CLINICAL STUDIES OF THE FELINE UROLOGICAL SYNDROME

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D.V.M., Colorado State University, 1945

A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Surgery and Medicine

KANSAS STATE UNIVERSITY
MANHATTAN, KANSAS

1970

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Major Professor

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INTRODUCTION

A commonly occurring disease involving the urinary system characterized by dysuria, urethral obstruction, and/or hematuria has been reported in cats of various ages, sex, breed, and from different environments. The syndrome may involve the urethra, urinary bladder and kidneys, or only the urethra and bladder. Various authors have described the entity as cystitis (1-2), urolithiasis (3-4-5-6-7-8), urethroadenocystitis (9), urethrostenosis (10), and urethral obstruction (11-12-13). Because of the diversity of nomenclature, the entity will be referred to as the feline urological syndrome (FUS) in this report.

A 5% incidence of FUS has been noted among the feline patients entering Dykstra Veterinary Hospital over a three-year peiod of time. The significance of the disease has long been appreciated by veterinarians and pet owners because of its high incidence, the difficulty of effecting a lasting cure, the need for long-term therapy, and the extreme discomfort to the patient.

The literature contains widely diverse conclusions regarding effective therapy and etiology of the syndrome. The differences are most notable concerning the significance of urinary pH values as an etiological factor and as a guide to effective therapy. Despite the face that there is little documented evidence of the importance of urinary pH, many

authors (3-5-13-14) have assumed effective therapy must include the use of urine acidifiers. Antibiotic therapy is widely recommended (2-18-9), even though proof that bacterial infection plays a significant role in FUS is lacking.

This study was undertaken to investigate some of the etiological and therapeutic aspects of the disease. It was postulated, contrary to many reports in the veterinary literature, that urinary pH values were not of particular significance to the disease. It was assumed that data obtained from a large number of naturally occurring cases of FUS might answer some of the basic questions concerning the incidence, etiology, and therapeutics of the disease that have remained largely unanswered. Three hundred case reports involving FUS were documented and classified.

LITERATURE REVIEW

Several authors (4-5-6-8-9-12-13-14-15) have described the clinical signs of FUS in detail. Most emphasize the dramatic aspect of the syndrome which is characterized by urethral obstruction and urine stasis (4-8-9-12-13-14-15). Whitehead (15) described affected cats with cystitis as having partial anorexia, listlessness, excessive thirst, frequent urination, and a urine which appeared normal or only slightly discolored. As the

cystitis became more severe, frank hematuria and occasional vomiting were seen. His description of the male cat with urethral blockage is echoed in less detail by other authors. According to Whitehead, "If the urethra becomes occluded the cat will be in its pan almost constantly, straining and crying in distress and passing only a few drops of urine or none at all. The urine may be blood-tinged. Often the owner thinks that the cat is constipated and all too often administers mineral oil or some other laxative. Cats with urethral obstruction may appear dull and uneasy, frequently looking at the flanks. The anxious expression of the eyes and the face is distinct evidence of pain. When approached the patient will wait until touched until moving from its position. The penis and sheath are often edematous and discolored. The penis usually protrudes to its fullest extent; the extremity moving spasmodically due to the effort to urinate. During the physical examination nothing abnormal may be found until the abdomen is palpated. The distended bladder can be felt through the wall of the abdomen as a large, firm, ovoid body. The temperature may or may not be abnormal depending on the duration of the condition and the complications which may be present. In long-standing urinary stasis the temperature is likely to be subnormal. In advanced cases the oral mucosa may be muddy and the breath uremic. The animal may be comatose, or nearly so, due to the uremia."

Veterinary literature contains conflicting statements concerning the etiology of the syndrome. Holzworth (18) states the basic cause is probably the cat's low threshold for phosphate excretion, but premature castration and cold weather may be contributing factors. Whitehead (15) comments that the etiology is obscure but lists several theories. He writes that some of the factors listed are known to be involved in other species but as yet there is insufficient evidence that they are causes in the cat. The possible etiological factors as listed by Whitehead are: (1) colloid-crystalloid imbalance (2) bacterial infection (3) avitaminosis (4) stasis (5) inadequate fluid intake (self imposed or otherwise) (6) retention due to the poor elimination facilities (7) endocrine imbalance (8) hyperparathyroidism (9) high ash diet.

Schein (19) noted that opinions are varied concerning the etiology. He reports that some veterinarians believe that the disease is due to infection while others hold to the theory that there is an association with the age that the cat is castrated. The theory suggests that the older the animal at the time of castration the larger the diameter of the urethra and therefore, the smaller the incidence of anuria or dysuria. Still others, says Schein, assume that a diet consisting largely of cholesterol-rich foods, such as liver, kidney, and eggs may be incriminated.

Meier (9) writes that while he has seen more castrated cats with urinary tract obstructions than non-castrated ones, it may be because more castrated cats are presented for medical care. He suggests that the claim that castration results in a narrowing of the urethra may not be valid. He comments that castration may produce atrophy of the prostate gland and that the atrophy might affect the striated muscles surrounding the urethra in such a way as to decrease the ability of the urethra to expel obstructive material.

Meier (11) states that the etiological factor is a microbe of the genus Pseudomonas in a large number of male cats. He recalls that the cause has been attributed to castration, calculi, triple phosphate crystals, chemicals in city drinking water, certain foods and climatic conditions (cold). It is Meier's belief that the syndrome begins with cystitis due to bacterial invasion and that triple phosphate crystals commonly found in the bladder and urethra are incidental. He concludes that crystals are a contributing factor only when there is sufficient inflammatory exudate from infection in the bladder to form a conglomerate.

Rich (20) states that one contributing factor in the development of urethral obstruction is the quantity of struvite crystals ($NH_4MgPO_4 \cdot 6H_2O$ -- Ammonium Magnesium Phosphate) in the urine. The cat's fluid intake, high urine specific gravity, and the

ability to go long periods without urinating predispose to urethral obstruction. Rich (21) cites Vermeulen (27) et al, who defined the essentials for stone formation as: (1) precipitation of crystalloids, (2) retention of crystalloids, (3) presence of matrix or binding substance. Rich suggests that these factors are generally present in the cat. He further states that such factors as low water intake, infection, and changes in urinary pH values may contribute to conditions favorable for precipitation. Stasis and retention allow time for stones to form. Calculi are not only aggregates of crystals but are also composed of a protein matrix which holds the stones together. Large calculi are not common in the cat, according to most authors (3-5-12-15-19).

Rich concludes that struvite crystals alone do not constitute the cause of urethral obstruction and that urinary acidification, even when prolonged, does not prevent recurrence of obstruction. In 1956, Dickensen (16) reported the addition of high levels of properly balanced calcium and phosphorus in the diet of young kittens did not induce the formation of urinary calculi. Gershoff (22) substantiated this conclusion. Bloom (23) compared the quantitative urinary excretion of inorganic phosphate fed in different diets. He concluded that cats fed a prescription diet* excreted considerably less inorganic

* Atlas Felodiet (A prescription Cat Diet)

phosphate in their urine than cats fed milk and raw meat, catfish and cereal diet and 3 other brands of prescription diets. He notes a common factor in the urethritis, cystitis, calculi syndrome as the precipitation of urinary inorganic phosphate, either as fine, sand-like calculi in the bladder or as secondary deposits of gritty material within the inflammatory exudate in the urethra. He suggests that since the urinary salts involved in the syndrome are inorganic phosphate, the logical goal should be directed toward decreasing their excretion in the urine. He concludes that the prescription diet used was the only diet in his study associated with a conspicuous reduction of inorganic phosphate in the urine. Foster's (24) statistical analysis failed to reveal evidence supporting theories that early castration is a causative factor in this syndrome. McCully and Liberman (17) and Meier (9) concluded that early castration does not produce a narrowing of the urethral lumen following studies of the histology of the urogenital tract in urolithiasis.

Other authors (3-5-13-14) stress the significance of urine pH values as an etiological factor. Carbone (13) indicates that pH abnormality is a cause of FUS. He states that as a result of metabolic fluctuation in the normal cat, urine pH rises above 6.8 which in turn results in the crystalization of struvite in

the urine. The elongated sharp-edged crystals may settle in the bladder or accumulate in the narrow penile urethra, thus producing a small plug in the distal portion. Carbone postulates that as the cat strains to pass urine, additional crystals pack into the partially occluded urethra, and finally, produce complete obstruction. The presence of crystals causes the urethral wall to become inflamed and swollen which further obstructs urine passage. During stasis, additional crystal growth may take place so that the fine-sand-like material becomes larger in size and quantity. Infection may occur, however, and Carbone suggests the bacterial infection is secondary. He hypothesized that crystals forming in normal urine with a pH value above 6.8 constitutes a primary cause of urethral obstruction in the male cat. Secondary pathological changes in kidney, bladder, and urethra lead to perpetuation of the syndrome or to later recurrences.

Holzworth (18), Carbone (13), Rich (20-21), Lumb (2), and Meier (9) have suggested that in nearly all cases the crystalline material obtained from the urinary bladder or urethra is struvite ($NH_4MgPO_4 \cdot 6H_2O$). Smith (25) reported uric acid crystals in a urethral obstruction. Jackson (26) described oxalate calculi. It is apparent, however, that struvite is the mineral almost universally identified in the feline urinary system. Rich (21) evaluated the crystals by x-ray defraction patterns and his

results provide excellent evidence that the mineral material is struvite.

Blood urea nitrogen values in affected cats have been reported. Whitehead (15) suggests that blood urea nitrogen measurement may be significant if the patient is vomiting or depressed and that high blood urea nitrogen values relate only to obstruction and will normalize when the obstruction is removed. Bloom (23) reported blood urea nitrogen values above 75 in obstructed cats. However, Meier (9-11) failed to find blood urea nitrogen values above the normal ranges. Carbone (13) noted the presence of glycosuria in 9 of 29 cats suffering from urethral obstruction. He suggested that glycosuria was probably due to renal tubular dysfunction, preventing the normal reabsorption of glucose. He further postulated that persistent obstruction and the resultant increased pressure caused complex physiopathologic events to occur in the upper urinary tract. Initially, glomerular filtration is greatly reduced, then muscular propulsion by the calices, pelvis, and ureters ceases, followed by severe reduction in tubular secretion. As internal parenchymal pressure rises, interlobular venous flow is obstructed which results in further reduction of glomerular filtration. The decrease in urine flow with concurrent decrease in electrolyte excretion does not completely stop renal activity. Modified urine

formation continues, although most urine must be absorbed. The end result is a rising blood urea level. Appropriate amounts of blood and tissue debris arising from vascular engorgement and hemorrhage in the renal parenchyma, from the degeneration of the renal papillae and later from degeneration and hemorrhage in the lower urinary tract appears in the urine. If obstruction is relieved, diuresis of dilute urine occurs and the blood urea nitrogen rapidly returns to normal. The diuresis is attributed to the rapid excretion of previously retained electrolytes and urea. Whitehead (15) also noted the presence of glucose in the urine. Blood sugar values remained within normal ranges according to both Carbone (13) and Whitehead(15).

