

**EXPERIMENTAL EVALUATION OF URINARY BLADDER
MARSUPIALIZATION IN MALE GOATS**

by

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Urinary bladder marsupialization has been successful in producing acceptable long-term resolution of clinical cases of obstructive urolithiasis in male goats. The purpose of this study was to evaluate the six-month outcome of urinary bladder marsupialization in male goats.

The urinary bladders of six male goats free from systemic disease were marsupialized following induced urethral obstruction. Renal ultrasonography, complete blood count, and blood chemistry analysis were evaluated preoperatively (day 0), at 7 postoperative days, and at 30-day intervals until 180 postoperative days. Stomal diameter was recorded at each interval. Necropsy examination was performed on day 180 or when stomal stricture or death occurred.

Stomal stricture occurred in one goat at 120 days, and another goat was found dead at 150 days. Necropsy of this goat revealed severe, suppurative cystitis. All goats developed mild urine scald dermatitis. All blood chemistry values remained within normal limits. Significant decreases in white blood cell count, serum creatinine, and stomal diameter were observed from day 0 to day 180. Except for the goat that died at

150 days, all urinary bladders were tubular in shape and the mucosa and serosa of all urinary tract organs appeared grossly normal at necropsy examination.

Histologic evidence of chronic suppurative cystitis and chronic, mild, lymphoplasmacytic pyelitis was present in all goats. Culture of renal tissue yielded bacterial growth in three of six goats, and culture of a swab of the urinary bladder mucosa yielded bacterial growth in all animals.

Although clinical signs of ascending urinary tract infection were not observed in goats with patent stomata, urinary bladder marsupialization may result in ascending inflammation or infection. Based upon the results of this study, urinary bladder marsupialization should be recommended with caution as the primary procedure in clinical cases.

This work is dedicated to my parents, Jon and Anne May, and my brother and sister-in-law, Erik and Christie May, for their unending support, patience, guidance, and enthusiasm as I followed my dreams.

It is also dedicated to my beloved horse, “CQ”, and all of the animals that have enriched my life and career.

Last but not least, this work is also dedicated to “Perry”, “Rusty”, “Billy”, “Billy Joe”, “Norman”, “Bebe”, “Banjo”, and all of the other goats whose value to their families continually encourages us to improve methods for the treatment of obstructive urolithiasis.

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INTRODUCTION

Historically, sheep and goats have been kept as livestock for milk, meat, or hair production purposes. Urolithiasis and urinary obstruction have been significant factors in morbidity and mortality rates, especially in intensively reared flocks and herds. Due to the emphasis on financial return for production investment, the majority of surgical and medical techniques developed for the treatment of urinary outflow obstruction have been geared toward metabolic stabilization until slaughter can be performed, and some financial return is possible.

However, there is an increasing number of sheep and goats kept as pets in the United States. These animals are often kept solely for companionship instead of production, and financial return is not often a factor in determining the course of therapy. Therefore, the temporary palliation obtained by previously developed techniques is not acceptable to owners of pet sheep and goats. For these reasons, there is a driving force for the development of techniques that result in long-term resolution of urinary outflow obstruction.

ANATOMY OF THE URINARY SYSTEM OF MALE SMALL RUMINANTS

The urinary system is comprised of the paired kidneys and ureters, the urinary bladder, and the urethra. In males, the urinary system is intimately associated with the reproductive system, and the distal urethra serves as a conduit for urine and reproductive fluids (Schummer et al, 1979). Functions of the urinary system include elimination of waste products and foreign substances from the blood and maintenance of water and electrolyte hemostasis (Schummer et al, 1979; Wenzel 1999). In addition, through the production of erythropoietin, the kidneys contribute to the maintenance of circulating levels of red blood cells and therefore play a role in maintenance of the oxygen-carrying capacity of the blood (Wenzel, 1999).

The production of urine begins in the kidneys, where waste products and fluid are filtered in the glomerulus. Further passage of the filtrate through the nephron, the functional unit of the kidney, allows additional modification of the fluid through absorption and resorption of electrolytes and water. Unlike the lobular kidneys of cattle, the kidneys of the small ruminants are smooth externally and have a common renal papilla and pelvis. The kidneys of the small ruminant are bean-shaped, and more closely resemble those of the dog. The renal hilus is located medially, and is the site of entry of the renal arteries, veins, nerves, and lymphatics, as well as the ureter (Schummer et al, 1979).

