged although no herniation was evident, so three sutures of the same type and material as described were placed. The wound was closed routinely.

The dog was discharged with chloramphenicol ointment TID and a seven days course of carprofen 20mg *per os* (PO), BID.

Postoperative management included restricted lead exercise for 4 weeks.

## Follow up

The dog was re-assessed a month after being discharged. The owners reported an uneventful recovery from the surgery and healing process. On palpation, no inguinal swelling was present, but some thickening due to fibrous reaction was evident.

At palpation, the closure of the defect could be noticed, and no inguinal swelling was present.

## 2.3 Discussion

Inguinal hernia has been described mainly in female middle-aged dogs (Dean and others 1998) or in young males (Waters and others 1993). This animal is unusual in that it is young and female. Thus the previously suggested contributory factors of hormonal

influence, pregnancy and obesity do not apply (Ashdown 1963, Parks 1981). No evidence of trauma was noted on exploration of the hernia (Read and Bellenger 1993).

Ventral midline incision was preferred as, even in simple cases, this approach allows visualisation of both inguinal rings and repair of bilateral herniation through a single incision. It also avoids the possibility of damage to the caudal superficial epigastric vessels and mammary tissue (Peedle 1980, Dean and others 1998).

Pre-placement of sutures to close the hernia was done to minimise the risk of damage to the external pudendal vessels and genitofemoral nerve, which exit from the caudo-medial aspect of the ring (Gourley and Gregory 1991).

The main post-operative complication is swelling, which may be caused by haematoma due to inadequate haemostasis during herniorrhaphy, or ensuing infection. Controlled walking soon after the surgery and limited exercise until suture removal are encouraged to decrease postoperative oedema, so that the prognosis for uncomplicated inguinal hernia repair is good (Smeak 1993).

Patients with large defects may require reinforcement of the primary hernia repair using an on-lay polyethylene mesh technique or a cranial sartorius muscle flap (Smeak, 1993). Due to the large dead space in large hernias excessive swelling can be avoided by means of caudal abdominal bandage and placement of drains (Waters and others 1993, Dean and others 1998).

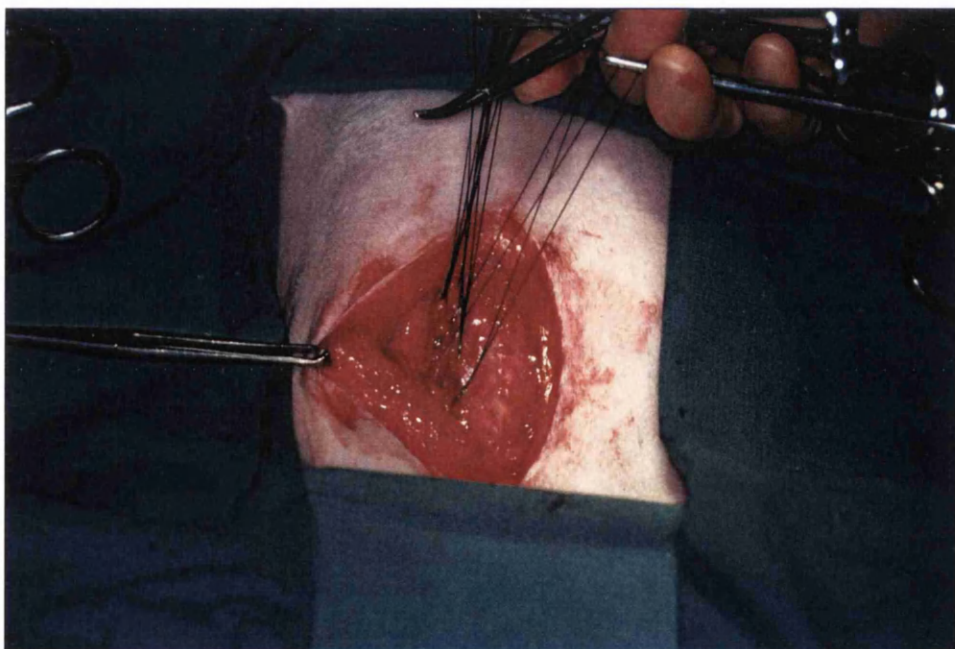
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**Figure 2.1: Inguinal hernia.** There is a marked inguinal swelling on the right with a more subtle one on the left (black arrow).



**Figure 2.2 Inguinal hernia.** The hernia is closed using pre-placed monofilament nylon sutures.

## **3. Bilateral Ectopic Ureter in a Male Dog**

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### **3.1 Introduction**

Ureteral ectopia is a rare congenital anomaly where one or both ureters fail to terminate normally in the trigone of the urinary bladder (Osborne and others 1995).

Embryologically, the metanephric ducts, the ureteric precursors, originate from the dorsal surface of the distal end of the mesonephric ducts. As the bladder develops from the urogenital sinus, the caudal end of the metanephric duct expands into the area that will eventually form the trigone, whereas the cranial end migrates forward to join the kidney. Seminal vesicles, vas deferens, and the prostatic urethra also derive from mesonephric ducts. The bladder neck originates in the urogenital sinus, therefore abnormalities in formation of this and the metanephric duct may lead to termination of ureters in locations other than the bladder trigone. The exact cause of ureteral ectopia remains unknown, though vitamin excess or deficiency during pregnancy have been suggested (Owen 1973a).

Ureteral ectopia may be also associated with other congenital abnormalities such as renal ectopia, hypoplasia or aplasia, ureterocoele, urachal remnants, agenesis or hypoplasia of the urinary bladder, urethral agenesis or ectopia and phimosis (Osborne and others 1995).

There is considered to be certain breed predispositions to ectopic ureter such as the Siberian Husky (Hayes 1984, Dean and others 1988, Stone and Mason 1990), Golden Retriever, Labrador Retriever, Sky Terrier (Holt and others 1982, Holt and Hotston-Moore 1995), miniature and toy Poodle (Smith and others 1981, Hayes 1984), New Foundland, West Highland White Terrier, Bulldog and Fox Terrier (Hayes 1984).

Dean and others (1988) and Johnston and others (1977) suggested a familial tendency when they observed the defect in Siberian Husky littermates. Conversely, Stone and Mason (1990) noted that three Siberian Husky females with ectopic ureter had a total of five litters with no observed incontinence. However, they did not to rule out heritability based on this observation.

The higher incidence of ureteral ectopia in females has been observed in different studies (Smith and others 1981, Hayes 1984, Stone and Mason 1990). Lane (1973)

the longer urethra and that the commonest site of ectopic orifice in this gender is proximal to the urethralis muscle. The sphincter action of this muscle could be expected to prevent incontinence.

As ureteral ectopia is a congenital anomaly, the associated urinary incontinence is usually recognised in young animals. However, in males the onset of incontinence may be delayed until adulthood, when urethral muscular tone declines, as some studies have reported (Lane 1973, Stone and Mason 1990). Therefore, although ureteral ectopia is the commonest cause of urinary incontinence in juvenile dogs (Holt 1990), it should be considered in the differential diagnosis of urinary incontinence in dogs of all ages (Holt and Hotston-Moore 1995).

Ureteric ectopia in males is most commonly recognised when they terminate in the prostatic urethra (Holt and Hotston-Moore 1995) and the cranial portion of membranous urethra (Osborne and others 1995), although vas deferens and seminal vesicles are other possible sites of termination (Dean and others 1988).

Independent of the termination site ectopic ureters may enter the bladder at the normal site on the dorsolateral caudal serosal surface, but continue through the bladder muscularis and submucosa, pass the trigone to an abnormal distal termination. These are defined as *intramural* ectopic ureters (Osborne and others 1995). These may open at the trigone in a normal fashion and then continuing submucosally to a second distal opening, so called *ureteral branching* (Dean and others 1988).

*Ureteral troughs* have been defined as intramural ectopic ureters that may open into the bladder in a normal or more distal location, but continue as an incomplete structure (trough) beyond the trigone into the proximal urethral sphincter (Stone and Mason 1990).

One of the commonest associated abnormalities is hydroureter and hydronephrosis (Smith and others 1981). The exact reason for this is unclear but may result from the partial obstruction of distal ureter owing to its abnormally long intramural course, abnormal ureteral peristalsis or ascending infection (Holt and Hotston-Moore 1995, Osborne and others 1995).

To diagnose ectopic ureter, the excretory urogram allows evaluation of renal function and side-by-side comparison of both, right and left, components of the upper urinary tract (Smith and others 1981).

Owen (1973b) preferred a large volume slow infusion technique because it can be used in patients with poor kidney function. The whole urinary tract is visualised at once and there is no need to compress the abdomen, which may distort normal anatomical relationships. Apart from obvious by-pass of the bladder one of the main findings is loss of the normal “J” shaped termination of the ureter as it enters the bladder (Waldron 1993). The exact site of the ectopic orifice may be difficult to locate due to masking by contrast material within bladder, poor renal excretion (Owen 1973b) or in cases of intramural ectopic ureter (Osborne and others 1995). Alternative techniques are retrograde contrast studies (Holt and others 1982).

No effective primary medical management is available for urinary incontinence caused by ectopic ureter. Surgical correction is the only viable option, and the choice of the procedure is based on the functional status of ureters, kidneys and the presence of concomitant abnormalities (Osborne and others 1995).

The creation of a *neostoma in situ* with ligation and transection of the distal portion is the procedure of choice for intramural ectopic ureter. *Ureterovesical anastomosis/ureteroneocystotomy* consists of ureteral transection and reimplantation (Stone and Mason 1990). *Ureteronephrectomy* is only indicated in cases of exclusively unilateral ectopia and when ureter and kidney are irreversibly damaged (Gregory 1996).

Branching distal extension is surgically corrected by occlusion with placement of simple interrupted sutures through serosa and muscularis and around the ureter. Closure of ureteral trough is made with synthetic absorbable suture material in a simple continuous pattern (Stone and Mason 1990).

## 3.2 Case details

### History

A one year male Border Terrier (8.5 kg body weight) was referred with a history of urinary incontinence since puppy hood. Constant urine dribbling was noticed, although the dog was able to produce a normal micturition stream, without signs of discomfort or

straining. The dribbling was worse when the dog was excited or asleep. Previous treatments with different courses of antibiotics and one of phenylpropanolamine syrup had failed to improve the condition.

### **Clinical examination**

The dog was alert and responsive. On examination regular urine dribbling was visible, hair surrounding the prepuce was stained with urine and the skin of the caudal abdomen was scalded.

### **Ancillary aids**

A blood sample was taken for haematology and biochemical studies. Increases in white blood cells ( $15.2 \times 10^9/l$ ), haemoglobin (18.3g/dl), albumin (39.0g/l), and glucose (5.8mmol/l) values were detected; whereas total globulin (24.0g/l) and phosphorus (1.11mmol/l) measurements were below the average range.

Biochemical analysis of urine revealed the presence of protein, specific gravity (SG) of 1.040 and pH 7.5. A large number of white blood cells and granular casts were found in the urine as well as the presence of cellular debris and large epithelial cells. Urine samples were also sent for bacteriological culture and sensitivity, but no bacteria were isolated.

Ultrasound revealed a dilated urethra, a grossly dilated left ureter with some left hydronephrosis (**Figure 3.1**). A double contrast cystogram combined with a retrograde urethrogram revealed a left enlarged ureter terminating in the prostate area (**Figure 3.2**).

### **Surgery**

The anaesthetic protocol consisted of IM pre-medication with acetylpromazine (0.2mg) and methadone (0.2mg), IV induction with methohexitone (3.2ml), followed by endotracheal intubation and maintenance of anaesthesia with a halothane/nitrous oxide/oxygen mixture.

Via a midline laparotomy, the bladder was exposed and two stay sutures (2 metric polyglactin 910) were placed in the apex for atraumatic handling. The left ureter was dilated entering the distal prostatic urethra and the right ureter terminated in a normal location.

A ventral cystotomy was performed and the dorsal trigone area inspected, but no effective urine flow was noticed from the right ureteral orifice. Gentle urethral occlusion caused bulging of the right ureteral intramural segment draining into the proximal prostatic urethra and a diagnosis of bilateral ectopia was confirmed.