Meier (11) emphasized the significance of bacterial infection of the post-renal urinary organs. He reports eleven cases of FUS due to Pseudomonas sp. infection. Staphylococcus sp., Streptococcus sp., E. Coli, and Aerobacter sp. have been identified in the urine of affected cats (14). Whitehead (15) observed that the urine is usually alkaline in cystitis whereas the normal cat urine ranges in pH from 5 to 6. He also noted albuminuria with urine sediment containing red blood cells, white blood cells, epithelial cells, renal cells and bacteria. Whitehead (15) describes histopathological changes in the urinary tract of affected cats. The kidneys showed interstitial round cell

infiltration, tubular damage, severe congestion of the medullary vessels with hemorrhage by diapedesis into the surrounding tissues. Looseness of the tissues at various levels in the tract was considered to be due to infiltration with escaped urine and contributory to the high blood urea nitrogen levels found. Meier (9) described the histological lesions of one cat. The lesions consisted of hemorrhage in the mucosa of the urinary bladder and urethra. The inflamed areas contained serous fluid, polymorphonuclear leukocytes, macrophages, lymphocytes and widely dispersed erythrocytes. Several thrombi were noted. Lesions in the parenchyma of the kidney included various degrees of degenerative change such as coagulation and caseation. Occasional areas of focal necrosis were seen in the mesenchymal tissues. Bloom (23) describes the occasional presence of tubular crystalline deposits, especially in the collecting tubules.

A wide variety of therapeutic techniques have been described. The majority of authors discuss various surgical techniques for the correction of the urethral blockage (2-4-5-9). Bloom (23), Whitehead (15), and Beamer (23), described in detail a precise method of back-flushing or washing of the obstructed urethra. Whitehead (15) suggests that when it is impossible to flush the debris out of the urethra, the urinary bladder may be emptied via a hypodermic needle to give temporary relief. In

some cases, the relief of urine pressure behind the obstruction seemed to alter its position, resulting in the semi-solid mass breaking into small particles and being washed away. Drainage may permit relaxation of the musculature of the bladder and urethra and so enable the patient to urinate voluntarily shortly after paracentesis vesicae.

Almost without exception authors have recommended urinary acidification in treatment and prophylactic aftercare of affected cats. Whitehead (15) proposed that urinary acidifiers be administered daily for 2-3 weeks. He preferred the use of ethylene diamine dihydrochloride over other drugs because of the convenient size and recommended that one tablet be given every eight hours for 2-3 weeks. He also mentioned using sodium acid phosphate or ammonium chloride to acidify the urine. Holzworth (18) suggested the use of dl methionine twice daily as a superior urinary acidifier.

Rich (21) reported that urethral obstruction resulted following inoculation of centrifuged urine from cats with urethral obstruction into the urinary bladders of unaffected cats. Filtration of urine from affected cats did not remove the causative factor. He isolated a picornavirus from muscle and urine of obstructed cats. The isolate from urine when inoculated into unaffected cats produced urethral obstruction in 3 of 4 inoculated cats.

MATERIALS AND METHODS

Three hundred cats affected with FUS were observed. The cats were patients at Dykstra Veterinary Hospital and were entered by their owners for diagnosis and treatment. A history was taken from each owner. The following items, when available, were recorded:

1. Number of cats in household
2. Breed, color, weight, and age
3. Sex - (male, female, altered male or female)
4. Diet and water source
5. Age at which neutering was done
6. Environment
   (a) indoor only
   (b) outdoor only
   (c) indoor and outdoor
7. Previous sickness or injury
8. Previous prophylactic vaccinations
9. Routine medications or vitamins
10. Chronic disease
11. Previous urinary problem
12. Owners observations of current condition

Each cat was examined as soon as possible after entry. The following information was recorded:

1. Attitude and approximate weight
2. Temperature, pulse, respiration
3. Size and texture of the urinary bladder, including an estimate of the quantity of urine in the bladder
4. Appearance of penis
5. Estimation of other clinical signs, such as anemia, constipation, obesity or emaciation, senility

Urine was obtained by cystocentesis. A 21-gauge 1-inch needle was inserted into the bladder through the abdominal wall and urine drawn into a 20 ml sterile glass syringe. A 5 ml sample was placed in a sterile tube and the remainder stored in a standard urine specimen container. Blood was obtained via venapuncture. Two ml was mixed with 3 drops of 10% EDTA anti-coagulant and two ml was allowed to clot. The specimens were subjected to the laboratory tests:

A) Urine
   1. Routine urinalysis
   2. Culture for microorganisms

B) Blood
   1. Complete blood count
   2. Examination of stained blood smear for blood parasites in cases where anemia was noted

3. Blood urea nitrogen

Therapy was initiated immediately in these cases of urine stasis in the male cat. Several different measures were used to relieve the crisis. Antispasmodics were administered in 51 male cats to relieve urethral spasm or obstruction. Thirty percent of the cases were mechanically back-flushed. Patency of the urethra was established by flushing water through a canula after its insertion into the distal segment of the penile urethra. In 38 cases the bladder was emptied by cystocentesis. Cystocentesis was performed when back-flushing failed to relieve the urinary stasis in 8 cases.

A variety of pharmaceutical products were used in treatment. The products used were as follows:

1. Antibiotic and Bacteriostatic drugs -- Furadantin, Chloramphenicol, Tetracycline, Oxytetracycline, Penicillin, Streptomycin, Sulfasoxisol, Sulfadimethoxine.
2. Urinary acidifiers -- Ammonium chloride, ethylene diamine dihydrochloride, sodium acid phosphate, dl methionine.
3. Antispasmodics -- Octin^{R}, Jenotone^{R}, Atropine, Scopolamine, Meperidine hydrochloride.
4. Miscellaneous drugs included proteolytic enzymes (Orenzyme^{R}, Kymar^{R}, Varidase^{R}), steroids, sodium

chloride and sodium bicarbonate.

Results of the various therapeutic methods were recorded throughout the course of the disease. Observations concerning the patient were recorded during its stay in the hospital. Attempts were made to physically evaluate the patient at least twice after release.

Patients which died were necropsied. Gross pathology was described and recorded and histopathological observations made.

Ten healthy male cats were obtained and housed in metabolism cages for the evaluation of pH values and crystalluria of normal cats. Four days were allowed for the cats to become accustomed to the cage environment. Canned cat food was fed daily. Fresh water was offered twice daily. Urine was collected twice daily. The pH was measured with a pH meter within 1 hour of obtaining the specimen. Each specimen was examined microscopically for crystalluria and the results were recorded. These measurements were recorded for ten consecutive days. Whenever normal micturition did not occur twice daily, a specimen was obtained by manual expression of the urinary bladder.

The same 10 normal cats were utilized in a study of the influence of acidifying drugs on urinary pH values and crystalluria. Three groups of three cats each were given one of the following drugs for five consecutive days. The tenth cat was

used as a control. No drug was given to the control. The cats were housed and fed as in the previous experiment. Urine specimens were obtained and examined microscopically for crystalluria. The urinary pH measurement were recorded. Drugs and dosages utilized were:

1. ethylenediamine dihydrochloride (Chlorethamine^{R})
   100 mg tablet every 8 hours
2. dl methionine (Odortrol^{R})
   .02 gm tablet every 8 hours
3. ascorbic acid
   250 mg tablet every 8 hours
4. acetylsalicylic acid
   5 gr tablet every 12 hours
5. ammonium biphosphate with sodium biphosphate and sodium acid pyrophosphate (pHospHaid^{R})
   0.25 gm tablet every 12 hours
6. ammonium chloride (enteric coated tablet)
   5 gr tablet every 12 hours

The three groups of cats were identified as Group A, Group B, and Group C. Group A received ethylenediamine dihydrochloride; Group B received dl methionine; and Group C received ascorbic acid for 5 days. After a three-day interval during which no drugs were administered, the three groups of cats were again

medicated. Group A received acetylsalicylic acid. Group B received pHospHaid[R]. Group C received ammonium chloride. Each drug was given to the cats in the assigned group for a period of 5 days.

Continued presence of struvite crystals in the urine of seven cats that had recovered from FUS made it feasible to investigate the efficacy of two drugs in the reduction of crystalluria. Only male cats were used. Their selection was based on the production of at least 50 mg of struvite crystals in the urine per 24-hour day. The cats, housed in metabolism cages, were fed and watered as in the previous study. The seven cats were given a 100 mg tablet of ethylenediamine dihydrochloride at eight-hour intervals. Urine was collected daily and examined for the presence of struvite crystals. The amount of struvite was recorded using a scale of 1 to 4. The scale was based on visual estimation. It was noted that 20 mg of struvite was the smallest volume that was easily detectable without the use of magnification. Estimations were recorded on the basis of the scale of 1 would equal approximately 20 mg or less of struvite; a scale of 2, 40 mg; a scale of 3, 60 mg; and a scale of 4, 80 mg or more of struvite. The study was continued for 6 days. In addition to the struvite estimation, urinary pH values were noted.

Following a 3-day waiting period to eliminate any trace of

drug from the seven cats, urine was again examined for the presence of struvite crystals. If present in approximately the same amount as in the previous study, each cat received 20 mg of the chelating drug tetrasodium ethylenediamine tetraacetic acid at 8-hour intervals for five days. Urine was collected daily, struvite and pH measurements were noted. If any cat still revealed gross struvite crystals in its urine, the 5-day study was repeated using 40 mg of the chelating drug instead of 20 mg. Further increases of the drug were administered until a maximum of 80 mg every 8 hours was used.

Eighteen young adult male cats were used in a study of the efficacy of four drugs for the reduction of cystospasm and urethral spasm. The cats were anesthetized with 2.5% thiamylal sodium solution. The urinary bladder was emptied of urine by manual expression.

Ten ml of Lugol's solution was then instilled into the bladder by means of a 20-gauge metal lacrimal canula. The Lugol's solution was allowed to remain in the bladder for 3 minutes and then was expressed from the bladder manually. The urethra was ligated with a single strand of fine suture* around the penis at the junction of the prepuce. Pressure within the bladder was measured 12 hours later using a water manometer. A size 6 French 6 foot long polyethylene tube was marked off in centimeters. A

* Vetafil^{R}

1½-inch 19-gauge hypodermic needle was attached to one end of the tubing by means of a gum rubber adapter. This needle was thrust through the abdominal wall directly into the bladder. The other end of the tubing was attached to the ceiling. The reading was taken at the point to which the urine was forced up the tubing. A minimum of restraint was exercised since struggling by the cat or excessive force by the handler resulted in a great increase in vesicular pressure. Cystocentesis was performed with the cat standing or resting in a sternal position. The manometer needle was directed dorsally and caudally from a point on either side of the abdomen about 4 to 5 cm behind the umbilicus and 3 to 6 cm lateral to the midline.

Following the initial pressure reading one of four drugs was administered by intramuscular injection. This was done with the manometer in position. The drugs used and dosages were as follows:

Jenotone^{R} - 1 mg per lb body weight

Octin^{R} - 20 mg per lb body weight

Atropine sulfate - 0.12 mg per kilo body weight

Meperidine hydrochloride - 10 mg per lb body weight

The intravesicular pressure was recorded 30 minutes after administration of the drug.

Two cats, used as controls, were anesthetized and ligated. The solution was instilled in the bladder. Following the 12-hour

pressure reading 2 ml of water was administered. The pressure reading was recorded 30 minutes later. Four groups of 4 cats each were evaluated in identical fashion with each group receiving a different antispasmodic drug.