The kidneys are located retroperitoneally, and in the small ruminant are encapsulated by thick, perirenal fat, the capsula adiposa (Schummer et al, 1979). In the normal goat, the kidneys are approximately 2-2.5 times the length of the second lumbar

vertebra, resulting in a range of 5.5 to 7cm (Cegarra and Lewis, 1977; Schummer et al, 1979). The right kidney lies ventral to the last thoracic vertebra and the first two to three lumbar vertebrae, and is in contact with the liver, pancreas, and small intestine (Schummer et al, 1979). The left kidney is located ventral to the fourth and fifth lumbar vertebrae, caudal to the right kidney. Due to the presence of the rumen, the left kidney is displaced to the right of midline, in contact with the spiral colon, dorsal sac of the rumen, and ascending duodenum (Cegarra and Lewis, 1977; Schummer et al, 1979). The left kidney is also more pendulous, and is almost entirely covered with peritoneum (Schummer et al, 1979; Wenzel, 1998).

Blood supply to the kidneys is provided by the renal arteries, which are large branches of the aorta (Schummer et al, 1979; Wenzel, 1999). The renal veins return blood directly into the caudal vena cava (Wenzel, 1999).

The ureters are paired, tube-like structures that collect urine from the ipsilateral renal pelvis and convey it to the urinary bladder. Like the kidneys, the ureters are located retroperitoneally. The right ureter of the ruminant follows a direct course from the hilus on the medial aspect of the right kidney to the urinary bladder. Due to the ruminal displacement of the left kidney, the left ureter begins on the medial aspect of the left kidney to the right of midline and crosses the median plane ventral to the right ureter to enter the left side of the urinary bladder (Cegarra and Lewis, 1977; Schummer et al, 1979; Singh et al, 1983; Wenzel, 1999). Both ureters enter the bladder dorsally at the neck region. Following penetration of the seromuscular layer of the bladder, the ureters tunnel for a short distance in the submucosa prior to penetrating the mucosa (Schummer et al, 1979; Wenzel, 1999). The ureters are lined with transitional cell epithelium

(Wenzel, 1999). Three muscle layers, a middle circumferential layer and inner and outer longitudinal layers, assist in urine conduction by peristalsis (Wenzel, 1999).

The urinary bladder serves as a receptacle and reservoir organ for urine. The organ is capable of great distention, accounting for its storage capacity, and can contract rapidly and forcefully to expel urine into the urethra during voiding. The bladder is located in the caudal abdomen, adjacent to the pelvic inlet. The organ is divided anatomically into the cranial apex, the body, and the caudally-located neck adjacent to the urethra (Schummer et al, 1979; Wenzel, 1999). The apex often has a small protuberance that corresponds to the remnant of the fetal urachus. The internal neck is termed the trigone region, and is a triangular area bounded by the urethral orifice and the two ureteral orifices (Waldron, 1993).

The bladder is lined with transitional cell epithelium (Schummer et al, 1979). The detrusor muscle consists of three layers of smooth muscle cell bundles, with a middle layer of transversely oriented bundles between an inner longitudinal layer and an outer longitudinal or oblique layer of muscle cells (Schummer et al, 1979; Wenzel, 1999). The external surface of the bladder exposed to the abdominal organs is covered by peritoneum, whereas the nonexposed surfaces are encased by adventitia (Schummer et al, 1979; Wenzel, 1999).

The bladder is supported by the paired lateral and single median ligaments. The lateral ligaments are bounded cranially and laterally by the round ligaments, the remnants of the umbilical arteries originating from the internal iliac arteries. The median ligament extends from the pelvic abdominal wall to the ventral aspect of the bladder, and is the

remnant of a supportive structure adjacent to the fetal urachus (Schummer et al, 1979; Wenzel, 1999).

Blood supply to the urinary bladder is provided by the cranial vesicular artery and the caudal vesicular artery. The cranial vesicular artery is a branch of the umbilical artery, and the caudal vesicular artery arises from the urogenital artery (Waldron, 1993).