Right ureteral ectopia was repaired by means of creation of a *neostoma in situ*. A small incision was made in the bladder mucosa directly over the ureter to expose its intramural portion. The ureter was incised at the level of normal ureteral opening and the proximal edges were spatulated and sutured to the bladder mucosa using a simple interrupted pattern (0.7 metric polyglactin 910). A 22 gauge catheter was placed through the neostoma into the distal portion which was occluded by placing sutures through serosa, muscularis and around the ureter. As the catheter was removed, the sutures were tied to occlude the ureteral lumen.

The procedure chosen for the left ureter was a *ureteroneocystotomy*. The ureter was ligated as close as possible to its termination in the urethra and transected just proximal to the ligature. After choosing a site for re-implantation, a stab incision was made in the urothelium and serosal surfaces at the anticipated ureteral entry and exit points. A tunnel was created by pushing a pair of haemostatic forceps through the bladder wall. The proximal end of the excised ureter was grasped by means of a stay suture attached to haemostatic forceps and passed through the new tunnel. The edges were spatulated and sutured to the bladder mucosa (0.7 metric polyglactin 910).

The cystotomy was closed using a simple interrupted pattern (2 metric polyglactin 910) and omentum was secured over cystotomy incision with the same type of suture material. The laparotomy was closed routinely.

### **Follow up**

The dog recovered well after surgery and when discharged was bright and alert although a wet bed was noticed postoperatively. On the second day post-surgery the dog went home with amoxicillin/clavulanic acid (100mg) PO BID for 4 weeks, and carprofen (20mg) PO BID for 5 days.

Four weeks post-surgery the owner reported a remarkable improvement. The dog only appeared wet sometimes and always when recumbent.

Treatment with phenylpropanolamine 1mg/kg body weight BID was proposed in order to improve urethral sphincter tone.

Six months later, the owner was contacted on the phone. Suggested treatment with phenylpropanolamine was carried out only for two days. The dog had maintained the degree of improvement reported after surgery but still had intermittent episodes of dribbling that were always present when excited. Bed wetting was also present, but the owner considered the degree of incontinence manageable without further treatment.

### **3.3 Discussion**

Dilation of the extramural ectopic ureter was evident from the radiograph and ultrasonographic studies, but the bilateral nature of the condition was only confirmed during surgery. Bilateral, rather than unilateral ectopic ureters, are known to account for 30-50% of cases (Osborne and others 1995, Rawlings 1998). The high incidence of bilateral involvement and our failure to recognise the second ureter highlights the difficulty of identification despite thorough examination.

In male patients with bilateral ectopia insufficient urinary bladder filling, which is associated with reduced size of the bladder, may prevent normal micturition. However, when the ureter opens into the urethra, distal urethral pressure and resistance are high enough to cause retrograde flow of urine into the bladder (Waldron 1993). This may permit bladder distension and normal micturition, in the presence of incontinence (Smith and others 1981)

To close the tunnelling ureter occlusion sutures were placed beneath the bladder mucosa so that the suture material would not be within the bladder lumen to act as a nidus for calculi formation (Stone and Mason 1990); for the same reason synthetic absorbable material should be used instead of non absorbable (Dean and others 1988). However, Gregory (1996) suggested that the distal suture should be of chromic catgut since this causes reaction and reduces the risk of re-canalisation, but Rawlings (1998) used nylon or polypropylene.

*Neostoma in situ* offers the advantages of using the normal submucosal tunnel length of the ureter to prevent reflux, while preserving ureteral blood supply and innervation which permits ureteral peristalsis (Rigg and others 1983).

Because of the potential hypoplastic bladder in cases of bilateral ectopia, a one layer appositional suture was used to close the cystotomy incision and omentum placed over it to prevent intra-abdominal urine leakage but maximise the bladder lumen as suggested by Waldron (1993).

Even though a better long-term result will be obtained when the ureters have some degree of peristalsis and are not extremely dilated, satisfactory results have been obtained in dogs in which markedly dilated ectopic ureters were re-implanted (Osborne and others 1995).

Fewer complications are reported after ureteronephrectomy than after ureteral transplantation, but it is generally considered preferable to salvage a viable kidney. The re-implantation of dilated ureters into the bladder may be associated with a certain degree of vesicoureteral reflux, which predisposes the patient to ascending infection of the kidney (Osborne and others 1995). However, postoperative infection of the urinary tract was described as an uncommon though serious complication (Holt and Hotston-Moore 1995).

Based on previous results in children, in which post-surgical bladder wall oedema led to distal ureteral stricture, Holt and others (1982), advised deferring surgery until the dog was at least 6 months old. Yet, McLaughlin and Miller (1991), concluded that, age at time of surgical correction had no effect on the rate of post-operative incontinence.

There appears to be a higher incidence of incontinence after corrective surgery if the urethra is involved, and it has been suggested that concomitant urethral sphincter incompetence may be the cause (McLaughlin and Miller 1991). The trigone area is richly supplied with alpha adrenergic receptors and their stimulation can increase the tone in this area (Rigg and others 1983).

Oestrogen can increase the response to endogenous alpha adrenergic stimulation (Dean and others 1988), but anatomical and correctable causes such as patent distal segment, or reduced bladder capacity, should be ruled out before prescribing lifelong alpha adrenergics such as ephedrine or phenylpropanolamine hydrochloride, or oestrogen therapy (Stone and Mason 1990).

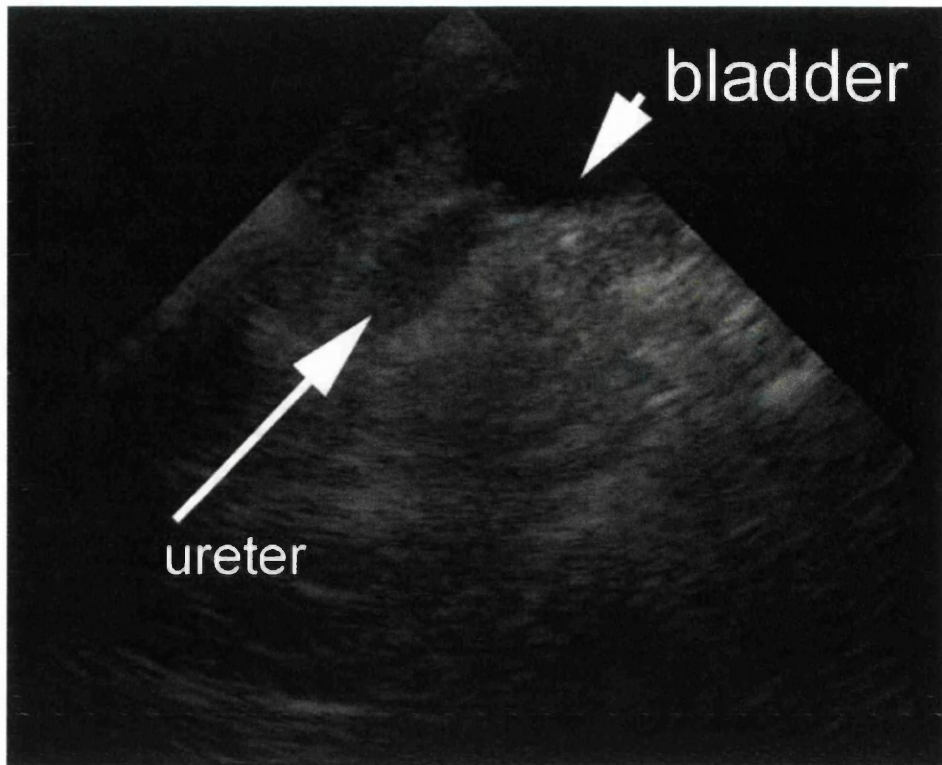
Other causes of surgical failure in correcting urinary incontinence might be distal remnant of an ectopic ureter traversing the bladder neck, thus interfering with normal

sphincter function, and a hypoplastic bladder unable to cope with the increased inflow of urine (Holt and others 1982).

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**Figure 3.1** Ectopic ureter, ultrasonogram. The dilated ectopic ureter is visible coursing close to the bladder.



**Figure 3.2** Ectopic ureter, lateral abdominal radiograph. The contrast can be seen to reflux from the prostatic urethra up a dilated ectopic ureter.

## **4. Traumatic Diaphragmatic Rupture**

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### **4.1 Introduction**

Diaphragmatic defects in cats and dogs are usually traumatic rather than congenital (Carb 1975). Typically, the defect results from indirect blunt trauma due to collision with motor vehicles (Sullivan and Lee 1989), although in a large number of cases there is no positive history of trauma (Garson and others 1980). Direct diaphragmatic injuries such as stab, gunshot or bite wounds are rare (Bellenger and others 1975).

During inspiration an increased positive intra-abdominal pressure is generated coupled with an increasing negative intra-pleural pressure. Upon coughing, straining and forceful muscular contraction high intra-abdominal pressures are generated. To offset this, the glottis closes producing a intra-thoracic positive pressure. Sudden pressure changes that lead to the glottis failing to remain closed may result in a critically high pleuro-peritoneal pressure gradient (Carb 1975). Boudrieau (1990) speculated that this gradient underlies the pathophysiology of traumatic diaphragmatic hernia (TDH). The specific area of the diaphragm on which the large pleuro-peritoneal pressure gradient focuses, and thus be most susceptible to rupture, depends to a great extent on the position of the animal (Carb 1975). However, tears usually affect the weaker muscular part of the diaphragm rather than the tendinous portion (Carb 1975, Garson and others 1980). In cats there is a higher incidence of circumferential tears (Garson and others 1980) and in some cases with muscles torn from the ribs allowing abdominal viscera to herniate into the ventro-lateral subcutaneous tissue (Wilson and others 1971).

The extent and location of the diaphragmatic rent, together with anatomical proximity and length of supporting ligaments or mesentery of individual organs, all influence the abdominal viscera that may herniate. Carb (1975) noted that any of the abdominal viscera may be involved. In a left tear the herniated organs are most commonly the stomach, spleen or small intestine. Whereas if right sided, small intestine and one or more lobes of liver herniate. The presence of liver is most frequently associated with ascites or hydrothorax (Wilson and others 1971, Garson and others 1980). However, unusual organ displacement such as the kidney or gravid uterus has been reported (Sullivan and Lee 1989). The complications associated with herniation, such as strangulation, obstruction and incarceration of abdominal organs are often due to

pressure applied by the edge of the diaphragmatic tear as the organs pass over it or result from the formation of fibrous adhesions (Johnson 1993).

The most common clinical signs are usually related to the respiratory system with varying degrees of dyspnoea, hyperpnoea and cyanosis due to the reduced tidal volume. The animal may be clinically normal, but subsequently develop signs of chronic respiratory disease such as intermittent chronic cough and fatigue following exercise (Wilson and others 1971). In some acute cases animals adopt a sitting or standing position with elbows abducted and head extended due to chest pain and in an attempt to lessen abdominal organ pressure on the thoracic structures (Carb 1975). Other signs such as anorexia, pallor, constipation, vomiting or abdominal pain have been reported (Garson and others 1980, Roe and others 1986). Cornell and others (1993) reported two cats with icterus due to extra-hepatic biliary tract obstruction subsequent to diaphragmatic rupture.

There is often a lack of abnormalities on physical examination. However, on auscultation abnormal heart sounds are found with muffling in the affected hemithorax (Wilson and others 1971). Thorax percussion may reveal hyper-resonance if a distended gas filled stomach is present, or dullness with liver herniation (Walker and Hall 1965). Borborygmus is not reliable and a *wasp waist* is not a common sign (Wilson and others 1971).

Wilson and others (1971) found that the greatest number of deaths occurred prior to surgical correction, with the critical periods being positioning for radiographs and induction of anaesthesia. The conclusion is that surgical repair should be delayed until cardio-respiratory function is stabilised. Furthermore, a compensated patient may become anoxic as a result of the stress of handling and restraint, as well as inappropriate radiographic positioning with the affected side downwards or for a ventro-dorsal view (Fagin 1989, Sullivan and Lee 1989).

Radiography is considered the most useful diagnostic aid (Johnson 1993). Frequent findings are loss of the diaphragmatic line (Fagin 1989) and masking of the cardiac shadow (Sullivan and Lee 1989). However, both of these may be difficult to detect in the presence of pleural effusion, which is a common sequel to TDH (Roudebush and Burns 1979). In this situation ultrasonography is useful because fluid is an excellent ultrasound-transmitting medium and allows detection of abnormally positioned viscera.