Forty-seven normal male cats were used in a transmission study. Each received an injection of 10 ml of urine taken from a cat affected with obstructive FUS.

The normal cat was anesthetized with intravenous pentobarbital sodium. A 3-cm area of the skin was shaved and cleansed with soap and an antiseptic applied.

Urine was taken from a cat which was presented for treatment of FUS. In order to qualify as a donor affected cats without a history of previous urinary disease were chosen. Urine was obtained by cystocentesis using a 10 ml sterile glass syringe and a 21-gauge 1-inch needle. The affected cats were not anesthetized prior to cystocentesis. Ten ml of urine was injected directly into the lumen of the bladder of the anesthetized cat via a 22-gauge 1-inch needle inserted through the abdominal wall.

Inoculated cats were observed for a minimum of two months. Routine urinalysis was done prior to injection, 24 hours after injection, and weekly thereafter until eight urinalyses had been made on each cat. Bacteriological studies were conducted at 24 hours, one week, one month, and two months.

A post mortem examination of each urinary tract was conducted at the conclusion of the study.

RESULTS

A total of 300 cats were observed. A careful history was obtained from the owner of each cat. The number of cats in each household wherein an affected cat was found ranged from one to 14.

In eight instances two cats from one household were treated for FUS at the same time or within two weeks of each other. See Table I.

The incidence of FUS according to breed, age and sex is recorded in Tables II, III, and IV.

The diets of affected cats is recorded in Table V. Some cats were fed some table scraps or a mixture of several forms of cat food. The mixture made up the highest percentage of the diet recorded.

Inquiry was made as to how the water was supplied and how often. Twenty-one owners mentioned that their cats drank from the toilet and 14 mentioned that their cats liked to drink running water from a sink in the kitchen or bathroom. These results are recorded in Table VI.

Information regarding the age at which neutering was done is recorded in Table VII.

A history of where the affected cats urinated was obtained and is recorded in Table VIII.

One hundred thirteen cats were diagnosed as having FUS with urinary stasis. These patients showed signs reflecting anxiety, pain, dysuria, and dehydration. Most had an enlarged or erect penis which often was very dark in color. Temperature was normal but pulse and respiration were elevated. Abdominal palpation revealed urinary bladders which were generally firm and painful. Fifty percent of these cats' bladders were the size and consistency of a tennis ball. With experience it became possible to estimate accurately the volume of urine contained. When empty bladders were palpated, they were generally contracted, thickened, and often seemed to be in rigid spasm so that the empty bladder felt like a round, firm ball approximately 3 cm in diameter. The volume of urine in the obstructed bladders varied from 30 to 40 ml. It was noted that when the volume of urine in the obstructed bladder exceeded 150 ml the texture or firmness of the bladder decreased. Those containing 250 to 300 ml became soft and fluctuating. When left alone or placed in a cage these cats would often assume the position of micturition and straining attempts to urinate would be frequently observed.

Vomition was a common sign in cats obstructed for 36 or more hours.

The remaining 117 cats demonstrated signs of cystitis consisting of frequent urination, dysuria and often hematuria. Hematuria was observed in all female cats affected with cystitis and 59% of the unobstructed males. Cats with hematuria had very little if any urine in their bladders. Most appeared alert and unconcerned. Temperature, pulse, and respiration were usually normal. When left alone they would frequently strain and attempt to urinate. Such attempts either resulted in only a few drops of urine or none at all. Male cats rarely demonstrated any change in the penis even though they could be seen licking the organ as the obstructed males did.

THERAPY OF FUS

Obstructed males were considered emergencies. The initial effort was directed toward relief of the obstruction. Early in the study back-flushing was attempted. If successful, the patient was placed on antibiotics and a urinary acidifier. Patients were discharged when micturition appeared normal. Urine acidifiers and antibiotics were dispensed for home administration. Forty-five percent of the patients were treated for recurrent obstruction. With each recurrence it appeared to be more

difficult to relieve the obstruction. On the assumption that the increasing difficulty might be due to the narrowing of the urethral lumen, the back-flushing method was discontinued.

The urine was removed from the bladder by cystocentesis in 51 of 113 obstructed cats. The procedure was avoided when the urinary bladder was distended to the point that it had lost its firmness for fear that the extremely distended thin-wall bladder would rupture when a needle was thrust into it. Such an event occurred on one occasion, and death of the patient ensued.

Following aspiration of the urine from the obstructed bladder, an antispasmodic was administered. Atropine, Jeno-tone^{R}, Octin^{R}, and meperidine hydrochloride were used. The regimen of cystocentesis and antispasmodics was repeated every 24 hours until the patient was urinating normally.

Antibiotic or bacteriostatic drugs were prescribed in all cats. They included oxytetracycline, tetracycline, sulfasoxisol, chloramphenicol, penicillin, streptomycin and Furadantin^{R}. Routine bacteriological methods were employed to detect bacterial infection. Bacteria were identified on the basis of morphologic and colony characteristics. Antibiotic sensitivity tests were conducted. The appropriate antibiotic was then administered for one month.

Ten of 18 obstructed male cats were relieved of their obstructions following one injection of meperidine. The dosage of meperidine was calculated at 10 mg per pound of body weight and administered subcutaneously.

Affected patients were hydrated, using Lactated Ringers Solution subcutaneously at a dosage of 50 ml per Kg three times daily for one to three days. Increase in the urine volume reduced the total amount of struvite production by 25% to 50% and in general was thought to reduce the significance of infection by dilution.

One hundred forty-three cases of FUS received ethylenediamine dihydrochloride, dl methionine, or pHospHaid^{R} for a period of 1 to 2 months. One hundred fifty-seven cases were treated without using acidifying drugs. Patient response did not indicate noticeable difference between the cats receiving a urine acidifying drug and those which received none.

Thirty cats were given a proteolytic enzyme on a daily basis. The drug, Orenzyme^{R}, consisted of 68% trypsin, 30% Chymotrypsin, and 2% ribonuclease. A regimen of one tablet twice daily was followed. The 30 patients receiving the drug were discharged an average of one day earlier than non-treated cats.

Ninety-one patients were discharged with instructions for

the owner to administer additional salt to their pet. The direction was to salt the cat's food daily with the maximum amount of salt that would be palatable. Four of the 91 patients failed to tolerate salted food and were given salt tablets at the rate of 250 mg twice daily. Thirteen percent of the 91 patients had a recurrence of FUS. Thirty-two of 160 cats not receiving salt had recurrences.

Of the 300 cats 32 died as a direct result of FUS. Eight died in the hospital from other causes. Table IX indicates the number of days the affected cats were hospitalized.

LABORATORY RESULTS

Laboratory results were recorded on the initial specimen taken from an affected cat.

Results of urine analysis, routine hematology and bacteriological urine culture were recorded.

The urine analyses included pH values, examination for hematuria, proteinuria, and microscopic examination of urine sediment for leukocytes, bacteria, fat, renal casts, and crystals.

The pH values from 203 cats were obtained using Labstix^{R} except where high protein or blood concentrations resulted in interference. In these cases a pH meter was used. A pH meter

was used exclusively on the urine specimens from 97 cats. Results are recorded in Table X.

Occult blood was present in 279 (93%) of the affected cats.

Proteinuria varying from 25 to 1000 mg % was noted in 294 (98%) of the cats. The mean protein level was 200 mg %.

Microscopic examination of urine sediment revealed significant leukocyturia in 144 (48%) of the specimens examined. Bacteriuria was observed in 60 (20%) of specimens examined. Fat droplets were reported in 84 (28%) of the urine specimens and renal casts in 18 (6%) of specimens.

Triple phosphate or struvite crystals were reported in 249 (83%) of the 300 urine specimens. Of the 113 urine specimens from the FUS cats affected with urinary stasis or urethral obstruction 94 (89.8%) contained struvite crystals. Crystals other than struvite crystals were not reported.

Bacteria assumed to be pathogens were cultured in 63 (21%) of the 300 specimens submitted. These results are tabulated in Table XI.

Of the 113 cats affected with urinary stasis 24 had positive urine cultures. The results are recorded in Table XII.

Blood was taken from the 113 male cats with urinary stasis, and from 57 cats chosen at random from the remaining

non-obstructed cats with FUS. The results of the hematological tests are recorded in Table XIII.

One hundred blood smears, stained for leukocytic differentiation, were examined for the presence of Hemobartonella felis. Twenty-one of the blood smears were found positive for the parasite.

Blood urea nitrogen measurements were made on 185 cats affected with FUS. Seventy-two non-obstructed cats had blood urea nitrogen measurements of 9-22 mg %. One hundred thirteen obstructed male cats had blood urea nitrogen measurements ranging from 12 to 224 mg %. These results are listed in Table XIV.

RESULTS OF THERAPY

The efficacy of the several therapeutic products and techniques was evaluated on the basis of the length of time required to eliminate the signs of dysuria and/or hematuria. The presence or absence of proteinuria, crystalluria, or bacteriuria was not used as a criteria for the evaluation of therapeutic efficacy.

Sixty-five female cats were treated for FUS. All manifested dysuria and hematuria. None died. The average length of time in the hospital was 5 days with a range of 1 to 27 days.

Twenty affected female cats were hospitalized without treatment. Each patient was discharged as soon as gross hematuria and obvious dysuria ceased. When indicated, therapy was continued at home by the owner. The data is listed in Table XV.

The length of time required to effect freedom from signs of dysuria and/or hematuria was recorded for 104 male cats without urinary stasis and in 65 female cats. The drugs given are recorded in Table XVI.

Results of therapy in 113 male cats with urinary obstruction or urinary stasis were extremely variable. Thirty-two patients died as a result of the urinary stasis. These patients were unable to urinate after back-flushing even when digital pressure was exerted in an attempt to force urine through the urethra. Following death of one patient, a necropsy failed to reveal the presence of crystalline solids, fibrinous or sabulous material either in the urethra or the bladder. Neither mechanical obstacle or stricture could be seen.

Following this observation, therapy was directed toward the relief of urethrospasm, urethral swelling or edema. Sixty-two male cats with urinary stasis were back-flushed upon entry into the hospital. Eight cats in which the obstruction and spasm of the urethra were such that it was impossible to force fluid through the urethra into the bladder were referred to

surgery for urethrostomy. Four cats died postoperatively and 4 were released following an average of 13 days of hospitalization.

A variety of therapeutic products were used in treating the remaining 54 cats following back-flushing. Thirty-six cats with obvious urethral obstruction passed urine immediately following instillation of a liquid into the bladder by back-flushing. When dysuria was observed the patient was examined carefully at frequent intervals in order to determine that micturition was occurring. Where urine was not observed in the cat's cage, the patient's bladder was expressed manually. Eighteen of the 54 cases failed to urinate freely following back-flushing. Four of the 18 did not urinate freely because of an apparent cystoplegia. Each had over 200 ml of urine in their bladder and one had a volume of 340 ml. The extreme overdistension apparently produced sufficient atony as to make normal micturition impossible. Such cases were treated with Urecholine^{R} given at a rate of 5 mg every 6 hours per os. Obvious benefit from the use of Urecholine^{R} was not observed. Antibiotic and fluid therapy was also administered. All four patients were later euthanatized when their owners became discouraged with the chronicity, cost, and effort required to empty the urinary bladders. It was necessary to manually express the cats' bladders because of the lack of ability to

urinate even though their urethras were patent. Fourteen cats experienced varying degrees of urinary stasis even though some urine could be voluntarily passed. Nine were discharged following an average of 10 days hospitalization. Two were euthanatized at the request of the owner. Necropsy was performed on these 5 patients and in each a diagnosis of urethrostenosis was made.