The urethra forms the distal urinary pathway that conveys urine and semen from the urinary bladder and male reproductive organs out of the body. Anatomically, the male urethra can be divided into three parts: the preprostatic, pelvic, and spongy portions. The preprostatic urethra is homologous to the female urethra, and describes that part of the urethra extending from the internal urethral orifice of the neck of the bladder to the colliculus seminalis (Schummer et al, 1979; Wenzel, 1999). The colliculus seminalis is a dorsal prominence that is the site of urethral entry of the ductus deferens and the excretory duct of the vesicular gland (Sisson, 1975). The preprostatic urethra transports only urine; the pelvic and spongy portions conduct urine and semen from the body. The pelvic portion of the urethra extends from the colliculus seminalis to the pelvic outlet (Schummer et al, 1979). The distal, spongy urethra begins at the pelvic outlet, curves around the ischiatic arch, and becomes encased in the penis as it extends to the external urethral orifice (Schummer et al, 1979).

With the exception of the external urethral orifice, which is lined with stratified squamous epithelium, the urethra is lined by transitional cell epithelium (Schummer et al, 1979). The mucosa of the pelvic portion is folded slightly, surrounded by the vascular stratum cavernosum layer, and encased by the thin, smooth urethralis muscle (Schummer et al, 1979). The spongy urethral mucosa has longitudinal folds, giving the structure a

“rosette” appearance on cross-section, and is enveloped by the erectile corpus spongiosum as it enters the urethral groove on the ventral penis (Schummer et al, 1979).

The urethral recess is a structure unique to cattle, small ruminants, llamas, Dromedary camels, and swine that exists adjacent to the spongy urethra just proximal to its curve over the ischiatic arch (Tayal et al, 1984; Garrett, 1987; Timm and Watrous, 1988; Hay, 1990; Hooper and Taylor, 1995; Wenzel, 1999). This structure is an oval-shaped recess located dorsal to the urethra at its junction with the ducts of the bulbourethral glands (Hinkle et al, 1978; Garrett, 1987), and is formed by a fold of tissue located dorsal to the pelvic urethra that divides and blends gradually with the lateral walls of the spongy urethra (Garrett, 1987). The recess is estimated to be 0.5cm deep in the small ruminant, and may act as a “check valve” to prevent backflow of urine and/or semen into the pelvic urethra (Garrett, 1987). The primary clinical importance of the urethral recess is frequent lodging of catheters in the recess during urethral catheterization attempts to relieve obstruction due to urolithiasis (Hinkle et al, 1978; Garrett, 1987; Wenzel, 1999).

The urethral process (also known as the vermiform or filiform appendage) is a structure that is unique to small ruminants, and describes that portion of the urethra that extends beyond the tip of the penis for approximately 2.5cm in bucks and 4cm in rams (Schummer et al, 1979; Wenzel, 1999). The process contains erectile tissue that stiffens during erection, and may be inserted into the cervical canal of the female during breeding (Schummer et al, 1979); however, amputation of this process does not impact urination, fertility or breeding function (Tayal et al, 1984; Smith, 1986).

The lumen of the urethra narrows gradually from proximal to distal (Wenzel, 1999), but has more pronounced narrowing, the isthmus urethrae, as it curves over the ischiatic arch (Schummer et al, 1979) and at the distal bend of the sigmoid flexure just proximal to the the attachments of the retractor penis muscles (Wenzel, 1999). At the distal sigmoid flexure, there is an abrupt decrease in the cavernous space that allows urethral expansion (Hooper and Taylor, 1995). It has been reported that the urethral diameter is decreased in males castrated before puberty (Kumar et al, 1982; Wenzel, 1999), suggesting an androgen-dependent increase in diameter during development. The sites of luminal narrowing are predisposed to lodging of urethral calculi, and the reportedly smaller urethral diameter of castrates may partially account for the higher occurrence of urethral obstruction in castrated males.

PHYSIOLOGY OF THE URINARY SYSTEM AND CONTROL OF MICTURITION

The primary functions of the urinary system are the maintenance of electrolyte and water homeostasis and the elimination of metabolic waste products. These functions are performed by the kidneys, while the lower urinary tract serves in the removal of the eliminated products from the body.

The functional unit of the kidney is the nephron, which consists of the glomerulus, the tubule (which is further broken down into the proximal tubule, loop of Henle, and distal tubule), and the collecting duct (Guyton, 1991a). From the collecting duct, filtered fluid enters the renal papillae, renal pelvis, and is then transported via the ureters to the urinary bladder.