Although it is not always possible to detect gaps in the band of echoes adjacent to the liver that represent the diaphragm on the sonogram (Fagin 1989, Johnson 1993). Nevertheless, thoracocentesis allows improved radiographic detail and perhaps more importantly alleviates hypoventilation (Johnson 1993).

Due to the occurrence of false positives and negatives contrast radiography such as barium studies and positive or negative peritoneography are not a substitute for careful evaluation of plain films. (Rendano 1979, Stickle 1984, Sullivan and Lee 1989).

## **4.2 Case details**

### **History**

A one year old male domestic short-hair cat (4.1kg body weight) was presented with a history of road traffic accident in the previous 24 hours. The owners had noticed that the cat was slightly depressed and was not able to extend the left front leg.

### **Clinical examination**

Palpation of extremities showed a fracture in the left humerus and an abnormal rotation of the right hock, which also appeared swollen and painful. Pulmonary auscultation was loud and harsh. No other abnormalities were detected.

### **Ancillary aids**

Blood samples taken for haematological and biochemical analyses showed no significant abnormalities.

A lateral radiographic view of the thorax showed a fat dense mass lying caudo-ventrally in front of and overlying the diaphragm, which appeared to be confluent with falciform fat. Left humeral and right distal tibial fractures were also identified.

### **Surgery**

The anaesthetic protocol consisted of IM pre-medication with 0.15mg of acetylpromazine and 0.8mg of methadone, followed by IV induction with 30mg of thiopentone. The maintenance of anaesthesia after placement of an uncuffed 5.5 endotracheal tube was done with a halothane-oxygen mixture. Assisted ventilation was established by means of a mechanical ventilator.

Midline laparotomy was performed from a point immediately caudal to the xiphoid to the umbilicus. A circumferential diaphragmatic tear at the costochondral junction was exposed with displacement of omentum through it. The omentum was gently retracted allowing examination of the lungs and heart but no further abnormality was noticed.

The defect was closed with pre-placed horizontal mattress sutures of 3 metric polypropylene. The sutures incorporated the ribs and intercostal muscles. Prior to abdominal closure the diaphragm was checked for hidden tears. An intra-thoracic drain was placed and 2ml of fluid and 10ml of air were removed (**Figure 4.1**). The drain was not left in place after definitive surgical closure.

The spleen showed some signs of severe traumatic congestion in its cranial portion, so the affected area was removed after clamping and vascular ligation (2 metric polyglactin 910)

The laparotomy incision was closed routinely.

### **Follow up**

Treatment with amoxicillin/clavulanic acid (50mg TID) and carprofen (10mg BID) was instituted, as a continuation of the peri-operative management.

Hourly monitoring of respiratory and cardiac rate as well as temperature and capillary refill time was done during the first 12 post-operative hours. The cat, bright and eating, was transferred to the orthopaedics service for fracture repair three days later.

### **Discussion**

Walker and Hall (1965) were of the opinion that the prognosis for TDH must be guarded. However, provided each case was assessed and treated on its own merits, a satisfactory repair could usually be achieved safely through a simple surgical procedure. More than thirty years later this statement is still valid. The real risk is inappropriate timing of surgery in a patient that has not been stabilised, with compromised respiratory and circulatory function, due to shock and direct lung compression caused by herniated organs (Bellah 1998). In this cat, the respiratory signs were minimal and the general state of the animal did not require an emergency surgical procedure.

Apart from the surgical repair, anaesthesia for TDH is a challenge as there is a need for intermittent positive pressure ventilation, and nitrous oxide should be avoided since its

solubility in blood allows this gas to accumulate in the pleural space resulting in further lung compression and the possibility of diffusion hypoxia at recovery. This will worsen any initial hypoxia (Johnson 1993). In addition, the diffusion of gas into displaced stomach or intestinal loops could increase lung compression (Bellah 1998).

Assisted respiration was immediately instituted following induction with positive pressure not exceeding 20cm to ensure adequate oxygenation, yet prevent barotrauma with consequent development of re-expansion pulmonary oedema after over-inflation of atelectic areas. The latter effect was found to be one of the main reasons for mortality (Garson and others 1980, Bellenger and others 1996).

The placement of a chest drain was done to eliminate air and fluid that may complicate the post-operative recovery period, but no attempts were made to keep the thoracic drain device after the surgery as indicated by Sullivan and Reid (1990) when repairing diaphragmatic tears in cats.

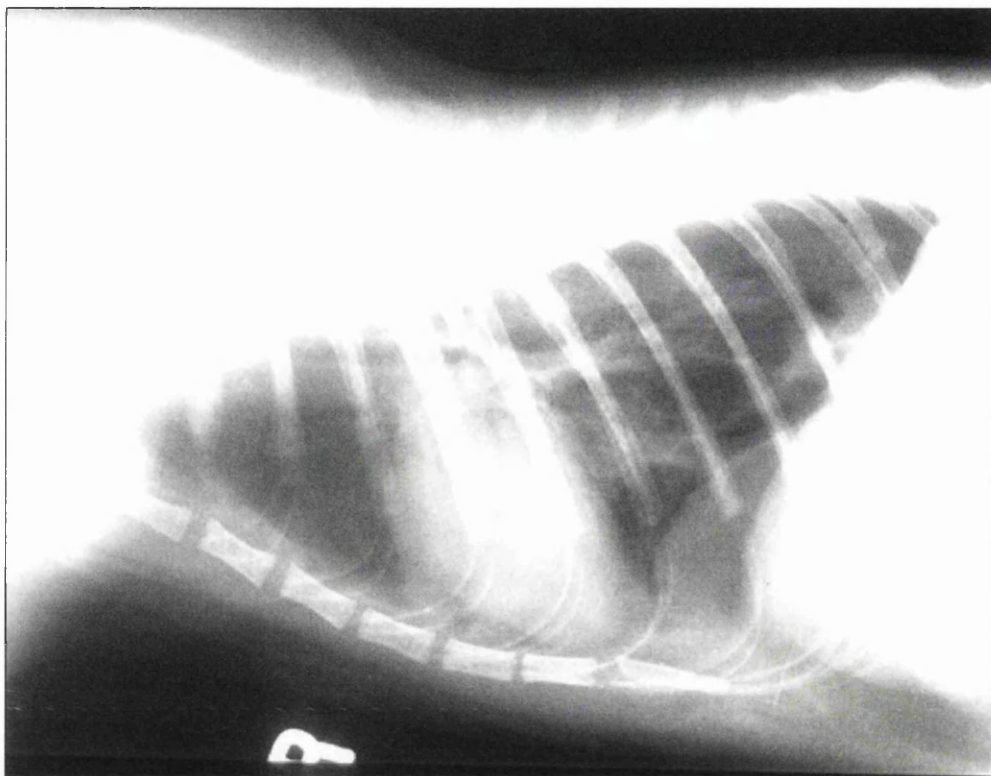
As indicated by Boudrieau (1990) the damaged portion of spleen was resected *in situ* without allowing re-establishment of the compromised splenic circulation in order to prevent toxæmia, which can result from bacterial endotoxins and exotoxins as well as products of tissue autolysis.

The tear in this case was circumferential as has been reported previously as common in cats (Garson and others 1980). The choice of suture material and pattern appears to be a matter of strong surgeon preference. Sullivan and Reid (1990) preferred silk. Johnson (1993) was of the opinion that a continuous suture of polyglactin 910 should be used as this avoided the stiff, sharp, cut ends of knotted polydioxanone sutures possibly injuring the abdominal viscera. However, our choice of polypropylene was based on the fact that this material is recommended for general herniorrhaphy (Bellah 1998).

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**Figure 4.1** Diaphragmatic rupture, lateral radiograph. Following surgery the diaphragmatic line is intact and normal aerated lungfields are visible.

## 5. Canine Nasal Aspergillosis

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### 5.1 Introduction

Mycosis has been described as one of the causes of chronic rhinitis/sinusitis in the dog (Bright 1981). The agent is generally *Aspergillus fumigatus*, which causes a destructive form of rhinitis (Sullivan and others 1986). Occasionally, *Aspergillus nidulans*, *Aspergillus niger*, *Aspergillus flavus* and *Penicillium spp* are incriminated. However, all are normal nasal microflora and as they cause a similar disease process they are grouped under the general term of nasal aspergillosis (Sharp 1989).

Pathogenesis remains unclear, only a few cases present with a history of cranial trauma or tumour. Cranial surgery that might have left a bone sequestrum to act as a nidus for infection is very uncommon. Immunosuppression, often seen in this disorder, has not been confirmed to be a cause or result from it, taking into account that canine cancer patients do not seem predisposed to nasal aspergillosis (Bright 1981, Sharp 1989).

Sharp and others (1991a) suggested that farm dogs might be more susceptible to this disease due to the large amount of spores that exist in hay or straw. These authors also reported a higher incidence of the disease in non-brachycephalic breeds, specifically Collies and Golden Retrievers, between one and seven years of age. A later study suggested that male dogs were at greater risk than females (Wolf 1992).

Bright (1981) described the action of the fungus as causing an initial mucosal and submucosal influx of neutrophils, macrophages, eosinophils and plasma cells, followed by proliferation of fungal hyphae that invade and occlude the vessels of the area. The body attempts to contain the invasion, which results in the formation of a dense fibrous layer throughout the submucosa with loss of normal epithelial and submucosal structures. A chronic irritation with resultant exudate production is the cause for the copious nasal discharge and chronic sneezing seen clinically. The discharge is sanguino-purulent and one of the most frequent clinical findings but is not characteristic (Sullivan and others 1986, Sharp and Sullivan 1989). Consequently, nasal foreign body, neoplasia, dental disease and chronic idiopathic rhinitis should be included in the list of differential diagnosis (Sharp and others 1991a). Where frank epistaxis occurs systemic causes such as coagulopathy should be considered (Sharp 1989). Nasal discharge may be absent in a few cases where infection is restricted to the frontal sinus (Sharp and

others, 1991a) and when affected may also show chemosis (Sharp 1989), exophthalmos (Wolf 1992), blepharospasm and epiphora (Sharp and others 1993) due to extension of fungal infection into soft tissues around the orbit.

Ulceration of the rhinarium and signs of discomfort around the nostrils or bridge of the nose are significant clinical signs (Sharp and Sullivan 1989).

The non-specific nature of clinical signs require ancillary aids for definitive diagnosis with radiology being the single most useful aid for this purpose (Sullivan and others 1986). The radiological features include turbinate loss producing rostral radiolucency, whereas a mixed density pattern may be observed caudally.

The rostro-caudal view is especially useful in the evaluation of the frontal sinus, which should not be overlooked as far as treatment is concerned. The features include opacity of the sinus, as well as thickening due to periosteal reaction stimulated by fungus, and a “*moth-eaten appearance*” or mottling of the frontal bone due to the osteolytic effect of the fungus or its toxins (Sullivan and others 1986).

If available, computer assisted tomography (CT) or magnetic resonance imaging (MRI), provide detailed definition of intranasal structures and lesions particularly in the ethmoid region (Wolf 1992).

Rhinoscopy is well suited to the investigation of aspergillosis due to the large air space created by the destructive rhinitis. Fungal colonies grow on the turbinate mucosa as greenish-white plaques having a “*white jam mould-like*” appearance (Lane and Warnock 1977, Sullivan 1987).

Direct smear and culture of intranasal swabs samples are unreliable in the diagnosis of nasal aspergillosis (Lane and Warnock 1977). Moreover 40% of normal dogs, and those with nasal neoplasia can also yield this fungus on nasal culture (Sharp and Sullivan 1992). A more accurate, but specific test, is serology. The false positive rates for the various assays such as ELISA, counter immuno-electrophoresis and agar gel immunodiffusion are approximately 15% (Sharp 1989).

Consequently, Sharp and Sullivan (1992) established the diagnostic criteria for nasal aspergillosis involving radiology, rhinoscopy, mycology (culture, cytology and histopathology) and serology, indicating that at least 2, but preferably 3 criteria should be satisfied for diagnosis.

Results in the trials carried out with systemic therapy showed that drugs like thiabendazole, ketoconazole or fluconazole tend to be effective in only 43-47% of cases (Harvey 1984, Sharp and Sullivan 1989, Sharp and others 1991b). The use of itraconazole has been also suggested as effective agent with few side effects (Legendre 1995).