Following the observation that not all urinary stasis was caused by obstruction of the urethra with solids, an alternate therapeutic approach was instituted. Fifty-one male cats with urinary stasis were given an antispasmodic instead of back-flushing as the initial treatment.

Eleven cats with obvious urethral obstruction received a single subcutaneous injection of meperidine. Ten of these patients urinated voluntarily 15 to 30 minutes after the injection. One cat urinated freely in 40 minutes, passing an elongated mass of sabulous material after receiving a single 0.5 mg subcutaneous injection of atropine sulfate. Twenty cats were able to urinate voluntarily following a single injection of meperidine and one cystocentesis.

Meperidine followed by cystocentesis was used 4 consecutive times on 4 cats before voluntary micturition was possible. Three cats received meperidine and cystocentesis therapy 8 times.

Three cats received meperidine with cystocentesis 3 consecutive times and then because of extreme depression the meperidine was discontinued but their bladders were emptied 12 times each by cystocentesis. These patients all received Lactated Ringer's Solution by hypodermoclysis until voluntary urination occurred. All patients received antibiotic or bacteriostatic drugs for 2 to 8 weeks. The results of antispasmodic and cystocentesis therapy are recorded in Table XVII.

Supplementary fluid and drug therapy was continued on all patients as deemed necessary. In general, the convalescence of the patients with urinary stasis was comparable to that of the patients exhibiting dysuria without stasis or obstruction.

Twenty-seven cats were necropsied. The direct cause of death is listed in Table XVIII.

A variety of gross lesions was found on necropsy of 27 cats. Kidney pathology was observed in all cases. The pathology ranged from renal atrophy with fibrosis to fatty degeneration, or acute inflammation and parenchymal hemorrhage.

A variety of lesions were noted in the bladders. The bladder epithelium of 17 cats showed necrosis, hemorrhage and edema. While the gross appearance was somewhat different in each of the 17 bladders, outstanding signs were total distortion of the epithelium by hemorrhage, fibrinous material,

ulceration, and swelling. The lesions appeared most obvious on the ventral bladder wall. Six cats revealed shallow irregular eroded areas in the lining of the bladder ranging in size from 2 mm to 1.5 cm. These lesions always occurred in the floor of the bladder wall. A mucoid fibrin-like material was attached loosely to the ulcer-like areas. The thickness of the bladder wall varied. Thickening was due in part to inflammatory edema and in part to fibrosis. Two cats showed extensive thickening of the entire bladder wall without gross changes in the epithelial lining. A correlation between the gross lesions of the bladder and the progress of the cases was not observed. Gross or microscopic lesions were absent in the bladders of two cats; although large amounts of the typical sand-like crystals were present within both bladders.

Urethral pathology was extensive in 15 cats. Three cats were totally obstructed with firm sabulous material extending in length up to 2 cm from the tip of the penis. All 15 cats had gross thickening of the urethra with hemorrhage in the penile and pelvic portions.

Histopathological examination of the kidneys from ten cats revealed lipoid nephrosis. In one case the medulla was congested and contained micro-abscesses. Tissue sections from kidneys of 18 cats showed microliths in the tubules. In the

remaining 9 cases, interstitial nephritis and a moderate nephrosis was the only consistent lesion. None of the kidney tissues were normal.

Epithelial changes in the bladder ranged from minimal inflammatory response to total erosion. In 16 cases myositis with hemorrhage and inflammatory infiltration of the muscle tissue was observed. One cat, which had previously undergone surgical removal of the epithelial lining of the bladder, showed a total regeneration of the transitional epithelium. Death in this case was attributed to interstitial nephritis. In 18 cases, the epithelial and muscular urethral tissues showed inflammatory changes with perivascular accumulations of lymphocytes and neutrophils around the blood vessels in the muscularis layer.

A typical pathological report is recorded in the appendix.

The results of the measurements of pH values and crystalluria of 10 normal cats were recorded in Table XIX. It was noted that 5 cats (cat nos. 2, 3, 5, 8, and 9) produced crystalluria once out of 20 urine specimens and one cat (# 8) had 4 urine examinations which revealed crystalluria. The highest pH value was 7.4 and the lowest was 5.6 with an average pH of 6.3.

Results of the study to evaluate the efficacy of certain

acidifying drugs on pH values and crystalluria were recorded in Table XX. Limited effect was observed in the ability of these drugs to influence crystalluria. However, two cats (# 2, # 3) which normally were found to produce crystalluria did not produce crystals while receiving ethylenediamine dihydrochloride. Simultaneously the 2 cats had a mean decrease of .4 and .32 in pH values.

Results of the study concerning the efficacy of ethylenediamine dihydrochloride and tetrasodium ethylenediamine tetraacetic acid (EDTA) are recorded in Tables XXI - XXV.

Ethylenediamine dihydrochloride had no significant effect on crystalluria although pH values decreased an average of 0.3. Cats receiving EDTA showed no change in pH values, however, an obvious reduction in crystalluria was observed. A dosage of 80 mg three times daily eliminated crystalluria. Cats receiving 40 mg of EDTA three times daily had slight softening of feces. Those receiving 80 mg t.i.d. had profuse watery diarrhea. Diarrhea ceased when the drug was withdrawn. Other adverse signs were not observed. An attempt to administer EDTA in food proved impractical because of poor palatability.

Results of the study to evaluate the efficacy of 4 antispasmodic drugs are recorded in Table XXVI. The results are expressed in number of centimeters of water pressure lowered

after the injection of one of the 4 drugs.

Results of attempts to transmit the disease were essentially negative. Clinical signs of FUS were observed in one case. This cat produced a bloody urine and demonstrated signs of dysuria on the second day following injection of urine from an FUS cat. A pure culture of hemolytic E. coli was grown on blood agar from a specimen taken on the second day. The donor cat also had an infection which was shown to be due to E. coli. Signs in the experimental cat disappeared on the third day while the donor cat revealed dysuria and hematuria for 8 days. Repeated attempts to demonstrate bacteriuria or urine abnormalities failed. Urine analysis, bacteriological culturing, gross and micropathological studies failed to reveal evidence of successful transmission of FUS.

DISCUSSION

No significance is attached to the relationship of the occurence of FUS and the environment of breed of affected cats. However, it seems apparent that, as Foster (24) has reported, FUS is an age-related disease. Although a range in age of 6 months to 17 years was noted in this study, most affected cats were from 2 years to 6 years in age with an average of 3.8 years. Data from the examination of 300 cases verifies Foster's (24)

observations regarding the lack of significant relationship between incidence and sex. Two hundred forty-five males, 137 castrates and 108 entire, were observed. Sixty-five females, 51 spayed and 14 entire, were also seen. While the incidence in males was much higher than that in females, it is clear that FUS is not limited to the male. Castration of the male or spaying of the female did not materially change the incidence, nor did the age at which it was done.

Inquiry was made as to the cat food sales in the immediate area. Eight supermarkets were surveyed. It was revealed that an average of 24 cans of all varieties of cat food was sold to every box or container of dry cat food. It was noted that an average of 10 to 20 daily feedings were in each container of dry food. One can provides food for 1 to 3 meals. Thus it would seem that a considerably larger percentage of canned food than dry food is consumed by local cats. Diet appeared to have an influence on incidence of FUS. One hundred forty-seven cases were seen which had been fed a dry cat food. This was compared with 81 cats which were fed canned cat food. It may be that cats eating a dry cat food had a lower water intake and thus a tendency toward crystalluria.

Crystalluria was noted in 83% of 300 cats with FUS. Nine percent without crystalluria were fed canned cat food. Three

percent of cats with no crystalluria were fed dry cat food. The data seems to point to an increased incidence of crystalluria in cats that regularly eat a dry cat food.

Unusual water drinking habits of cats may also be a factor in the incidence of FUS. Freedom of the cat to urinate in a litter box inside or outside was ascertained on most of the 300 cats with FUS. No pattern could be detected which would indicate any relationship with the disease.

While clinical signs were very diversified the disease could be divided into two categories on bases of the signs observed. Those cats with hematuria and dysuria only comprise one group and those with urinary stasis comprise the second group. It was noted that cats in the first category might recover spontaneously or if males become obstructed and require emergency treatment. A clinical sign of considerable significance was the extreme pressure created in the bladder by the violent spasm of the bladder.

Pressures over 100 cm of water were recorded in 13 male cats with urinary stasis. Histopathological examination of bladder sections of 3 such cats revealed massive numbers of erythrocytes infiltrated between the muscle fibers. The obvious pain exhibited by most affected cats is an important sign and may well be the triggering influence for cystospasm and urethrospasm.

It was observed that in those patients that maintained a firm or spasmodic bladder for several days, in spite of therapy and whether or not the urethra was patent, a prolonged convalescence was the end result. It was not fully understood whether the spasm of the bladder muscle was a result of pain or of some other influence. It was obvious, however, that this dramatic, powerful smooth muscle spasm was a highly significant phenomenon, and that the early recovery of the feline patient with this sign was dependent on how quickly and how completely the spasm could be aleviated.

Therapy for the 300 FUS cats was administered according to the signs demonstrated. Antibiotic or bacteriostatic drugs were used in most cases on the premise that even though known bacterial infection was not present, the involved tissues would have an increased susceptibility to infection. Male cats with urinary stasis were routinely handled as an emergency. The first 50% of the obstructed cats were treated by back-flushing or canulizing the penile urethra. Following the observation that more than 50% of these cases were being returned with a recurrent obstruction it was postulated that repeated trauma from manipulations of the penis and urethra by means of a canula or catheter was responsible for narrowing of the penile urethra. It was theorized that the manipulation produced sufficient

trauma to stimulate fibrosis and/or loss of normal urethral elasticity. Fifty-one of the 113 obstructed cats were then treated without back-flushing. Meperidine or atropine was administered parenterally. If urination did not occur voluntarily, the urine was removed via cystocentesis. Back-flushing was not done unless the above method was unsuccessful.

Surgical alleviation of the obstruction by urethrostomy was only done as a last resort. Immediate results were better when the back-flushing method was used. However, the long term disadvantage of a possible urethrostenosis produced by urethral back-flushing negates the routine use of this method. Ten of 18 cats with urinary stasis urinated following a single injection of meperidine. This would appear significant when considered in the light of reports from the 5 necropsies in which no solid material was found lodged in the urethra.