Blood enters the capillary network of the glomerulus via an afferent arteriole and fluid is filtered into Bowman's capsule, which surrounds the glomerulus, by an interaction of hydrostatic and oncotic pressures. Due to the relative impermeability of the glomerulus to proteins and the strong negative charge on the basement membrane of the glomerulus (that repels the negatively charged plasma proteins and further inhibits protein filtration into the glomerulus), the resulting filtrate resembles plasma devoid of protein (Guyton, 1991a). Modification of the filtrate proceeds in the tubules as a result of primary active transport of sodium from the lumen into the cells, co-transport of chloride, amino acids, and other organic compounds, passive absorption of chloride and urea, secretion of hydrogen, potassium, and urate ions into the tubule, and net resorption of water (Guyton, 1991a, 1991b, 1991c; Brown, 1993). Under normal circumstances,

approximately 40-60% of blood urea is reabsorbed due to its relatively small molecular size and permeability of the tubules. In contrast, virtually no creatinine is reabsorbed into the blood (Guyton, 1991c).

Autoregulation of renal blood flow occurs through a complex series of interactions and events. The principle goal of the autoregulatory mechanism is to maintain an optimum glomerular filtration rate (GFR) and rate of fluid movement through the tubules, thereby optimizing the kidney's ability to reabsorb necessary solutes while excreting unwanted waste products.

The tubuloglomerular feedback system involves two main mechanisms: the afferent arteriole vasodilator feedback mechanism and the efferent arteriole vasoconstrictor feedback mechanism. These mechanisms are controlled by the juxtaglomerular apparatus, consisting of a group of modified epithelial cells (called the macula densa) in the distal tubule that are in close apposition to the afferent and efferent arterioles (Guyton, 1991a; Brown, 1993). Although the exact mechanism of control by these specialized cells is not clear, the cell complex appears to respond to ionic concentration gradients.

Decreased GFR results in excessive reabsorption of sodium and chloride ions in the ascending limb of the loop of Henle, thereby decreasing the ionic concentration at the juxtaglomerular apparatus. In response, a signal from the macula densa to the afferent arteriole causes arteriolar dilation and results in increased blood flow into the glomerulus and a subsequent increase in GFR due to increased hydrostatic pressure (Guyton, 1991b, 1991c; Brown, 1993). This series of events is referred to as the afferent arteriole vasodilator feedback mechanism.

The efferent arteriole vasoconstrictor feedback mechanism also occurs in response to low ionic concentration at the macula densa, but is mediated by the renin-angiotensin mechanism. Release of stored renin by the juxtaglomerular cells in response to low ionic concentration in the distal tubule causes the formation of angiotensin II. The efferent arteriole is more sensitive than the afferent arteriole to the effects of angiotensin II, and constricts in response to its presence. Glomerular filtration rate is then increased by decreasing the rate of exit of blood from the glomerulus (Guyton, 1991a; Brown, 1993). The net effect of these feedback mechanisms under normal circumstances is maintenance of the GFR within a narrow, optimum range despite systemic fluxuations in vascular pressures.

Once the filtered fluid has reached the renal pelvis, no further reabsorption or secretion occurs. From the renal pelvis, urine enters the ureter. The ureters are capable of peristaltic activity that can transport urine against an obstruction with intraureteral pressures as high as 50-100 mmHg (Guyton, 1991d; Brown, 1993). Ureteral peristalsis is regulated by interactions of the autonomic nervous system; the peristaltic wave frequency and intensity are reduced by sympathetic nervous system transmission and increased by parasympathetic nervous system activity (Guyton, 1991d; Brown, 1993).

Micturition, or urination, is the process by which the bladder empties and the urethra conducts the urine outside the body. This process is characterized by a complete, reflex evacuation of the bladder by relaxation of the urethral sphincters and contraction of the detrusor muscle (Guyton, 1991d; Tammela et al, 1991; Brown, 1993; Fletcher, 1996). The detrusor muscle functions as a single unit, coordinated by neurogenic and myogenic pathways (Tammela et al, 1991).

Innervation to the urinary bladder is a classic example of the antagonistic actions of the divisions of the autonomic nervous system. Storage and voiding of urine is dependent on the interplay of the sympathetic and parasympathetic nervous systems. Somatic innervation via the pudendal nerve (S1-S3 spinal cord segments) provides voluntary control of the skeletal muscle of the external urethral sphincter. The storage of urine is mediated by sympathetic tone via the hypogastric nerve (spinal cord segments L1 or L2 through L4 or L5). Relaxation of the detrusor muscle is mediated by sympathetic α -adrenergic receptors, and β -adrenergic receptors maintain tonic contraction of the trigone and proximal urethral smooth muscle as well as inhibition of parasympathetic activity (Claridge and Shuttlesworth, 1964; Guyton, 1991d; Brown, 1993; Hosgood and Hedlund, 1993; Fletcher 1996).