Topical therapy with enilconazole by means of indwelling tubes implanted through trephine holes in the frontal sinus, to lie in the mid-nasal chamber has proved to be a successful treatment with a relatively low cost and few side effects (Sharp and Sullivan 1986).

The necessity for week long treatment generally requires hospitalisation. Treatment with topical clotrimazole 1% has been described recently by means of a single non invasive intranasal infusion with one hour contact time (Burbidge and others 1997, Smith and others 1998).

## **5.2 Case details**

### **History**

A ten year old neutered female Old English Sheepdog (23kg body weight) was referred with a 2 month history of sneezing and nasal discharge which had progressed from watery to thick sanguinopurulent secretion. The owner had noticed an increasing dullness and a marked pain if the nostrils were touched, which was also evident when the dog chewed food.

### **Clinical examination**

Detailed examination of the nose revealed discomfort on nasal palpation and an obvious yellow-green, thick bilateral nasal discharge but with marked discoloration of the left nasal sill (**Figure 5.1**). An airflow test was positive and the left submandibular lymph node was noticeably enlarged.

### **Ancillary aids**

Blood samples were taken for serological, haematological and biochemical studies. Increases in MHCH (36.7g/dl), white blood cells ( $17.6 \times 10^9/l$ ), neutrophils ( $14.08 \times 10^9/l$ )

and chloride (118mmol/l) values were found, whereas platelets ( $195 \times 10^9/l$ ) and albumin (23g/l) were found to be lower than normal.

Radiographic films were taken of the skull using dorso-ventral intra-oral and rostral-caudal views. There was marked turbinate destruction on the left with an increased radiolucency, similar but less severe change is evident on the right with some thinning of the vomer/septum (**Figure 5.2**). The left frontal sinus was nearly completely opacified, and the right to a lesser extent (**Figure 5.3**).

Rostral endoscopy was performed and the presence of yellow-grey fungal plaques were confirmed in both nasal chambers (**Figure 5.4**).

## **Surgery**

The anaesthetic protocol consisted of IM premedication with acetylpromazine (0.8mg) and methadone (5mg), IV induction with thiopentone (200mg), followed by endotracheal intubation and maintenance of anaesthesia with a halothane/nitrous oxide/oxygen mixture.

The dog was placed in sternal recumbence and the pharynx was packed with a saline moistened gauze. A 2cm skin incision was made over the frontal sinus off-midline. The underlying muscle was separated to expose the periosteum. Using a 5mm osteotome and a hammer, windows were opened bilaterally at the centre of a theoretical triangle formed by skull midline, frontal crest and supra-orbital rim. After the fungal-filled frontal sinus was emptied, a tube was passed through the stoma towards the ostia and into the nasal chamber. A second tube was placed but terminated in the frontal sinus itself. Two similar tubes were placed contra-laterally.

The skin was closed routinely. Polypropylene tubes were secured with mattress sutures and Chinese snare (Nylon 3 metric) before anchoring them to the skin behind the ears with two butterfly tapes. Finally, an Elizabethan collar was placed prior to recovery from anaesthesia to prevent patient interference with the tubes.

Starting the following day, enilconazole was administered at a dose of 10mg/kg twice daily for seven days. The stock solution of the drug (100mg/ml) was drawn into a syringe and mixed with an equal volume of saline. Total volume was divided equally between both chambers, taking care to position the dog's nose downwards during flushing. The drains were removed after treatment was completed and the dog was

discharged. Clinical signs had almost completely resolved by the end of treatment, with only a slight degree of residual sneezing and occasional nasal discharge, but not as thick as at the initial presentation.

### **Follow up**

The dog was assessed five weeks later, and still remained asymptomatic, thus the owner declined further rhinoscopic and radiological exams for a more accurate evaluation of the nasal cavity.

### **Discussion**

Enilconazole is a highly effective agent against *Aspergillus fumigatus*, *Penicillium Sp.* and several species of *Dermatophytes*, having its activity in the vapour phase, which enhances its penetration at the level of the nasal cavity. Side-effects are only related to the gastrointestinal system, but the drug is not absorbed after oral administration (Sharp 1989). Occasionally profuse salivation for a short time after administration and mild inappetance have been noted, probably due to the head being more horizontal allowing pharyngeal run-off. However, these changes are not associated with serum biochemical or haematological abnormalities (Sharp and others 1993).

The emulsion formed when enilconazole is mixed with saline can solidify within 5 minutes of preparation (Sharp and others 1993). Richardson and Mathews (1995) concluded in their studies that some areas of the nasal cavity were not contacted with medication and suggested that with this type of administration, remaining septal structures separating different compartments of the rostral sinus, might leave some affected areas untreated.

Other suggested reasons for enilconazole treatment failure are fungal resistance or fungal infection extending to the peri-orbital structures. Where the latter problem arises a combined topical with a systematically active antifungal agent therapy is advocated such as ketoconazole (Sharp and others 1993) or itraconazole (Legendre 1995).

Drain dislodgement is recognised as the most frequent complication of topical therapy (Sharp and others 1993). Richardson and Mathews (1995) suggested that catheter placement through the ostium may result in impaired sinus drainage, due to granulation

tissue formation, particularly if catheter dislodgement occurs frequently, necessitating multiple replacements.

Open treatment has been mentioned as alternative as a primary approach to nasal aspergillosis or for cases that are unresponsive to previous medical management, excising the diseased turbinate bones and removing the affected lining of the involved frontal sinuses (Pavletic and Clark 1991). In this case, the therapy with enilconazole followed the aforementioned technique, thus combining the advantages of surgical and medical treatments.

A single non invasive intranasal infusion of clotrimazole is another option in aspergillosis treatment, with side effects of mild pharyngeal oedema, ptialism and sneezing during recovery and post-anaesthetic vomiting (Caulkett and others 1997). To prevent more serious complications such as aspiration pneumonia or leakage into gastrointestinal system, the nasopharynx needs to be packed off with Foley catheters and sponges. In addition, if the cribriform plate is breached the drug could have access to the central nervous system (Mathews and others, 1996). The authors concluded that this single dose administration of clotrimazole allowed more consistent distribution of medication in nasal cavities and sinuses than the enilconazole technique, although Bray and others (1998) reported a poor therapeutic efficacy using the non-invasive technique with 10% solution of enilconazole.

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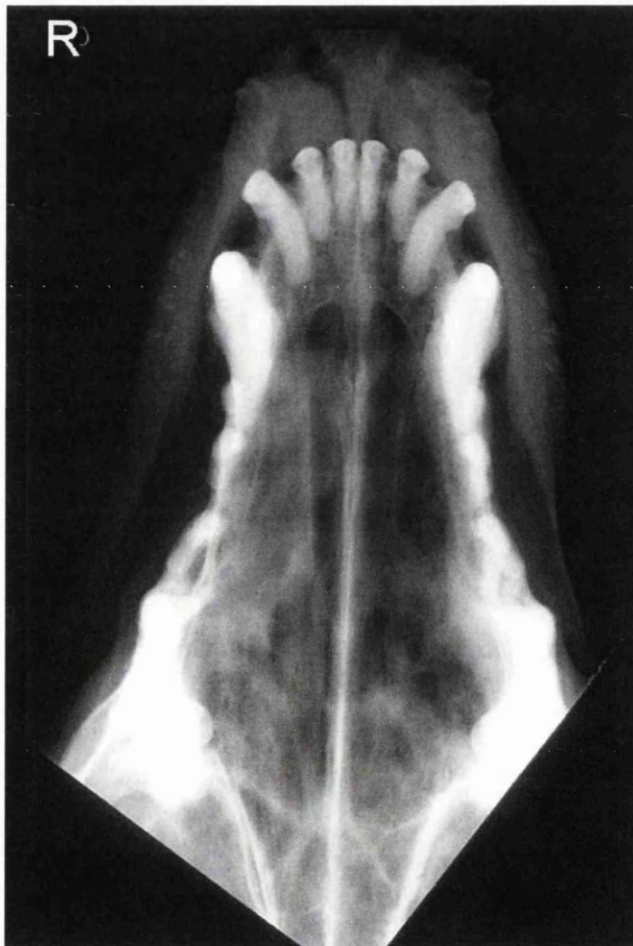
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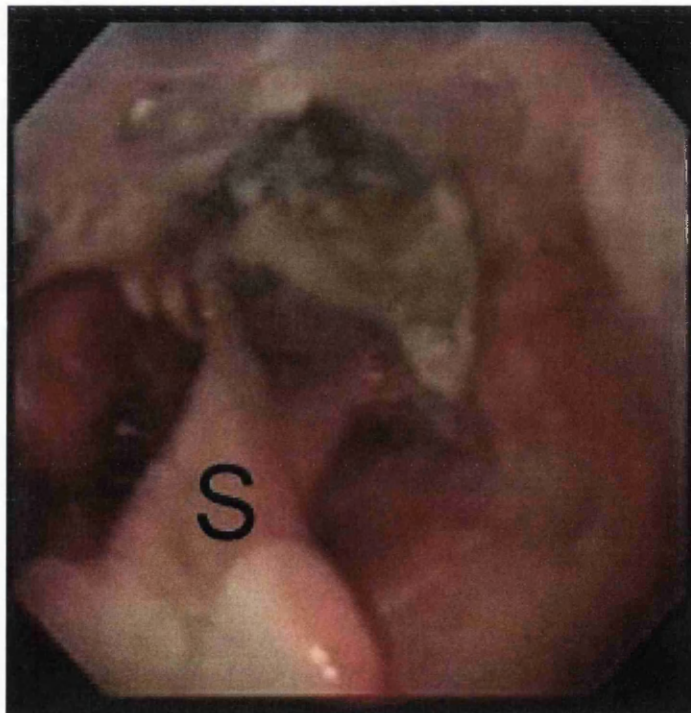
**Figure 5.1** Nasal aspergillosis. There is profuse discharge present and ulceration of the nasal sill is evident.



**Figure 5.2** Nasal aspergillosis, dorso-ventral intra-oral radiograph. There is marked turbinate destruction on the left rostrally and extending into the caudal portion of the nasal cavity. The vomer/septum is thinned and there is a degree of turbinate destruction on the right with increased radiolucency.



**Figure 5.3** Nasal aspergillosis, rostro-caudal frontal sinuses. There is near complete opacification of the left frontal sinus, with the right affected to a lesser degree.



**Figure 5.4** Nasal aspergillosis, rostral rhinoscopy. There is extensive turbinate destruction and a large fungal plaque is visible. The septum (S) has been partially destroyed so that the right nasal cavity is visible to the left.

## **6. Upper Airway Obstruction in a Brachycephalic Dog**

### **6.1 Introduction**

The brachycephalic head shape is the result of an inherited developmental defect in the bones of the skull base, which grow to a normal width but reduced length. The soft tissues of the head are not proportionally reduced and often appear redundant. These anatomic exaggerations result in increased airflow resistance and a bigger inspiratory effort, causing serious abnormalities in airway function (Hedlund 1998).

The wing, the dorso-lateral portion of the nares, is the most mobile part of the nares because it is covered by the levator nasolabialis muscle which is responsible for the increased diameter of the nostrils on inspiration. In some brachycephalic breeds the wing collapses medially, causing a degree of nasal occlusion that if severe, causes inspiratory distress with open mouth breathing. It also interferes with normal olfactory function, as well as the warming, moisturisation and filtration of inspired air (Lammerding and Howard 1981). Stenotic nares are rarely present alone. In a study of 28 cases of stenotic nares, overlong soft palate was also diagnosed in all these dogs (Harvey 1982a).

The normal soft palate, usually extends 1 to 3 mm caudal to the tip of the epiglottis (Hedlund 1998). Elongated soft palate is a condition found primarily in the Poodle, Cocker Spaniel, Beagle and all brachycephalic breeds (Bright 1990). English bull dogs are significantly over-represented and younger at the time of surgery compared with other breeds (Harvey 1982b).

Although Harvey (1983) found the most common components of brachycephalic airway obstruction were stenotic nares and overlong soft palate, there appeared to be two groups of animals. Young dogs tend to have stenotic nares and overlong soft palate often with no laryngeal abnormalities. In contrast, older dogs tend to have a worse prognosis due to the presence of an overlong soft palate and laryngeal disease, despite the absence of nasal stenosis (Harvey 1982c).