It must be assumed that some male cats demonstrating typical signs of FUS with urinary stasis do not have urethral obstruction in the sense that solid material is occluding the urethra. The 5 cats without demonstrable urethral obstruction also failed to show freely movable particles in the bladder urine which might have moved into the neck of the bladder during attempts to micturate. Therefore, it is theorized that the inability to urinate through an unobstructed urethra is caused by urethrospasm

and/or urethral swelling. The repeated gross and microscopic examination of urethral tissues did not reveal tissue or cellular changes which could be linked to the phenomenon. It is possible that acute dysuria accompanied by constant and vigorous straining efforts might so affect the vascular circulation of the urethra that a circulatory edema of the epithelial and muscular tissues of the urethra occurred and so eliminated urethral patency. An important aspect in therapy was the use of parenteral fluids. Over-concentration of urine resulting from water deprivation seems to be significant and should be a major consideration in therapy of cats with or without urinary stasis. An impression was gained that the proteolytic enzyme, Orenzyme^{R}, seemed to shorten the course of the disease, however, specific supporting data was not obtained. One hundred twenty-five cats were discharged with instructions to the owners to add table salt to the diet. Examination of the data relating to the frequency of recurrence of signs of FUS reveals that recurrence occurred on an average of every 7.5 months for cats which did not and 11.2 months for those cats which did receive salt in the diet.

One 4-year-old male cat was successfully treated for obstructive FUS on three separate occasions. This cat had also been treated for non-obstructive FUS 5 times. The interval

between recurrences was 2 - 3 months. The first episode occurred in May of 1967 and the most recent episode was in March, 1968. Following a 4-day convalescence in the hospital the patient was released to his owner with instructions to add table salt to every feeding of the canned cat food which had always been used. These instructions were carefully carried out and further recurrences were not observed until December, 1969. Three days prior to this attack the owner had left the cat with a friend who neglected to give the prescribed salt. Following apparent recovery the cat was again released to his owner. In Feburary, 1970, a similar situation occurred and the salt was not given. Five days later the patient was in the hospital with obstructive FUS. No home medication had been used except salt following the March, 1968, episode. Definitive conclusions cannot be drawn from a single case; however, it does lend support to the hypothesis that salt may be a significant therapeutic aid.

Owners of 10 cats treated for FUS in this series reported that the addition of 1/8 teaspoon salt in their cats' diet each day increased total water consumption from 25 to 50%. This observation was made over a 12-month period of time.

Information obtained from the laboratory was especially significant relative to the existence of bacteria. Bacteria

were cultured from the urine of 21% of the 300 affected cats; 79% had no apparent infection. Bacteriuria was noted by microscopic examination of centrifuged urine sediment in six instances in which organisms were not cultured by routine procedures. Sixteen urine specimens from affected cats, 2 of which had apparent bacteria in centrifuged portions, were cultured in anaerobic media. All 16 anaerobic cultures were negative. The presence of bacteria in the urine which yields negative results in culture suggests the possibility that a bacterial pathogen may multiply in the urine-free environment of the transitional epithelium and when released into the bladder's urine may be lysed or altered in such a way as to make the pathogen unable to grow on laboratory media. Bacteria were not observed in the tissue sections of the bladder.

Eighty-three percent of urine specimens from the 300 cases contained typical struvite crystals. The significance of crystals from the nutritional or etiologic viewpoint is difficult to evaluate. It is obvious that certain cats will produce these typical crystals in the urine. Even when a high volume of crystals is regularly observed in the urine of normal cats it cannot be assumed that urinary disease is or ever has been present. The massing or aggregation of the crystals in the urethra leads to a localized pathological element which is

responsible for the obstruction which leads to urinary stasis. Studies did not indicate whether the cystospasm or urethrospasm was initiated by urethral obstruction or vice versa. No information was obtained as to why some affected cats did not produce crystals. Fifty-one cats of the 300 affected cats were free of crystalluria at the times the specimens were examined.

Difficult to evaluate was the usually low blood urea nitrogen values taken from cats which had been unable to urinate for 48 to 72 hours. Three male cats were specifically studied in this respect. All 3 had urinary stasis and an estimated 10 to 20 ml of urine in their bladders. This volume increased approximately 50 ml every 12 hours. Blood urea nitrogen values averaged 18 mg % at the time of entry. Twenty-four hours later the blood urea nitrogen averaged 23 mg %. Forty-eight hours later the average blood urea nitrogen was only 35 mg %. At 72 hours the blood urea nitrogen measurement averaged 87 mg %. Urine volume averaged 220 ml. These cats did not urinate, nor were they treated in any way for 72 hours. Five percent of the 113 obstructed cats in this series had much higher blood urea nitrogen values. The unusual compensating ability of cats during urinary stasis is notable.

Histopathological evidence of microcrystals forming in the renal tubules suggests at least some crystalluria originates in

the kidney. Microcrystals were occasionally noted in the distal and collecting tubules. There was no evidence of tubular damage occurring as a result of the intrarenal crystals. Studies of nutritional and physiological aspects of the feline urinary system in relation to the production of struvite in the urine are sorely needed.

The presence of major pathological changes in the urethra emphasizes the extreme care that the clinician must exercise in manipulating this tissue. Of major importance were the pathology reports of urethrostenosis in cats which had received repeated back-flushing of the urethra. One 10-year-old male cat which died following an FUS obstructive crisis was observed to have thickening of the penile urethral epithelium and fibrosis of the lamina propria. This cat had been back-flushed a total of 18 times. The progressive decrease in size was reflected by the urine stream becoming increasingly small. Clinically an important prognostic sign was the size of the urine stream.

Those cats with very small streams became obstructed with smaller amounts of struvite. A significant conclusion is that every precaution should be taken to avoid trauma to the urethra because of the possibility of producing a permanently narrowed lumen.

Several trials demonstrated the lack of efficacy of certain urinary acidifiers. Ethylenediamine dihydrochloride reduced the pH values of normal cat urine by .4%. This was not considered clinically significant especially in view of the fact that 60% of the affected cats with crystalluria had acid urine pH. Ethylenediamine dihydrochloride reduced crystalluria by approximately 20% in normal crystal producing cats. The mechanism by which the crystalluria is reduced is not known. One speculation is that the drug has some type of chelating action. Certainly the acidification action of the drug was not sufficient to reduce the crystalluria.

EDTA when given orally at the rate of 80 mg three times daily eliminated crystalluria in 7 cats; however, the accompanying diarrhea essentially eliminated the product for therapeutic use. Lower dosage levels eliminated some crystalluria.

Meperidine hydrochloride proved to be a superior antispasmodic when used in cats with experimental cystitis and urethral obstruction. Meperidine was compared with Jenotone^{R}, Octin^{R}, and atropine sulfate. The clinical success of meperidine was considered highly significant in view of the findings that back-flushing was contraindicated. It is suggested that the antispasmodic action relaxes a urethrospasm and enables an obstructed cat to urinate. It should be noted that meperidine

is a well known analgesic. It is possible that the superiority of meperidine over atropine was due to the combined analgesic and antispasmodic action of meperidine. Pain associated with some aspects of the syndrome may be a triggering influence producing cystospasm and/or urethrospasm.

SUMMARY

Three hundred cats affected with FUS were observed. An anamnesis was obtained and recorded on each cat. Treatments were administered and evaluated. Pathological data was recorded.

An observation that the technique of back-flushing stimulated urethral tissues to thicken prompted an investigation of other methods for relieving acute urine stasis. Meperidine hydrochloride was found effective when given to obstructed cats in place of back-flushing. Cystocentesis enabled the operator to relieve urine pressure until normal urination occurred.

Urine acidifying drugs proved to be of little value in lowering urine pH or reducing crystalluria.

Female cats were commonly affected with the syndrome. Evidence is suggested that the syndrome was self-limiting in some females.

Attempts to transmit the syndrome by injecting urine from affected cats into the bladders of normal males were routinely unsuccessful.

Experience with recovered males suggests that the volume of water consumed may be correlated to the possibility of recurrence. Results obtained indicated that the use of sodium chloride as follow-up therapy reduced the incidence of recurrence by increasing total water consumption.

TABLE I -- NUMBER OF CATS IN THE HOUSEHOLD

One cat only	179
Two cats	49
Three cats	13
Four cats	6
Five cats	0
Six cats	3
Seven to ten cats	0
Eleven cats	1
Twelve to thirteen cats	0
Fourteen cats	1
No history obtained	48

TABLE II -- BREED OF CAT

Domestic shorthair	149
Siamese	79
Longhair (Persian, etc.)	19
No history obtained	56

TABLE III -- AGE OF THE AFFECTED CATS

6 months	1
9 months	2
1 year	13
2 years	51
3 years	43
4 years	50
5 years	35
6 years	29
7 years	7
8 years	13
9 years	9
10 years	5
11 years	5
12 years	1
13 years	3
14 years	1
15 years	1
16 years	1
17 years	1
History not obtained	29

TABLE IV -- SEX OF CATS WITH FUS

Males	245
Castrated males	137
Entire males	108
Females	65
Spayed females	51
Entire females	14

TABLE V -- DIET OF THE AFFECTED CATS

Canned dog food	10 cats
Canned cat food (standard)	62 cats
Canned cat food (gourmet)	19 cats
Dry cat food	147 cats
Mix dry and canned food	12 cats
Glandular meat (liver-kidney)	20 cats
No history obtained	30 cats

TABLE VI -- WATER CONTAINER

Plastic dish	14
Metal pan	37
Ceramic container	62
Bathroom toilet	21
Running water from sink	14
No history obtained	52

TABLE VII -- AGE AT WHICH NEUTERING WAS DONE

Males	
6 months	34
1 year	82
2 years	11
3 years or more	5
No history obtained	5
Females	
6 months	10
1 year	35
2 years	3
3 years or more	1
No history obtained	2

TABLE VIII -- ENVIRONMENT OF AFFECTED CATS

Access to outside through a "cat door"	6
Used no litter box but had to be let outdoors	37
Used litter box only inside	152
Used litter box inside but could go outside	81
No history obtained	22
Farm or outdoor cats	2

TABLE IX -- LENGTH OF HOSPITAL STAY

Number of Cats	Number of days Hospitalized
1	2
2	19
3	82
4	49
5	32
6	20
7	16
8	10
9	6
10	5
11	1
12	4
13	1
14	3
15	4
17	2
23	1
34	1

THIS BOOK CONTAINS NUMEROUS PAGES WITH DIAGRAMS THAT ARE CROOKED COMPARED TO THE REST OF THE INFORMATION ON THE PAGE.

THIS IS AS RECEIVED FROM CUSTOMER.