Bright (1990) reported that stenotic nares interferes with laryngeal function, drawing the already overlong soft palate deeper into the larynx, in a way that the “*To and Fro*” movement of respiration, will eventually cause inflammation, oedema and increased

thickness not only of the palate but also the lining of the laryngeal inlet. Indeed, Butler (1974) had suggested stenotic nares as an important factor in the development of airway obstruction that might be avoided with early surgical correction during puppy-hood.

The glosso-epiglottic mucosa is voluminous and, even in normal dogs, it can be easily lifted away from the epiglottic cartilage and the base of the tongue. It can be displaced into the *rima glottidis* in dogs with chronic inspiratory dyspnoea thus causing further impairment and triggering an increase in inspiratory effort (Bedford 1983)

Sudden presentation of brachycephalic dogs with acute signs of respiratory distress, collapse or cyanosis is very common despite the lifelong presence of predisposing anatomy. In such a compromised respiratory system a small additional load can result in acute de-compensation. This may be caused by exogenous factors (increases in heat and humidity, exercise, restraint, or sedation) or endogenous ones such as fever, respiratory infection, neuromuscular disease and abnormal chest or abdominal contents (Hendricks 1992).

Cooling of the patient and oxygen therapy, corticosteroid administration or other relevant drugs, may sometimes be necessary before a thorough examination or further diagnostic tests can be done for co-existing abnormalities (Hobson 1995, Hedlund 1998).

The most common indication for surgery is the presence of respiratory distress (Harvey 1983). In very mild cases, or when owners are not keen on surgical intervention, treatment of exacerbating factors should be carried out. A single dose corticosteroid treatment (0.5 to 2mg/kg prednisolone IM/IV) to reduce pharyngeal or laryngeal oedema or inflammation can relieve temporary swelling (Hendricks 1992).

## **Case details**

### **History**

A six year old neutered female Pug (9kg body weight) was referred with a 18 month history of gradual onset respiratory distress. The first sign noticed by the owners was a cough that seemed related to feeding and associated with retching and choking. The symptoms had worsened gradually, and lately, the owners reported noisy respiration even when the dog was relaxed, but which worsened with exercise, excitement or heat.

This resulted in exercise intolerance, large amounts of salivary foam and some episodes of collapse. Treatment with corticosteroids had slightly improved the condition but never cleared it up.

### **Clinical examination**

The dog had a marked harsh inspiratory noise located in the upper respiratory tract with episodes of coughing/choking, which were linked with a slight degree of lingual cyanosis. Circling movements, suggestive of respiratory struggling were also evident. The nostrils were markedly narrowed slit-like and there was no evidence of alar fold movement.

### **Ancillary aids**

Under general anaesthesia, endoscopic examination confirmed the presence of an elongated and hypertrophied soft palate, as well as transient displacement of the glosso-epiglottic mucosa during inspiration.

### **Surgery**

The anaesthetic protocol consisted of IM pre-medication with acetylpromazine (0.3mg), glycopyrrolate (0.1mg) and pethidine (25mg), IV induction with propofol (50mg), followed by endotracheal intubation and maintenance of anaesthesia with a halothane/nitrous oxide/oxygen mixture. Prednisolone (0.5mg/kg) was also administered peri-operatively IV.

The dog was placed in sternal recumbence and a gag was inserted to hold the mouth open. A surgical frame was used to fix the head upwards and pharyngeal sponges were placed to avoid the risk of aspiration.

Bilaterally, a wedge of rhinarial epithelium and cartilage of each alar fold was removed by means of two connecting incisions, rostro-lateral and caudo-medial respectively. Epithelial edges were closed with 1.5 metric polyglactin 910 using a simple interrupted pattern.

Allis tissue forceps were used to draw the soft palate rostrally, and the caudal border of tonsils was considered as a guide for the level of resection, such that the soft palate overlapped the epiglottis slightly (**Figure 6.1**). Stay sutures of 2 metric polyglactin 910 were placed just behind the proposed line of resection. One half of the width of the

palate was excised, followed by apposition of the oropharyngeal and nasopharyngeal layers of mucosa in a continuous pattern. When the midline was reached, the remaining palate was incised, the suture continued and finally tied.

The excess glosso-epiglottic mucosa was drawn from beneath the epiglottis and resected with Metzenbaum scissors from its dorsal point of attachment on the lateral epiglottis to its point of reflection on the dorsum of the tongue.

### **Follow up**

The dog recovered uneventfully from anaesthesia. Immediate improvement was noticed during the post-operative period, with reduction of inspiratory noise. The dog was discharged two days after surgery.

In short-term telephone follow-up after five weeks, the owner reported that the dog had not had any episode of collapse and was able to exercise as before the respiratory problem started.

### **Discussion**

Postoperative oedema and swelling are major concerns regardless of the specific technique used in soft palate resection (Clark and Sinibaldi 1994). Harvey (1982b) considered that despite the fact that electrocautery caused far more postoperative oedema than scalpel resection, the use of prednisolone pre- and post-surgery minimised that risk and permitted better control of haemorrhage. However, Bright (1990) felt that that electrocautery should be avoided entirely.

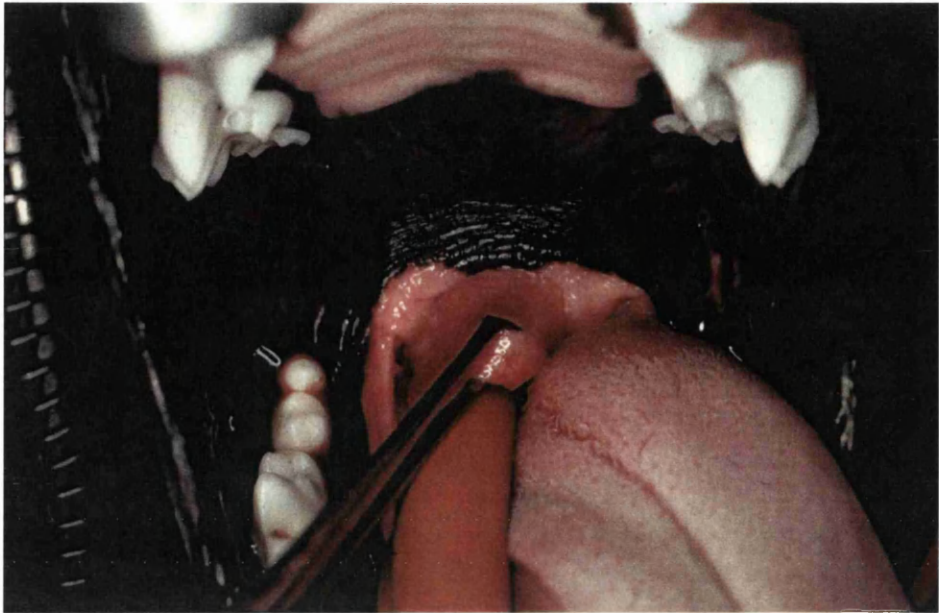
Glosso-epiglottic mucosa resection caused minimal haemorrhage and was not sutured as complicating post-operative sub-epiglottic adhesions or granulation have not been reported using this technique (Bedford 1983)

Stenotic nasal resection induces pronounced haemorrhage, which is rapidly controlled with the placement of sutures (Harvey 1982a). Alternatively, a fine bladed electro-scalpel can be used to control the haemorrhage. However, this can result in a degree of depigmentation lasting 4-8 weeks (Hobson 1995). Dehiscence may occur if the patient frequently licks or rubs its nose with healing occurring by second intention and resulting in a pink scar (Hedlund 1998). Harvey (1982a) reported a 95% of success rate judged by improvement or normality in breathing.

Harvey (1982b) observed that post-surgical gagging, retching and vomiting were common complications of elongated soft palate resection. Hendricks (1992) advocated that in the postoperative period food and water are withheld 12 to 24 hours, then starting with small amounts of water with later introduction of frequent small amounts of liquidised food.

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**Figure 6.1** The elongated soft palate has been grasped and drawn forward prior to resection.

## 7. Cranial Pubic Ligament Rupture in a Dog

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### 7.1 Introduction

The cranial pubic ligament, also called the pre-pubic tendon, is a tendinous band which extends from the ilio-pectineal eminence and the tendon of the *pectineus* muscle to the same structures on the opposite side, into which the *rectus abdominis* muscle inserts (Miller and others 1964).

The most frequent site of occurrence and recurrence of abdominal hernias was found to be the area of the caudal ventral abdomen, involving the pre-pubic tendon, either being torn in its body or detached from the pubic brim (Waldron and others 1986) as a result of the action of avulsion forces (Smeak 1993).

The main cause of pre-pubic tendon rupture is blunt trauma arising from road traffic accidents (Waldron and others 1986). Not surprisingly, associated injuries such as pelvic and femoral fractures, coxofemoral luxation, skin laceration, pelvic limb paresis and vascular thrombosis were found in cases of pre-pubic tendon rupture (Mann and others 1986). The effect of blunt trauma is associated with the lack of elasticity of the abdominal wall when compared with that of the linea alba (Parks 1981).

The most prominent sign of an abdominal hernia is a bulging mass beneath the skin or asymmetry of the abdominal contour (Smeak 1993). However, the location of the swelling caused by abdominal content does not necessarily mark the site of the anatomical defect. Contents can migrate subcutaneously or along fascial planes between muscular layers (Waldron and others 1986). Organs such as intestine may migrate down the medial thigh or along the abdominal wall to the thorax (Smeak 1998).

When palpation is difficult due to excessive subcutaneous swelling or pain, radiology is a useful tool. However, signs found are not pathognomonic of cranial pubic ligament rupture, and confirmation is by exploration and finding subcutaneously located abdominal organs and cranially retracted rectus abdominis muscle (Mann and others 1986).

Radiologically, decreased density outwith the abdominal wall may occur when intestinal loops are present within the hernia or when subcutaneous emphysema is present due to perforation of the intestinal wall (Bartels 1972). The intestines can be recognised by

their curvilinear shape, while subcutaneous emphysema tends to be more linear or irregularly shaped (Burk and Ackerman 1996). Delineation of ventral abdominal hernias usually does not require the use of contrast media. If there is any doubt about the diagnosis, a small amount of an organic iodine medium can be injected subcutaneously or into the abdominal cavity. Also barium suspension could be given orally to outline the hernial contents (Bartels 1972).

Ultrasound is very useful in cases of fluid-filled hernias that may mimic other soft tissue masses (Smeak 1993). It is much better than plain radiology at detecting the presence of urinary bladder within the hernia (Burk and Ackerman 1996) and simpler than using pneumocystography or double contrast cystography as suggested by Bartels (1972).

## **7.2 Case details**

### **History**

A 4 year old male collie cross (24kg body weight) was referred shortly after having being reversed over by a forklift truck. The only sign that was noticed afterwards by the owners was some degree of abdominal discomfort.

### **Clinical examination**

The dog was depressed and restless. The caudal abdomen was tense and painful on palpation, but no other neurological or orthopaedic complications were evident.

### **Ancillary aids**

Immediately after admission radiography was performed. The lateral thorax was unremarkable. However, a lateral abdominal view showed a soft tissue opacity caudoventrally, due to swelling and displacement of abdominal viscera. The urinary bladder integrity was checked by means of retrograde urethrogram, which was intact but herniated (Fig 2.7).

Haematological studies were performed and WBC ( $15.9 \times 10^9/l$ ), MCH (24.7pg) and MCHC (37.4g/dl) values were increased, whilst the haematocrit was low (0.36l/l)

Urine samples were sent for analyses and large amounts of red and white cells were detected. Protein (920mg/dl) and SG (over 1.055) were also high.

A tentative diagnosis of pre-pubic tendon avulsion was made and analgesia was provided overnight (methadone, 5mg IM). Peri-operative antibiotics was also started with amoxicillin/clavulanic acid 250mg TID.

## **Surgery**

The anaesthetic protocol consisted of IM pre-medication with acetylpromazine (1.25mg) and methadone (5mg), IV induction with methohexitone (125mg), followed by endotracheal intubation and maintenance of anaesthesia with a halothane/nitrous oxide/oxygen mixture.

As part of the surgical preparation, the bladder was catheterised and drained. The catheter was sutured to preputial skin and connected to a giving set and empty fluid bag for urine collection.

With the dog in dorsal recumbence, a caudal midline laparotomy was performed. Beneath the subcutaneous tissue, abdominal fat, omentum, bladder and some intestinal loops were found. Prior to returning these to the peritoneal cavity, it was explored but apart from some blood clots no internal organ damage was found. Remnants of the prepubic tendon were still attached to the pubis but the rectus abdominis was completely avulsed. The left inguinal ring appeared enlarged enough to allow the passage of omentum and fat. Using a drill and bit, several holes were made along the cranial pubic brim, and polypropylene (3 metric) sutures were pre-placed through them and the rectus abdominis muscle in a horizontal mattress pattern.

The inguinal hernia was repaired with the same material and pattern, avoiding damage to the external pudendal vessels and genital nerve. A Penrose drain was placed and the laparotomy incision closed routinely.

Treatment with carprofen (50mg BID) was initiated immediately after surgery. Antibiotics were continued for seven days.

The following day the drain had been pulled out. In the succeeding days the surgical wound became quite swollen and painful. Consequently, supplementary analgesia (methadone, 5mg IM) was provided. The scrotum was found to be also bruised, swollen and painful and a cream based on benzoic, malic and salicylic acid was used for local relief.

On the third day, as the dog was still showing signs of discomfort, abdominal radiography was repeated and the bladder was found in an inguinal location, suggestive of a breakdown of the repair. Therefore, the wound was re-explored. All the sutures had torn from the pubis, though the inguinal repair was still intact. The hernial repair was repeated using the same suture pattern with nylon (4 metric).

Five days after surgery the dog was discharged with the antibiotics and carprofen treatment. A Penrose drain was again pulled out by the dog, and use of Elizabethan collar was recommended as well as strict exercise restriction for 2 weeks to prevent the animal attacking the wound and reduce stress on the repair.

### **Follow up**

The dog was reassessed in 4 weeks and no signs of discomfort or herniation were noticed. The owners reported that for two weeks a persistent firm swelling was noticed at the wound site, which gradually resolved.

### **7.3 Discussion**

The mere presence of an abdominal hernia in itself, even if very large, is not necessarily an indication for immediate surgery. It is preferable to delay surgery for 3 to 5 days if possible, which allows acutely traumatised tissue to revascularise, thereby reducing infection. Also less inflammation and haemorrhage are present, allowing better exposure of structures and suture holding. Emergency surgery is indicated in cases that do not satisfactorily stabilise or that deteriorate despite intensive medical therapy such as animals with penetrating wounds or suspected incarceration (Smeak 1993).

Closure of this type of defect is facilitated by proper patient positioning. With the dog in dorsal recumbence, hind limbs are flexed and pulled cranially, creating a truncal ventro-flexion and reducing the size of the defect, which results in decreased tension during the repair (Smeak 1998).

Ideally the repair of a pre-pubic ventral hernia should begin with the identification of all abdominal muscular layers involved, so that repair of each layer may be accomplished, providing a grid-like closure of the wound and a stronger repair (Bojrab and Taylor 1983). Technical failures such as the use of incorrect suture material or inappropriate tissue layers are important causes of recurrence (Parks 1981).

Rupture of the cranial pubic ligament is often associated with concurrent inguinal ligament damage, therefore a femoral or inguinal hernia may be present, in which case the pre-pubic component of the hernia is repaired first to align the tissues correctly for anatomic reconstruction of other ruptures (Smeak 1998).

Reattachment to the pubis by sutures placed from the pubis may be either through holes drilled in the pubic bone or through the cranial aspect of *obturator foramina* (Mann and others 1986). Sutures are initially pre-placed and those that are located in the deepest parts of the wound tied first. Adjacent sutures are simultaneously tightened during knot tying to reduce fascial tearing (Smeak 1993).

When a large defect makes approximation of local tissue impossible without undue tension, prosthetic implants are used, most commonly made of polypropylene or polyethylene. Such materials are well tolerated in wounds, do not disintegrate with age and can stretch in two directions to distribute the load evenly. Granulation tissue and capillaries grow through the mesh, building a strong layer of connective tissue. The disadvantages of the use of this material include the possibility of rejection and irritation of adjoining tissue (Read and Bellenger 1993).

The most common complications of surgery are recurrence and wound infection with skin dehiscence (Waldron and others 1986). An abdominal bandage is recommended for 5 to 10 days postoperatively to obliterate dead space and to provide external support (Mann and others 1986). Also gravity dependent drains such as Penrose or closed suction drain systems should be used. It is advisable to avoid placing an open drain system directly against buried mesh to reduce the risk of ascending infection (Smeak 1998)

Aftercare includes placement on padded beds, taking care to turn the animal frequently, and after patient discharge exercise limitation of at least 2 weeks in order to reduce tension on the repair site (Smeak 1993).

The prognosis of this type of herniation after repair is good but it must be borne in mind that accompanying injuries can influence the outcome (Mann and others 1986).

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## 8. Intestinal Adenocarcinoma in a Cat

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### 8.1 Introduction

Tumours of the intestine are uncommon in domestic animals, and represent less than 1% of all malignancies (Straw 1996). Adenocarcinoma is the most common non-haematopoietic gastrointestinal tumour in cats (Turk and others 1981), although it occurs infrequently compared to the dog and man (Patnaik and others 1976).

The occurrence of intestinal adenocarcinoma in middle-aged to old cats has been confirmed by several authors (Brodey 1966, Patnaik and others 1976, Turk and others 1981, Cribb 1988, Kosovsky and others 1988). All of them also found that Siamese and domestic short hair were the breeds most affected. Although occurring sporadically in other breeds, one case in a Russian blue (Turk and others 1981) and one Persian cat (Cribb 1988). No sex predilection has been noted, apart from a marked male predisposition reported by Patnaik and others (1976).

Cachezia (Cribb 1988) and vomiting (Kosovsky and others 1988) have been reported as the most consistent findings in cases of intestinal adenocarcinoma. Other clinical signs include depression, anorexia, swollen and palpable masses (Patnaik and others 1976). Firm, moveable cylindrical masses in the cranial or mid-abdomen are often identified. As it is an annular tumour it may lead to some degree of obstruction, thus causing abdominal pain and fluid or gas accumulation (Crow 1985).

Clinical features of intestinal neoplasia mimic those of non-neoplastic disorders of the bowel. As a result it is difficult to diagnose this type of tumour using routine methods and they are frequently undetected in the early stages. However, early diagnosis is especially important because debilitated cats are more susceptible to the surgical stress and metastasis is more likely to have occurred (Cribb 1988).

Although blood analyses has been reported as rarely contributory in most cases (Crow 1985), Cribb (1988) reported neutrophilia and lymphopaenia as the most common haematological abnormalities in his series, as well as hypokalemia consistent with vomiting and decreased food intake. Also hypoproteinaemia and high activity of ALT and ALP have been described (Kosovsky and others 1988).

Kosovsky and others (1988) found that abdominal palpation and a positive contrast gastro-intestinal radiological study were the most reliable indicators of an encircling mass in the bowel wall causing stenosis of the lumen and partial obstruction. But final diagnosis of the cause of obstruction is achieved by means of exploratory laparotomy and histological examination (Cribb 1988).

Although Patnaik and others (1976) described the ileum as the commonest site, later authors reported the jejunum as the most usual location (Turk and others 1981, Kosovsky and others 1988) followed by ileum and ileo-caeco-colic region.

Ulceration at the primary site, severe desmoplastic reaction, muscular hypertrophy and luminal stenosis are characteristic features at the annular constriction caused by the tumour. Wall thickening is commonly present in the intestinal segment proximal to the neoplastic lesion, being considered to be a compensatory reaction to obstruction of the contractile movement. It is due mainly to hypertrophy and hyperplasia of the inner circular muscle. Post-stenotic dilation is also common and often the mesentery and/or the omentum adhere to the neoplastic section of intestine (Patnaik and others 1976).

Metastasis of small intestine adenocarcinoma has been described mainly to the peritoneum and regional lymph nodes, but also may appear in other sites such as liver, spleen, kidneys, lungs or diaphragm (Brodey 1966, Patnaik and others 1976).

Surgical resection is the treatment of choice for annular or discrete tubular neoplasia of the small intestine when unresectable metastases are not detected. Carcinomas that have metastasised can be reduced in volume by cyto-reductive surgery but have an unfavourable prognosis (Crow 1985). However, Kosovsky and others (1988) found that lymph node metastasis and carcinomatosis may not always indicate a poor prognosis and that recurrence of the primary tumour was the cause of the death of most long-term survivors.

## **Case details**

### **History**

A nine and half year old neutered male Siamese cat (2.7kg body weight) was referred with a 3 week history of intermittent vomiting and noticeable weight loss, which was unresponsive to medical management with antibiotics, steroids and metoclopramide.

The owners reported that the vomit looked like undigested food, although on some occasions had a faecal appearance. The faeces were of a normal consistency but produced in small volumes.

### **Clinical examination**

The cat was emaciated, dehydrated but still alert. The intestines were prominent and excessively gassy on abdominal palpation, as well as hyperperistaltic. Tachycardia and a soft left systolic murmur were also noticed.

### **Ancillary aids**

Blood and urine samples were taken for analyses. There was elevation in plasma urea (18mmol/l) combined with non-regenerative anaemia (HCT 0.21l/l) and high number of Heinz bodies. FIV and FeLV antibody tests were negative and urinalysis was unremarkable.

Contrast radiography, using barium-impregnated polyethylene spheres, was performed with lateral and ventro-dorsal abdominal views taken 12 hours post-administration (**Figure 8.1**). At that time a delay in the passage of some of the large spheres suggested decreased gut motility. Exploratory laparotomy was scheduled in order to achieve a definitive diagnosis.

### **Surgery**

The cat was put on fluids from the day of admission and IV antibiotic therapy was also started with amoxicillin/clavulanic acid (60mg) and metronidazol (30mg) TID.

The anaesthetic protocol consisted of pre-medication with pethidine (18mg IM) and ten minutes later diazepam (0.5mg IV). Induction was with propofol (18mg IV), followed by intubation and maintenance of anaesthesia with a halothane/nitrous oxide/oxygen mixture.

Midline ventral laparotomy was performed and a localised concentrically thickened area about 3 cm long was found at the ileo-colic region (**Figure 8.2**). Doyen intestinal forceps were placed with a 2cm healthy wall margin on either side and, after ligation and transection of mesenteric and arcadian vessels of the area, the bowel and adjacent mesentery were resected. The intestinal mucosa was trimmed and an end-to-end anastomosis was carried out in a simple interrupted fashion (2 metric polyglactin 910,

taper point needle) with the first suture placed at the mesenteric border. A pedicle of omentum was wrapped around the anastomosis and tacked to the intestinal serosa with simple interrupted sutures (2 metric polyglactin 910).

The rest of the abdominal viscera were unremarkable, and the regional lymph nodes were biopsied. Lavage of abdominal cavity was performed with warm sterile saline solution and laparotomy closed routinely.

Recovery was uneventful and therapy started on admission was continued. After 24 hours, the cat was alert and began to eat. By the following day, the cat was keen to walk about and there were palpable faeces in its colon. Clinical parameters at this point were unremarkable.

Seventy-two hours post-surgery, the cat became disinterested, dehydrated and pale with palpable intestinal gas content. A blood smear showed toxic neutrophilia with bizarre red cell morphology. The patient became progressively duller and the owners requested euthanasia.

## **Discussion**

In the treatment of tumours of the intestinal tract, careful attention to surgical technique in anastomosis is important, owing to the frequent debilitated state of the patient and potential risk of poor healing (Straw 1996). The clinical picture of this cat 72 hours after surgery suggested sepsis with or without peritonitis. Lymph node biopsy results did not show tumour infiltration. Unfortunately, post-mortem examination was declined by the owner.

Hypotension associated with anaesthesia and surgery adds to the circulatory disturbance created by fluid loss from vomiting and diarrhoea. Also in intestinal surgery, IV antibiotics should be administered at induction and continued post-surgery for a maximum of 24 hours (Orsher and Rosin 1993).