TABLE X -- pH Values of 300 Affected Cats

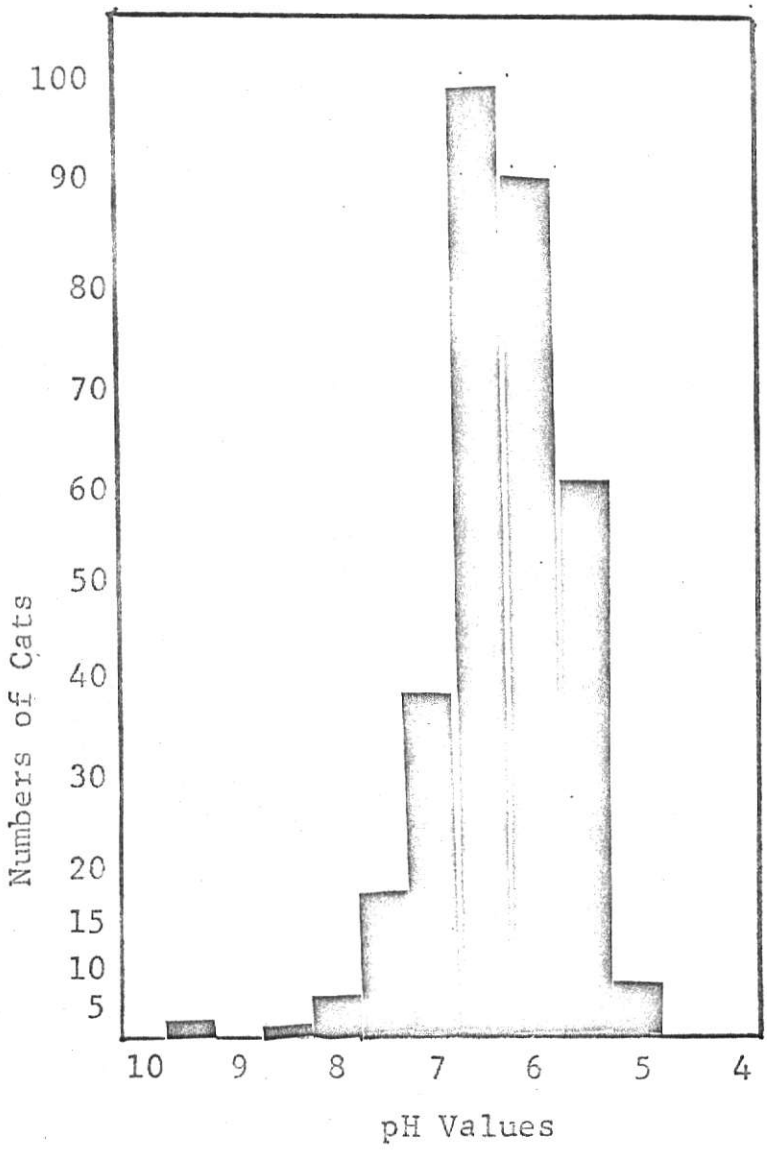


TABLE XI -- RESULTS OF BACTERIAL CULTURE

Pathogen	No. of Cats	%
Escherichia coli	28	9.3
Staphylococcus sp.	15	5.0
Proteus sp.	8	2.7
Streptococcus sp.	7	2.3
Aerobacter sp.	3	1.0
Pseudomonas sp.	2	0.7

TABLE XII -- RESULTS OF URINE CULTURE IN URINARY STASIS

Bacterial Pathogen	No. of Cats
Escherichia coli	10
Staphylococcus sp.	2
Proteus sp.	5
Streptococcus sp.	6
Pseudomonas sp.	1

TABLE XIII -- HEMATOLOGICAL DATA

	Average	Low	High
Hemaglobin Gm. %	11.3 ±	6.9	16.2
Packed cell volume %	36.0	18.2	46.1
Total Leukocytes	10,300	5,100	28,000
Differential Leukocyte Count			
Neutrophiles	65%		
Lymphocytes	32%		
Eosinophils	2%		
Basophils	---		
Monocytes	1%		

TABLE XIV -- BLOOD UREA NITROGEN LEVELS

No. obstructed Cats	BUN in Mg %	No. obstructed Cats	BUN in Mg %
1	224	1	37
1	160	1	35
1	144	1	36
1	111	3	32
1	97	1	30
1	94	2	28
1	80	1	27
1	74	1	26
2	60	2	25
1	57	3	23
1	55	3	22
1	54	10	21
1	53	5	20
1	50	5	19
2	49	9	18
1	45	15	17
1	44	6	16
3	43	6	15
1	42	8	14
1	40	1	13
		5	12

TABLE XV -- THERAPY AND LENGTH OF HOSPITAL STAY OF FEMALE CATS WITH FUS

No. of Female Cats	Pharmaceuticals administered	Ave. No. of Days of Hosp.
7	Chloramphenical and Octin[R]	3
15	Nitrofurantoin and ethylene-diamine dihydrochloride	6
5	Bactrovet[R] and dl-methionine	5
10	Manisul Minitabs[R] and Jenotone[R]	6
8	Tetracycline and ethylene-diamine dihydrochloride	8
20	No treatment	5

TABLE XVI -- THERAPY AND RESULTS IN NON-OBSTRUCTED CATS

No. of Cats	Pharmaceuticals Administered	Ave. No. of Days of Hosp.
10	Gantrisin[R]	4
4	Gantrisin[R] and Octin[R]	3
12	Chloramphenicol	5
8	Chloramphenicol and ethylenediamine dihydrochloride	5
6	Nitrofurantoin	7
10	Nitrofurantoin and meperidine	3
4	Nitrofurantoin and Octin[R]	5
2	Tetracycline	2
4	Tetracycline and Jenotone[R]	3
2	Tetracycline and sodium bicarbonate	6
4	Bactrovet	3
2	Bactrovet and Curecal[R]	3
6	Bactrovet and Placidyl[R]	5
2	Meperidine and sodium chloride	1
6	No drugs given	4
2	Placidyl	4
4	Sodium bicarbonate	5
6	Curecal[R] and meperidine	3
2	Curecal[R] and Octin[R]	6
2	Curecal[R] and atropine	6
6	Manisul Minitabs[R]	3

TABLE XVII -- RESULT OF ANTISPASMODIC/CYSTOCENTESIS THERAPY

No. of Cats	Antispasmodic Drug	No. of Cystocentesis Per Patient	Ave. No. of Days of Hosp.
10	meperidine	0	5
1	atropine	0	2
20	meperidine	1	6
8	meperidine	2	9
2	atropine	3	6
4	meperidine	4	10
3	meperidine	8	8
3	meperidine	12	7

TABLE XVIII -- RESULTS OF NECROPSY

No. of cats	Cause of Death
4	anesthetic death
2	rupture of bladder
3	leakage of bladder (due to cystocentesis)
7	nephritis and uremia
10	uremia due to urine stasis, urethrospasm or urethrostenosis
1	cause of death undetermined

TABLE XIX -- pH Values and Crystalluria of 10 Normal Male Cats

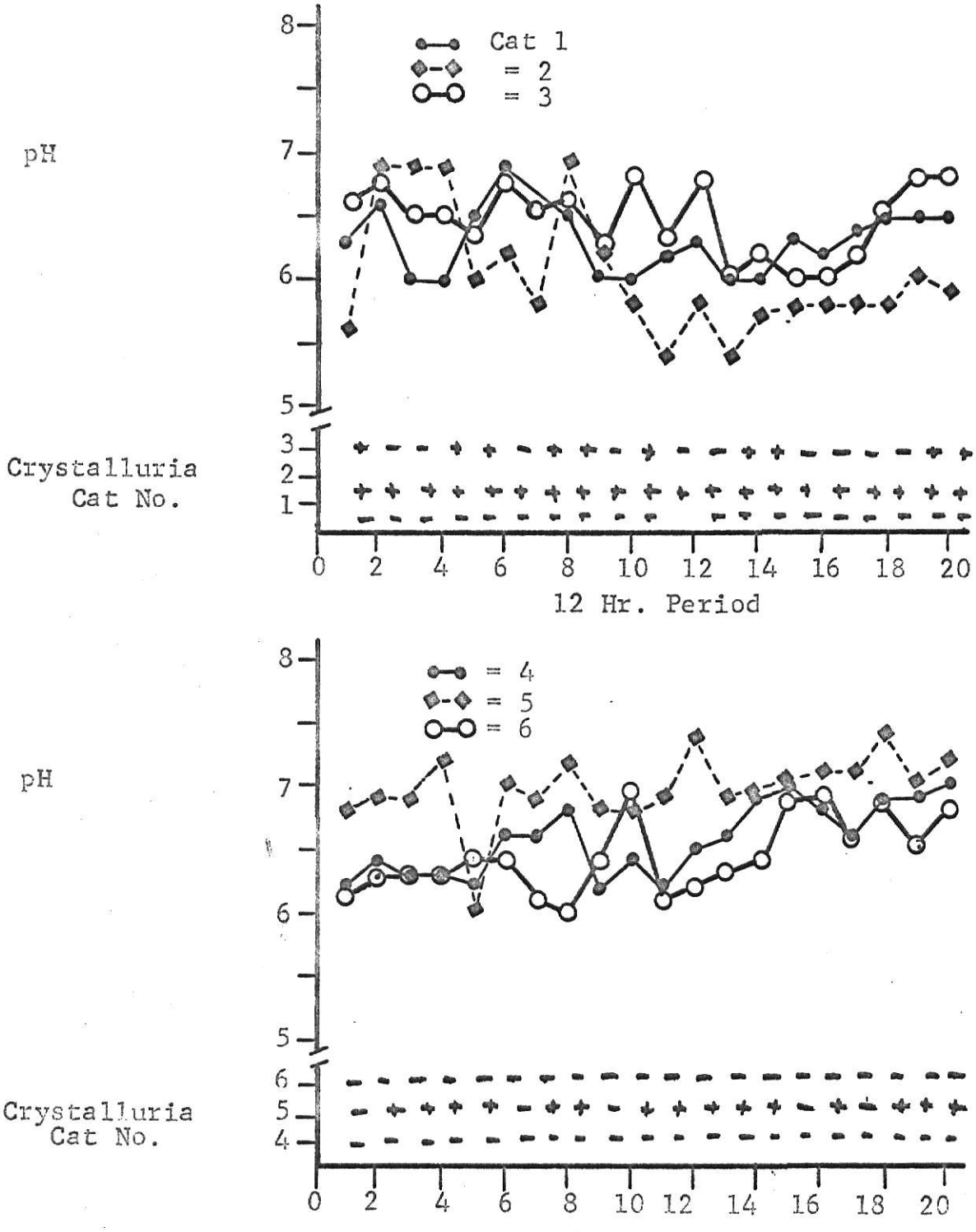
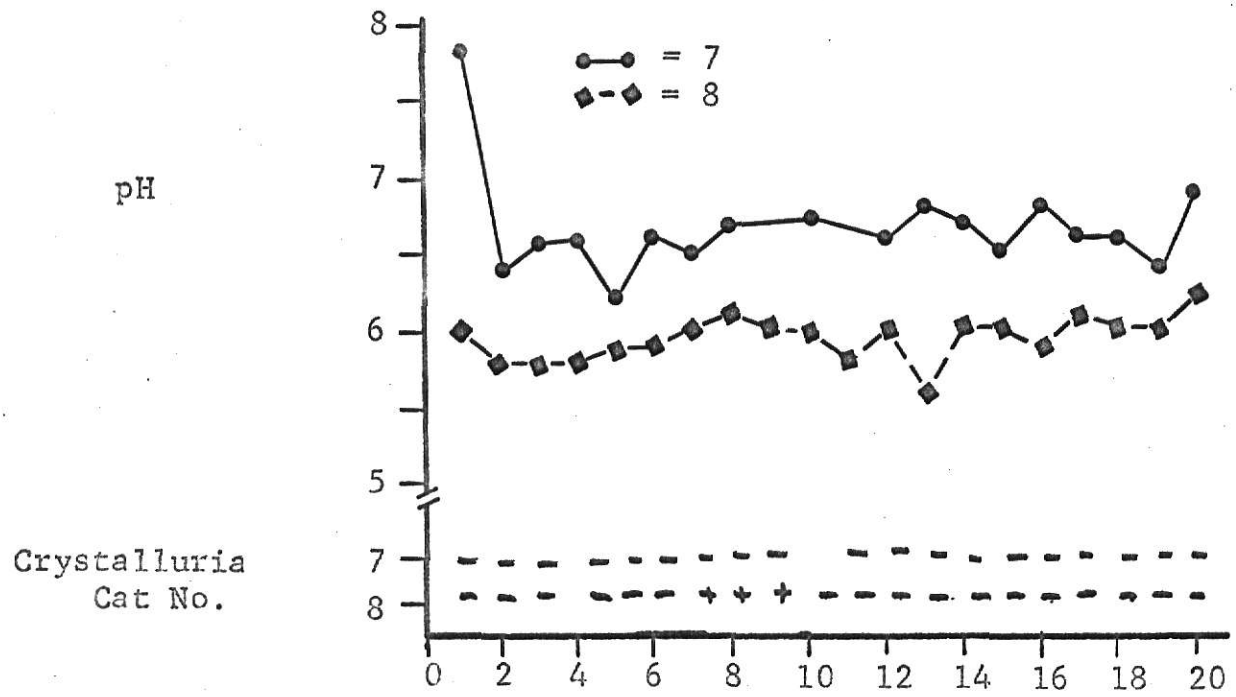


TABLE XIX (Continued)



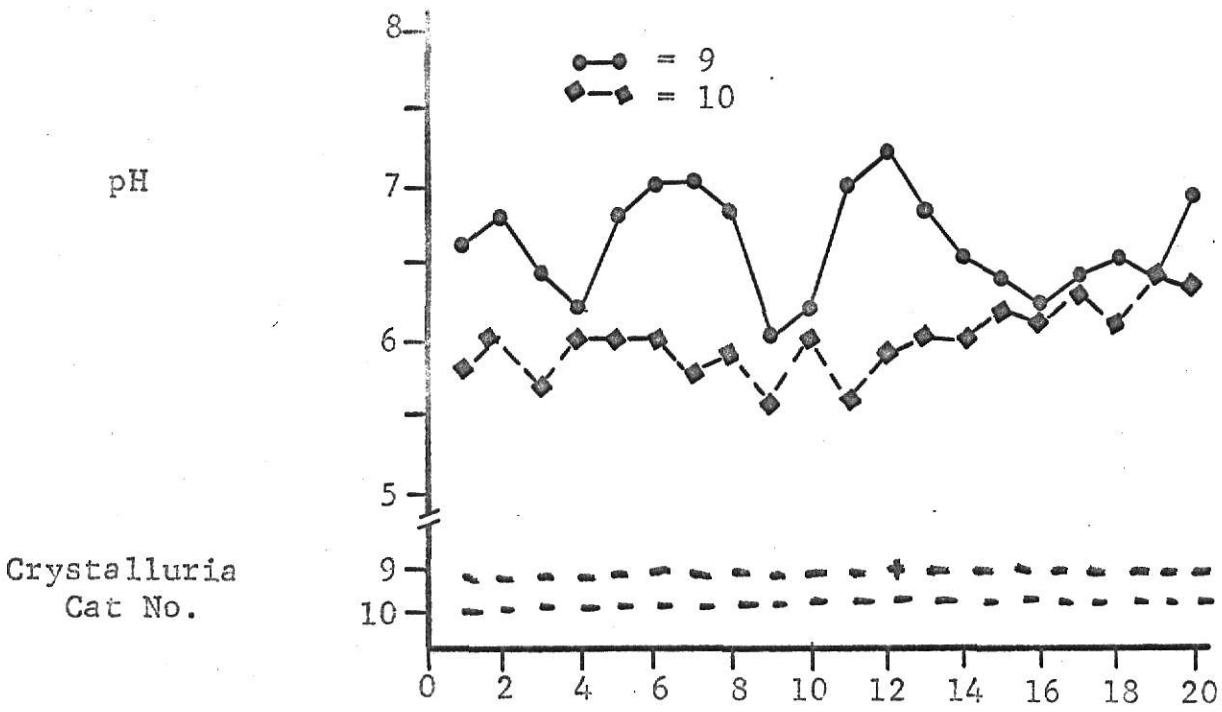


TABLE XX -- EFFECT OF DRUGS ON pH AND CRYSTAL FORMATION IN 10 NORMAL MALE CATS

Group A -- Ethylenediamine dihydrochloride 100 mg t.i.d.

Cat	Day 1		Day 2		Day 3		Day 4		Day 5	
	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.
1	6.0-	6.0-	5.8-	5.6-	5.7-	5.7-	5.4-	5.6-	5.6-	5.5-
2	5.5-	5.4-	5.4-	5.5-	5.6-	5.5-	5.6-	5.4-	5.3-	5.2-
3	6.4-	6.3-	6.5-	6.0-	6.2-	6.1-	6.0-	5.9-	6.1-	6.0-

Group B -- dl Methionine 0.2 gm t.i.d.

Cat	Day 1		Day 2		Day 3		Day 4		Day 5	
	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.
4	6.0-	5.8-	6.0-	5.8-	5.8-	5.7-	5.6-	5.7-	5.6-	5.4-
5	6.9+	6.5	6.4+	6.5	6.2+	6.4+	6.4	6.6	6.0	6.5+
6	6.6-	6.4-	6.5-	6.4-	6.8-	6.9-	6.7-	6.6-	6.4-	6.4-

Group C -- ascorbid acid 250 mg t.i.d.

Cat	Day 1		Day 2		Day 3		Day 4		Day 5	
	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.
7	6.2	6.2	6.0	6.2	6.8	6.7	7.1	6.9	6.6	6.7
8	5.5	5.4	5.6	5.1	5.7	5.6	5.4	5.4	5.5	5.4
9	6.9	7.0	6.7	6.5	6.0	6.2	6.1	5.9	6.3	6.1

Group D -- Control Cat

Cat	Day 1		Day 2		Day 3		Day 4		Day 5	
	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.
10	6.0	6.1	5.8	6.0	6.2	6.0	6.5	6.4	6.1	6.3

(The + and - indicate the presence or absence of crystalluria)

TABLE XX (Continued)

Group A -- Acetylsalicylic acid -- 0.5 gm b.i.d.

Cat	Day 1		Day 2		Day 3		Day 4		Day 5	
	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.
1	5.8	5.6	5.4	5.4	6.5*	7.0	6.8	6.4	5.8	5.6
2	5.4+	5.3	5.8+	5.4+	5.7	5.4+	5.4	5.3+	5.5+	5.4
3	6.3+	6.3+	6.4+	6.4+	6.0+	6.4+	6.1+	5.8+	5.7+	5.9+

Group B -- pHospHaid -- 0.25 gm b.i.d.

Cat	Day 1		Day 2		Day 3		Day 4		Day 5	
	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.
4	5.5-	5.4-	5.6-	5.6-	5.8-	5.6-	5.4-	5.5-	5.4-	5.5-
5	6.9+	6.8+	6.5	6.6+	6.4	6.6	6.9+	6.6	6.4+	6.2
6	6.5-	6.4-	6.4-	6.3-	6.6-	6.6-	7.0-	6.8-	6.5-	6.4

Group C -- Ammonium Chloride 0.5 gm bi.d.

Cat	Day 1		Day 2		Day 3		Day 4		Day 5	
	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.
7	5.7-	5.4-	5.0-	5.0-	5.4-	5.3-	5.6-*	6.3-	6.9-*	7.2-
8	5.2-	5.0-	6.8-*	6.9-*	7.0-*	7.2-*	8.4-*	8.5-*	7.0-	6.1-
9	6.9-	6.4-	6.4-	6.0-	5.9-*	6.9-*	7.6-*	0*	0	7.2-

Cat 9 -- days 3 & 4 depressed. No urine obtained a.m. day 4 -- a.m. day 5

Group D -- Control Cat

Cat	Day 1		Day 2		Day 3		Day 4		Day 5	
	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.	a.m.	p.m.
10	5.9-	5.8-	6.0-	6.1-	6.4-	6.0-	6.5-	6.0-	6.1-	5.8-

* Cat vomited

TABLE XXI

Ethylenediamine dihydrochloride 100 mg

Cat	Day 1	pH	2	pH	3	pH	4	pH	5	pH	6	pH
1	2	6.5	3	6.4	2	6.6	2	6.0	1	6.2	1	6.2
2	4	6.8	4	6.8	4	7.0	4	6.6	2	6.2	0	6.0
3	2	6.2	2	6.3	2	6.0	2	6.4	2	6.1	1	6.1
4	4	7.5	4	7.2	4	7.0	4	7.0	4	6.9	3	6.8
5	2	6.9	1	7.0	1	7.1	1	6.4	0	7.2	0	7.0
6	3	6.1	1	5.8	1	5.8	1	5.6	0	5.8	0	6.0
7	2	6.0	1	5.6	1	5.8	1	5.7	1	5.9	1	5.9

TABLE XXII

EDTA 20 mg

Cat	Day 1	pH	2	pH	3	pH	4	pH	5	pH
1	2	6.4	2	6.2	2	6.5	2	6.6	2	6.6
2	4	7.0	4	6.8	4	6.6	4	6.9	4	6.8
3	1	5.9	1	6.0	2	6.0	2	6.1	1	6.0
4	3	6.9	3	7.3	4	7.0	3	7.0	3	7.0
5	1	6.8	0	6.6	1	6.9	1	7.0	1	6.6
6	3	5.5	2	5.4	2	5.9	2	5.9	2	5.8
7	1	5.6	1	6.0	1	6.1	0	6.0	1	6.0

TABLE XXIII

EDTA 40 mg

Cat	Day 1	pH	2	pH	3	pH	4	pH	5	pH
1	2	6.0	1	6.3	2	6.1	2	6.4	1	6.0
2	4	7.4	4	7.0	3	7.0	3	6.6	3	6.9
3	0	5.6	1	5.9	1	5.9	1	6.0	1	5.9
4	3	7.0	3	6.8	3	6.8	3	7.0	3	7.0
5	1	6.5	1	6.8	0	6.4	0	6.6	0	6.6
6	2	6.0	2	5.9	2	5.6	2	6.1	1	5.8
7	0	5.4	0	5.4	1	5.8	0	5.5	0	5.6

TABLE XXIV

EDTA 60 mg

Cat	Day 1	pH	2	pH	3	pH	4	pH	5	pH
1	2	6.2	1	6.4	0	6.4	0	6.2	0	6.3
2	3	6.8	2	7.0	2	6.9	1	7.0	1	7.1
3	0	5.9	0	6.1	0	6.0	0	6.2	0	6.5
4	3	7.0	2	6.6	2	6.9	1	7.1	1	6.9
5	0	6.8	1	6.8	0	6.6	0	6.9	0	6.8
6	2	6.0	0	5.8	0	6.1	0	6.0	0	6.2
7	0	6.0	0	5.8	0	5.9	0	5.6	0	5.7