Preliminary ligation of the vascular trunks to avoid tumour emboli and minimal manipulation of the neoplasm is desired in order to minimise metastasis and recurrence (Cribb 1988). Instruments used during intestinal resection and anastomosis should be discarded after the procedure is completed. The peritoneal cavity should be lavaged and closure of the incision in the mesentery and abdominal wall performed with clean instruments and suture material (Orsher and Rosin 1993).

Of all the suture patterns that can be used in end-to-end intestinal anastomosis, approximating ones seem to have a number of advantages; such as increased lumen diameter, rapid and precise healing and minimal post-operative intestinal adherence. However, a simple continuous fashion is often preferred to an interrupted one to minimise mucosal eversion, provide a better serosal apposition and primary intestinal healing. Minimal mucosal eversion and proper incorporation of the tough submucosa are vital in performing consistent and successful anastomosis (Ellison 1998). A small taper-cut or reverse cutting needle penetrates the tough submucosa easily, which has a distinct advantage over a taper point or round bodied needle (Orsher and Rosin 1993).

Synthetic braided absorbable suture material such as 910 polyglactin is acceptable but has a degree of tissue drag, which is not present in monofilament absorbable sutures as polydioxanone or polyglyconate. Omental wrap has the advantage of isolating potential anastomotic leakage from the peritoneal cavity. The omentum can seal off infection and perforations, and impart new blood supply to viscera (Orsher and Rosin 1993).

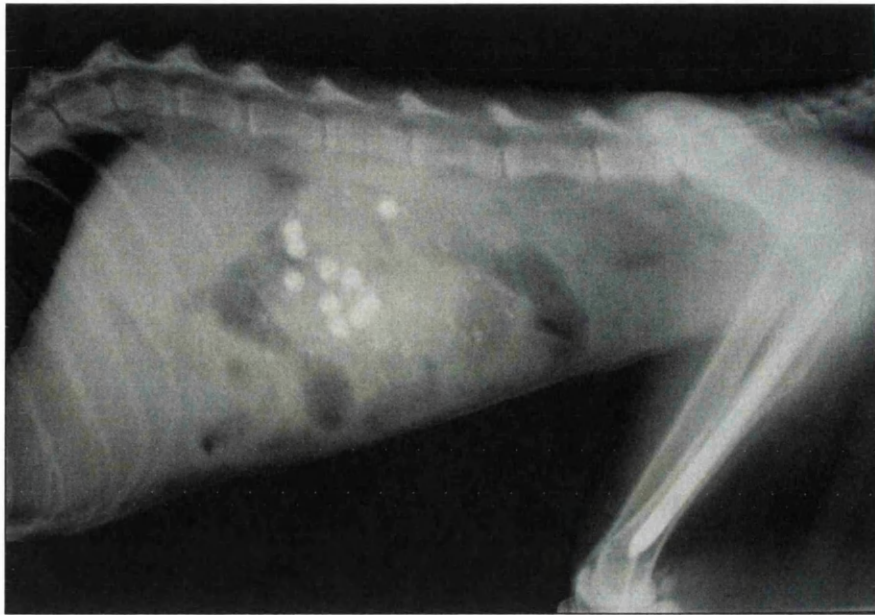
It is unfortunate that the owners declined post-mortem examination to determine the cause of death and the state of the enterectomy repair.

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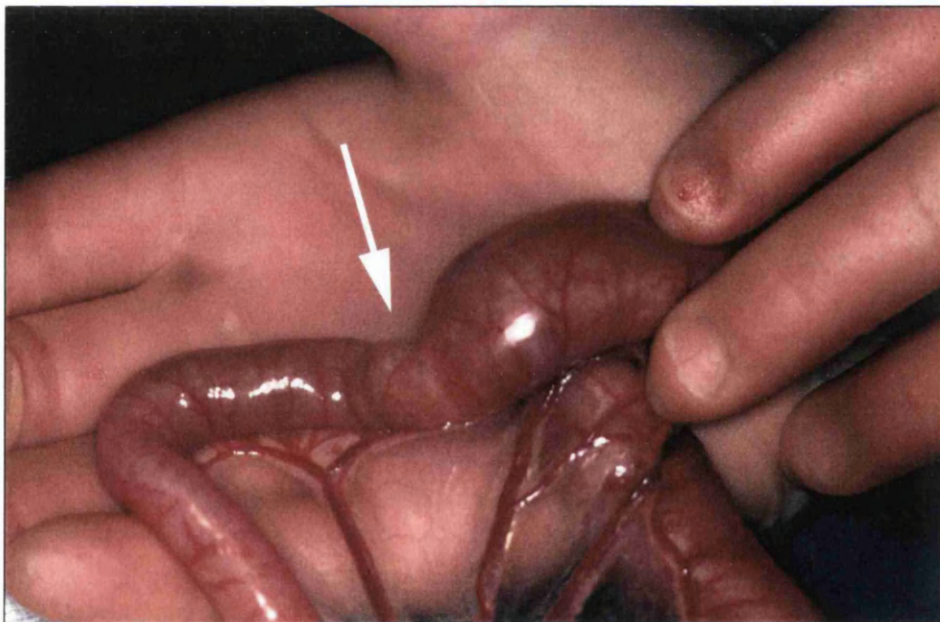
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**Figure 8.1** Intestinal adenocarcinoma, lateral abdominal radiograph. A dilated loop of bowel is evident with retained barium impregnated polystyrene spheres. Movement blur due to respiration is evident.



**Figure 8.2** A portion of the small bowel has been exteriorised to isolate the tumour in the intestinal wall (arrow).

## 9. Tracheal Puncture in a Dog

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### 9.1 Introduction

Injuries of the trachea usually result from blunt trauma to neck and/or thorax, which often involves hyper-extension of the head and neck (White and Milner 1995). However, despite the high incidence of this type of trauma, the occurrence of airway injury is low (Kellagher and White 1987). This is because, in most cases, the impact is lateral and the trachea can move instead of being crushed against the spine (Nelson 1993).

Minor perforations caused by bite wounds lead to few immediate respiratory problems (Kellagher and White 1987), but some authors have reported complete tracheal avulsion with formation of a thickened tracheal adventitia and pseudo-airway between severed segments that allows air intake to the lungs (Kennedy 1976, Barber and Rawlings 1981, Brouwer and others 1984).

Punctures or lacerations supported by healthy peri-tracheal tissue are self-limiting, even in the intra-thoracic segment or bronchi (Nelson 1993). However, air leaking into the peri-tracheal tissue may cause subcutaneous emphysema and even pneumomediastinum (Kellagher and White 1987).

Traumatic tracheo-bronchial rupture is the most likely cause of generalised subcutaneous emphysema. This condition may appear days to weeks after the injury and the cause has been suggested to be a dislodged fibrin seal or a gradual tracheal necrosis (Bauer and Currie 1988).

Apart from clinical examination, a palpable gap in the trachea or a wound in the skin may indicate the position of the rupture (Harvey and O'Brien 1972) but absence of skin puncture does not exclude it. Consequently, radiology and endoscopy are essential for an accurate diagnosis (Bauer and Currie 1988). Major lacerations of the trachea are normally visible on plain radiographs alone, whereas endoscopic evaluation may be necessary for the identification of smaller defects (Kellagher and White 1987). Lately, computed tomography has been suggested as a diagnostic tool for tracheal disruption (Fingland 1998).

Radiographic findings may include evidence of emphysema, focal narrowing of tracheal lumen, lack of tracheal continuity and gas-filled spherical dilation between severed segments. Pulmonary hyperinflation was reported in a case where a loose necrotic tag of tracheal wall was present at the orifice of the distal segment, creating a flap-valve effect (Barber and Rawlings 1981).

The accumulation of large amounts of air within the mediastinum may cause rupture of the pleura, converting the condition to a pneumothorax (Kellagher and White 1987). The latter may be controlled by means of suction drainage. Mediastinal and subcutaneous emphysema can be reduced by placing a tracheostomy tube which decreases airway resistance. Failure to resolve pneumothorax after 3 to 4 days is a criterion for surgical intervention (Nelson 1993).

## **9.2 Case details**

### **History**

A five year old female Whippet (10.5kg) was referred, following a dog fight 24 hours previously, with a left cervical swelling that had gradually developed to a generalised subcutaneous emphysema.

### **Clinical examination**

The dog was alert and responsive. Mucous membranes were slightly pale, although CRT was less than 2 seconds. The left cervical area had a large crepitant swelling and signs of haematoma. On palpation there was a widespread crepitation all over the dog, with only the right hind spared. Tracheal auscultation was harsh and especially difficult to hear on the left side.

### **Ancillary aids**

The dog was admitted and lateral and dorso-ventral thoracic, as well as lateral cervical radiographs were taken. Pneumomediastinum was confirmed with visualisation of the internal and external tracheal margins (**Figure 9.1**). A slight degree of pneumothorax was also noticed, and in the cervical view cranial tracheal damage could be detected.

A blood sample was taken for haematological and biochemical evaluation. Decreases in the values of WBC ( $4.30 \times 10^9/l$ ), RBC ( $5.77 \times 10^{12}/l$ ), lymphocytes ( $0.34 \times 10^9/l$ ), calcium (1.89mmol/l), albumin (24g/l), globulin (18g/l) and total protein (42g/l) were noticed.

MCH (25.20pg), MCHC (37.20g/dl), chloride (120 mmol/l), ALP (244U/l), AST (180U/l) and ALT (71U/l) measurements were above normal.

Acetylpromazine (0.2mg) and butorphanol (2mg) were administered IV before placement of a chest drain to permit hourly drainage. The first tap produced 235 ml of air. Fluid therapy was started at a maintenance rate, as well as antibiotics with metronidazole (10mg/kg TID IV ) and amoxicillin/clavulanic acid (20mg/kg BID IV).

Endoscopic examination was performed 12 hours after admission and a cervical tracheal defect was found.

## **Surgery**

The animal was pre-oxygenated and induced with propofol (10ml IV). Following careful endotracheal intubation, anaesthesia was maintained with a halothane/oxygen mixture.

The dog was placed in dorsal recumbency and the trachea was exposed via a ventral midline incision caudal to the larynx. A small defect was found at the level of the first to second tracheal spaces on the left. The defect was closed by passing three simple interrupted sutures (1.5 metric polypropylene) around the adjacent tracheal rings and a Penrose drain was placed. The incision was closed routinely.

The chest was drained on three further occasions before drain removal. Small amounts of soft food were offered every two hours and rapidly eaten, so fluid therapy was stopped 12 hours after surgery.

Recovery in the hospital was uneventful, and after 4 days the Penrose drain was removed and the dog sent home with on carprofen (20mg BID) and amoxicillin/clavulanic acid (250mg BID) for 10 days. There were still signs of emphysema but with an obvious daily improvement. The owners were advised to maintain the soft diet and to keep the dog on leash until the stitches were removed.

## **Follow up**

The dog was returned for a re-examination four weeks later. No evidence of subcutaneous emphysema was present and the owners reported a normal respiratory pattern and exercise tolerance.

### 9.3 Discussion

The main feature of this case was the generalised subcutaneous emphysema that is associated with the presence of pneumomediastinum, and is a specific indicator of upper airway leakage (Kellagher and White 1987). Nelson (1993) considered that even intra-thoracic lacerations of the trachea were self-limiting as long as pneumothorax was controlled by suction drainage. However, despite the small defect, it was decided to proceed surgically due to the large degree of emphysema present, a primary concern of the owners when the dog was referred.

One of the precautions that should be taken is careful intubation, because a forced manoeuvre or excessive inflation when using cuffed tubes, may worsen original injuries and even trigger a respiratory crisis (Hill 1974).

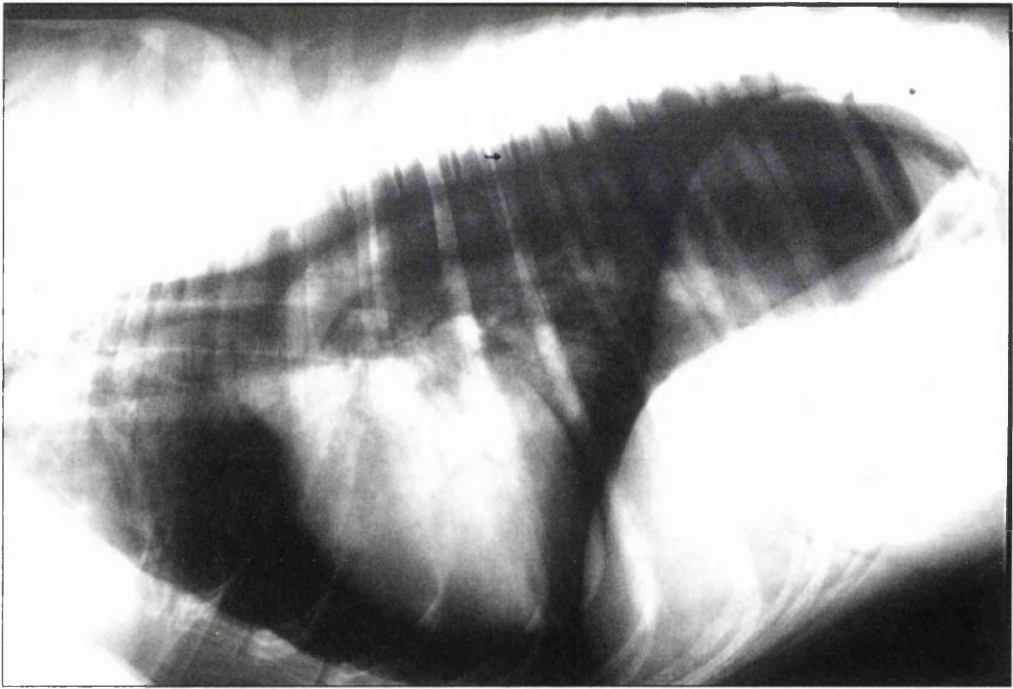
The size of the tear avoided the need for more complicated techniques such as the placement of tension sutures in order to appose the two segments (Hedlund 1984). With this small defect, it was not necessary to carry out a large reconstruction with sutures, as it has to be borne in mind that in tracheal surgery, excessive sutures are always undesirable. Intra-luminal knots stimulate granulation tissue production at each site (Nelson 1993), acting as a foreign body in the trachea (Harvey and O'Brien 1972)

Postoperatively respiratory distress can result from laryngeal or pharyngeal oedema, occlusion of the tracheal lumen at the anastomotic site or iatrogenic laryngeal paralysis from intra-operative recurrent laryngeal nerve injury (Fingland 1998).

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**Figure 9.1** Tracheal rupture, lateral thoracic radiograph. The cardiac shadow is elevated from the sternum and there is an increased radiolucency indicative of pneumothorax. The structures contained in the cranial mediastinum are visible indicating a pneumomediastinum.

## **10. Abdominal Unilateral Cryptorchidism in a Cat**

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### **10.1 Introduction**

Cryptorchidism is a developmental defect with failure of testicular descent into the scrotum. It may be bilateral or unilateral (Rhoades and Foley 1977). Unilateral cryptorchidism, which is more common than bilateral (Millis and others 1992), is often incorrectly referred to as monorchidism, a term that implies lack of one of the testicles, regardless of its location (Rhoades and Foley 1977).

In most species, cryptorchidism is thought to be a simple autosomal recessive genetic defect, where females might be carriers (Burke 1986).

Some of the factors associated with cryptorchidism include lack of androgens, lack of gonadotropin releasing factors or incompetent gubernaculum. Testicular descent occurs in two morphological phases. First is transabdominal translocation, which is non-androgen dependant and during which the gubernaculum does not contract. Thus the relative position of the gonad is changed as a result of foetal growth. Second is inguino-scrotal descent, which is not only androgen dependant, but under the influence of the genitofemoral nerve and gubernaculum (Buergelt 1997).

The testicles are just inside or within the internal inguinal ring and they reach the scrotum after scrotal development, at 10 to 14 days of age. Scrotal development may be delayed in some breeds of kittens rather than in puppies, especially the Persian, where it may take up to a year (Burke 1986).

Cryptorchidism in cats has been infrequently reported, although feline prevalence was found to be similar to that reported in dogs (Millis and others 1992). Other authors suggested that the defect may also be unnoticed due to the large inguinal fat pad (Richardson and Mullen 1993).

Retained testicles may be located pre-scrotally in subcutaneous inguinal fat, in the inguinal ring or within the abdomen. Initially, it was thought that in most cryptorchid cats the testicles were located subcutaneously (Noffsinger and Carbone 1978). Later reports have suggested a similar incidence in abdominal and inguinal locations (Millis and others 1992, Richardson and Mullen 1993). The same authors found the right and

left testicles were equally affected and that abdominal testicles were mostly located near the bladder, although in some cases they may be found close to the caudal renal pole.

Cryptorchidism implies lack of spermatogenesis of the affected testicle due to the high temperature compared with the normal scrotal position, therefore bilateral cryptorchid animals are sterile. Testosterone production may be somewhat lower than normal but is still present in the retained gonad (Rhoades and Foley 1977).

Stimulation of testosterone production with intravenous human chorionic gonadotropin has been used in the diagnosis of feline cryptorchidism in unilaterally castrated cats (Memon and others 1992). Richardson and Mullen (1993) reported that in 50 cats the location of the retained testicle was correctly palpated in only 21 due to interference by the fat pad or abdominal position.

Cryptorchidism may be linked to undesirable syndromes in affected animals, such as anal gland adenoma, sertoli cell tumour, seminoma, prostatic hyperplasia and temperamental behaviour (Wolff 1981). Tumours of retained testicles are more often malignant than those of descended ones (Burke 1986), although testicular tumours are rare in cats (Feldman and Nelson 1996). There is no evidence to suggest that they are prone to torsion of the affected testicle by twisting of vas deferens and spermatic vessels around each other as has been reported in the dog (Guernsey 1984).

Orchiopexy of the ectopic testicle is unethical, because it results in a fertile animal with a heritable defect (Kirby 1980). Hormonal supplementation, such as megestrol acetate, has been used unsuccessfully (Richardson and Mullen 1993). Total orchietomy is the treatment of choice.

## **10.2 Case details**

### **History**

An 11 month old male Siamese (4.95kg body weight) was admitted for neutering. The owners reported that, otherwise, the cat was completely healthy.

### **Clinical examination**

The cat was bright and alert. No abnormalities were noticed except for the presence of only one intra-scrotal testicle. On palpation the left testicle was suspected to lie in the inguinal region. No other ancillary tests were performed and neutering was scheduled.

## **Surgery**

The anaesthetic protocol consisted of IM pre-medication with acetylpromazine (0.1mg) and pethidine (27.5mg), IV induction with propofol (40mg), followed by endotracheal intubation and maintenance of anaesthesia with a halothane/nitrous oxide/oxygen mixture.

After the cat was clipped for surgical preparation, inguinal palpation was performed again but the retained testicle could not be identified.

With the cat in dorsal recumbency routine pre-scrotal castration was performed on the right descended testicle. Caudal ventral midline incision was done to allow dissection deep into the inguinal fat pad and therefore visualisation of the external inguinal ring and better exposure of testicular vessels and ductus deferens of what was suspected to be an inguinal testicle.

The ectopic gonad was not found, so the dissection was deepened to that of celiotomy for abdominal exploration. A hypoplastic testicle was eventually located laterally, mid-way between bladder and left kidney (**Figure 10.1**). The testicular vessels and ductus deferens were clamped, ligated (2 metric polyglactin 910) and divided. The testicle was removed, the vessels checked for haemorrhage and the abdomen closed in a routine fashion.

## **Follow up**

The cat was discharged two days after surgery with a treatment of carprofen (10mg SID PO) and amoxicillin/clavulanic acid (50mg BID PO) and placement of Elizabethan collar advised.

Sutures were removed after 10 days, and no complications were observed during post-surgical period.

## **10.3 Discussion**

Unilateral cryptorchidism involves the presence of a normally located testicle that allows the animal to be fertile. However, affected animals should not be used for breeding as carriers of a genetic defect (Rhoades and Foley 1977). Removal of the scrotal testicle allows the surgeon to identify the side on which the search should be concentrated (Hardie 1984).

Ectopic testicles, by producing testosterone, are responsible for undesirable signs of male behaviour such as aggressiveness, urine spraying, loud vocalisation and mounting of female cats (Memon and others 1992, Richardson and Mullen 1993).

The testicle is only reliably palpated in a pre-scrotal location, caudal to the fat pad (Richardson and Mullen 1993). Sometimes, the superficial inguinal lymph node can be confused with a retained testicle (Hardie 1984). When the testicle is palpated, a para-median incision should be made over the inguinal fold (Millis and others 1992). However, because of the unreliability of palpation, and the fact that approximately half of the testicles were identified in an abdominal location, the caudal abdominal surgical approach is recommended if palpation fails to identify the gonad. If the testicle is not readily evident in the inguinal region, gentle traction of the abdominal portion of the spermatic cord can help to locate the testicle in this area, thus avoiding unnecessary dissection of the inguinal fat (Richardson and Mullen 1993).

Millis and others (1992) recommended the caudal ventral mid-line approach as the best one for orchietomy of cryptorchid cats allowing progression to standard midline celiotomy. The same authors reported that in all but one case of abdominal cryptorchidism the testicle was found near the bladder. Hardie (1984) suggested that in cases where it is difficult to localise the testicles, the ductus deferens should be located as it enters the dorsal aspect of the prostate gland. Alternatively, the testicular vessels may be traced from the caudal aspect of the kidney to the testicle.

Kirby (1980) and Hakala (1984) used the snook hook in abdominal cryptorchidectomy suggesting that it would reduce anaesthetic time as well as the risk of postoperative herniation and evisceration. However, removal of abdominal testicles through small incisions or use of spay hook is not recommended because of the risk of damage to the ureters, which are located near the ductus deferens and may be avulsed from the trigone of the bladder or incised (Millis and others 1992).

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**Figure 10.1** Abdominal cryptorchidism, intra-operative picture. The abdominal testicle has been exteriorised.

**APPENDIX 1: Blood reference values**

| <b>Parameter</b>  | <b>Feline</b> | <b>Canine</b> |
|---|---------------|---------------|
| WBC (White Blood Cells) x 10 <sup>9</sup> /l              | 5.5-15.5      | 6.0-12.0      |
| Neutrophils x 10 <sup>9</sup> /l                          | 2.5-12.5      | 3.0-11.8      |
| Lymphocytes x 10 <sup>9</sup> /l                          | 1.5-7.0       | 1.0-4.8       |
| RBC (Red Blood Cells) x 10 <sup>12</sup> /l               | 5.0-10.0      | 5.5-8.5       |
| HB (Haemoglobin). (g/dl)                                  | 10.0-15.0     | 12.0-18.0     |
| HCT (Haematocrit). (l/l)                                  | 0.30-0.45     | 0.37-0.55     |
| MCH (Mean Corpuscular Haemoglobin). (pg)                  | 12.5-17.5     | 19.5-24.5     |
| MCHC (Mean Corpuscular Haemoglobin Concentration). (g/dl) | 30.0-36.0     | 32.0-36.0     |
| PLT (Platelets). (x 10 <sup>9</sup> /l)                   | 300-800       | 200-500       |
| Chloride. (mmol/l)  | 94-113        | 95-115        |
| Phosphate. (mmol/l)                                       | 1.29-2.84     | 1.29-2.90     |
| Calcium. (mmol/l)   | 1.60-2.56     | 2.34-3.00     |
| ALP (Alkaline Phosphatase). (IU/l)                        | <100          | <230          |
| AST (Aspartate Amino Transferase). (IU/l)                 | <30           | <40           |
| ALT (Alanine Amino Transferase). (IU/l)                   | <35           | <90           |
| Cholesterol. (mmol/l)                                     | 1.8-5.2       | 2.0-7.0       |
| Glucose. (mmol/l)   | 2.7-5.5       | 3.3-5.5       |
| Urea. (mmol/l)  | 2.7-9.2       | 2.5-8.5       |
| Creatinine. (umol/l)                                      | 91-180        | 45-155        |
| Total Protein. (g/l)                                      | 60-85         | 50-78         |
| Globulin. (g/l)   | 27-45         | 28-42         |
| Albumin. (g/l)  | 26-36         | 29-36         |

(l) = litres, (g) = grams, (dl) = decilitres, (pg) = pico-grams, (mmol) = mili-mol, (IU) = international unit, (umol) = micro-mol