TABLE XXV

EDTA 80 mg

Cat	Day 1	pH	2	pH	3	pH	4	pH	5	pH
1	0	6.4	1	6.0	0	6.2	0	6.2	0	6.0
2	1	7.0	0	6.9	1	7.2	1	7.0	1	6.9
3	0	6.0	0	6.2	0	6.4	0	6.0	0	6.4
4	1	7.2	0	7.2	1	7.4	0	7.0	0	7.2
5	0	6.9	0	7.0	0	7.1	0	6.9	0	7.1
6	0	5.6	0	5.9	0	5.8	0	6.0	0	6.0
7	0	5.9	0	5.6	0	5.9	0	6.2	0	6.4

TABLE XXVI -- EFFECTS OF ANTISPASMODIC DRUGS ON VESICULAR PRESSURE

Jenotone®

Cat	Cm Decrease in Vesicular Pressure
1	2
2	0
3	7
4	3

Octin®

Cat	Cm Decrease in Vesicular Pressure
5	4
6	9
7	0
8	6

Atropine

Cat	Cm Decrease in Vesicular Pressure
9	11
10	16
11	4
12	7

Meperidine

Cat	Cm Decrease in Vesicular Pressure
13	18
14	12
15	27
16	28

Control

Cat	Cm Decrease in Vesicular Pressure
17	0
18	0

BIBLIOGRAPHY

1. Johnson, J.: Cystitis in the Feline Male. Southeastern Vet., 14, (1963): 41.

2. Lumb, W. V.: Cystitis and Urolithiasis. Vet. Med., 50, (1955): 26-30.

3. Stansbury, R.: Feline Urolithiasis. Mod. Vet. Pract., 40, (1959): 55.

4. Whitehead, J. E.: Urolithiasis in the Feline. Small Anim. Clin., 1, (1961): 307-319.

5. Fishler, J: Urolithiasis or Urethral Impaction in the Cat. J.A.V.M.A., 127, (1955): 121-123.

6. Krabbe, A.: Urolithiasis in Dogs and Cats. Vet. Rec., 61, (1949): 751.

7. Frost, R.: Cystic Calculi in the Cat. Vet. Rec., 61, (1958): 765-766.

8. Bower, C.: Urinary Calculi in Castrated Male Cats. North Am. Vet., 38, (1957): 224.

9. Meier, F. W.: Urethroadenocystitis in the Male Cat. J.A.V.M.A., 151, (1967): 1059-1071.

10. Swift, W.: Urethral Stenosis in a Cat. Vet. Med., 64, (1949): 516.

11. Meier, F. W.: Urethral Obstruction and Stenosis in the Male Cat. J.A.V.M.A., 137, (1960): 67-70.

12. Armistead, W.: Urethral Obstruction in Male Cats. North Am. Vet., 34, (1953): 276.

13. Carbone, G. M.: Phosphocrystalluria and Urethral Obstruction in the Cat. J.A.V.M.A., 147, (1965): 1195-1200.

14. Wilkinson, G. T.: Disease of the Cat. Pergamon Press Ltd., Oxford (1966): 128.

15. Whitehead, J. E.: Diseases of the Bladder in Feline Medicine and Surgery, Edited by E. J. Catcott. American Veterinary Publications, Inc., Santa Barbara, California, (1964): 270-276.

16. Dickensen, C. D., and Scott, P. P.: Failure to Produc Urinary Calculi in Kittens by the Addition of Mineral Salts, Derived from Bone Meal, to the Diet., Vet. Rec. 68, (1956): 858-859.

17. McCully, R. M. and Lieberman, L. L.: Histopathology in a Case of Feline Urolithiasis. Can. Vet. Jour., (Feb. 1961): 52-61.

18. Holzworth, Jean: Disorders of the Urinary System. Cornell Vet., 53(1), (1963): 157-159.

19. Schein, Herbert W.: Anuria and Dysuria in the Male Cat. Vet. Med., 46, (1951): 372.

20. Rich, L. J.: Urethral Obstruction and Urolithiasis in Cats. Kirks Current Vet. Therapy III, W. B. Saunders, Philadelphia, (1968): 635-638.

21. Rich L. J.: Feline Urethral Obstruction: Mineral Aspects. Am. J. Vet. Res., (1968): 2149-2156.

22. Gershoff, S. N.: Vitamin B6 Deficiency and Oxalate Nephrocalcinosis in the Cat. Am. J. Med., 27, (1959): 72.

23. Bloom F.: Report number 1 submitted to Atlas Canine Products, Inc., Glendale 27, N. Y., (Jan. 1, 1958): 1-3.

24. Foster, S. J.: The Urolithiasis Syndrome in Male Cats; A Statistical Analysis of the Problems, with Clinical Observations. J. Sm. An. Prac., 8, (1967): 207-214.

25. Smith, J. E.: Urine Reactions in Urolithiasis of Cats. North Am. Vet., 32, (1951): 628.

26. Jackson, O. F.: Oxalate Calculi and Oxaluria in the Cat. Vet. Rec. 83, (1968): 417-419.

27. Vermeulen, C. W., Miller, G. H., and Sawyer, J. B.: Some Non-Surgical Aspects of Urolithiasis. Medical Clinics of North America, 39, (1955): 281-295.

28. Beamer, R. J.: Ureterocholostomy for Relief of Urinary Stenosis in the Domestic Cat. J.A.V.M.A., 134, (1959): 201-204.

29. McCully, R. N.: Antepubic Urethostomy for the Relief of Recurrent Urethral Obstruction in the Male Cat. J.A.V.M.A., 126, (1955): 173-179.

30. Skidmore, I. F. and Whitehouse, M. W.: Biochemical Properties of Anti-inflammatory Drugs - X: Inhibition of Serotin Formation in-vitro and Inhibition of Esterase Activity of a Chymotrypsin. Biochem. Pharmacol., 16, (May, 1967): 737-751.

APPENDIX

"HISTORY: This animal was submitted for necropsy with a clinical diagnosis of cystitis, urethral stasis, urethral obstruction, and uremia.

GROSS PATHOLOGY: There were approximately 8 ulcers in the stomach varying in size from 5 mm to 2 cm in diameter. The bladder was extremely distended with hemorrhagic urine. There was a fibrinous exudate covering the entire mucosal surface of the bladder. There was no apparent necrosis of the urethra and no obstruction could be found. There was fatty degeneration of the kidneys.

HISTOPATHOLOGIC RESULTS: H & E section of the penile urethra revealed desquamation of the mucosa. There was some hemorrhage and edema and marked congestion of the submucosa. There was no cellular response. Sections of the pelvic urethra also revealed desquamation of the mucosa. There was no apparent histopath in the submucosa. Examination of H & E sections of the bladder revealed desquamation of the mucosal lining. There was a marked degree of hemorrhage in the submucosa and also some hemorrhage under the serosal surface. In some areas there were erythrocytes separating the muscle fibers. In all the sections of the bladder, there was no cellular inflammatory response.

Examination of sections of the kidneys revealed fatty degeneration. There was proteinaceous material in the tubules. A few crystals could be found in the tubules. Examining these crystals with polarized light revealed some of them to resemble oxalate crystals but others that did not resemble oxalates. There were a few microcalculi in the distal tubules. There did not appear to be any nephrosis resulting from these crystals or the microcalculi. There was one focus of interstitial fibrosis extending from the capsule to the medulla of that kidney. There was congestion in the medulla of both kidneys.

CONCLUSIONS AND COMMENTS: It was impossible to force urine through the urethra during the necropsy. Upon opening the bladder, though, there was no apparent obstruction such as sand or mucous plugs. It is possible that sheets of fibrin from the bladder wall was occluding the urethra.

DIAGNOSIS: Uremia due to the feline urological syndrome."

Pathologist,

Delbert G. Miles, D.V.M.

ACKNOWLEDGMENTS

The author wishes to thank the members of his Graduate Committee, Dr. J. E. Mosier, Dr. Embert Coles, and Dr. John Noordsy. Dr. Mosier's advice and direction was invaluable in the initiation of this project. Members of the Pathology Department gave much time and made significant contributions in areas of clinical, gross, and microscopic pathology. The author wishes to thank Dr. George Osbaldiston for his generous help in the beginning of this project.

CLINICAL STUDIES OF THE FELINE UROLOGICAL SYNDROME

by

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D.V.M., Colorado State University, 1945

AN ABSTRACT OF A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Surgery and Medicine

KANSAS STATE UNIVERSITY
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1970

A syndrome involving the urinary tract of cats has been referred to as cystitis, urolithiasis, or urethral obstruction. Since the disease may involve any part of the urinary system and may or may not involve obstruction or uroliths, it is herein referred to as the feline urological syndrome (FUS). The syndrome is clinically important because of its incidence, uncertain etiology, and the diverse therapeutic regimens.

Three hundred cats affected with FUS were observed. The anamnesis was recorded, clinical signs were described, and a therapeutic regimen prescribed in each case.

Urine pH studies were conducted on 10 normal cats. The cats were then used in a study of the effects of 4 urinary acidifying drugs. Seven cats regularly demonstrating crystalluria were used in a study of the efficacy of ethylenediamine dihydrochloride and tetrasodium ethyldiamine tetracelic acid in the reduction of crystalluria.

The efficacy of 4 urinary antispasmodics was studied in 18 cats. Forty-seven cats were used in an attempt to transmit FUS from the naturally occurring cases presented for diagnosis and treatment. The transmission attempts were routinely unsuccessful.

One hundred thirteen of 300 affected cats had urinary stasis. The remaining 187 patients demonstrated signs of dysuria and/or hematuria. Emergency therapy consisting of either

mechanical back-flushing or the use of urinary antispasmodics was instituted in those cats presented with urinary stasis. A variety of methods and techniques were evaluated.

Results of urinalysis and hematologic examinations were recorded. Hematuria was present in 93%, proteinuria in 98% and bacteriuria in 20% of the cats. Struvite crystals were noted in 83% of the urine specimens from the 300 cats. Significant abnormalities did not appear in the hematological results. Bacterial pathogens were cultured in 21% of 300 specimens submitted. Urine pH ranged from 9.5 to 5.0 with a mean of 6.5.

Thirty-two cats died as a result of FUS. Four of these died following a surgical procedure designed to relieve urinary tract obstruction.

Therapy of the urinary stasis crisis was most effective when mechanical back-flushing through the penile urethra was performed immediately. However, frequent recurrence of the obstructive crisis due to urethral stenosis suggested that mechanical manipulation in the obstructed penile urethra would tend to result in a narrowed lumen and predisposes to further obstruction. Fifty-one male cats with urinary stasis were treated with an antispasmodic. Meperidine hydrochloride given parenterally effectively relieved 10 of 18 obstructed cats. Cats which were unable to urinate following injection of an antispasmodic were relieved by cystocentesis.

Trials involving normal cats indicated that urinary acidifying drugs had little effect on urine pH values. Ethylenediamine dihydrochloride when administered to cats with consistent crystalluria reduced the gross amount of crystals by 13%. EDTA given orally at the rate of 80 mg three times daily eliminated crystalluria but produced an undesirable enteritis.

Conclusions based on this study include that the mechanical technique of urethral back-flushing should not be routinely practiced. Altering the urinary pH is of questionable value. Bacterial infection was not a direct cause of the syndrome. The combined analgesic and antispasmodic action of meperidine is an effective adjunct to therapy. The use of sodium chloride as follow-up therapy may reduce the incidence of the syndrome.