Parasympathetic innervation of the bladder is transmitted via the pelvic nerves from the sacral plexus (spinal cord segments S1-S3). As bladder distention occurs and intravesicular pressure increases, mechanoreceptors in the bladder wall result in sensory fiber activation of the pelvic nerves and results in spinal cord-mediated activation of the parasympathetic pathway (also via the pelvic nerve). Direct cholinergic stimulation of the detrusor muscle causes a wave of depolarization that results in strong, coordinated detrusor contraction. Concurrently, parasympathetic inhibition of spinal cord interneurons results in pudendal nerve inhibition and subsequent reduction in the tone of the striated muscle of the urethral sphincter. Sympathetic inhibition (via the hypogastric nerve) facilitates micturition by allowing relaxation of the trigone and proximal urethra (Claridge and Shuttlesworth, 1964; Guyton, 1991d; Brown, 1993; Hosgood and Hedlund, 1993).

Once the bladder has emptied, pelvic nerve discharges are decreased, reducing stimulation to the detrusor muscle and inhibition of the hypogastric and pudendal nerves. As normal sympathetic tone is regained and parasympathetic tone diminishes, the bladder returns to a resting, storage state (Guyton, 1991d; Brown, 1993; Hosgood and Hedlund, 1993).

Voluntary control of micturition arises from micturition centers in the pons and cerebral cortex. These centers exert their action primarily on the pudendal nerve supplying the external urethral sphincter (Guyton, 1991d; Brown, 1993), and receive sensory nerve transmission via the spinal cord in response to bladder distention and pain (Brown, 1993; Hosgood and Hedlund, 1993). Voluntary brain stem control is usually inhibitory to micturition.

The internal and external urethral sphincters are functional, not anatomical, structures. The internal urethral sphincter is located at the neck of the bladder, and is formed by smooth muscle fibers that are continuous with the detrusor muscle (Brown, 1993). The external urethral sphincter, composed of striated muscle, is innervated by somatic nervous system via the pudendal nerve (Brown, 1993). Both sphincters are inhibited during the voiding phase of micturition (Brown, 1993).

URINARY SYSTEM HOST DEFENSES

Because of its communication with the environment through the external urethral orifice, the urinary tract would be consistently at risk of infection if there were no defensive mechanisms present. Although hematogenous origin infections do occur, the majority of urinary tract infections are the result of ascending migration of bacterial, viral, or fungal pathogens (Parsons et al, 1975; Hosgood and Hedlund, 1993). Host defense mechanisms include normal voiding of urine, antibacterial properties of urine, anatomic barriers to infection, and mucosal defense barriers (Tanagho, 1969a; Parsons et al, 1975; Lees and Osborne, 1979; Hosgood and Hedlund, 1993;). Compromise of one or more of these barriers to infection may result in urinary tract infection.

Normal, unimpeded micturition provides defense against ascending infection in a number of ways. Mechanical elimination of urethral bacteria by the urine stream and complete emptying of the bladder contribute to interference with bacterial colonization. Because normal urine is sterile, dilution of bacterial population also occurs in the urethra (Hosgood and Hedlund, 1993). Masih et al (1970) observed that bacterial colonization in the urethra was increased in the presence of bacterial cystitis, and a high level of colonization persisted once established.

Anatomic factors in host defense against ascending infection include urethral length, vesicoureteral junction anatomy, and ultrastructural characteristics of the urinary transitional epithelium (Lees and Osborne, 1979; Hosgood and Hedlund, 1993). The increased urethral length of the male necessitates long-distance ascent of bacterial pathogens to gain access to the proximal urinary tract. In female humans and dogs, a

mid-urethral high-pressure zone is present that inhibits the migration of bacteria (Tanagho et al, 1969b; Hosgood and Hedlund, 1993). Bacteria inoculated into the urethra below this zone resulted in ascending infection in 4 of 15 dogs, whereas inoculation near or above the high-pressure zone resulted in ascending infection in 11 of 15 dogs (Mayo and Hinman, 1973). This zone may represent an adaptation to provide compensatory protection for a shorter urethral length. The midurethral high-pressure zone is composed of striated muscle that contributes over 50% of static urethral resistance in the female (Tanagho et al, 1969b). Through the action of this muscular zone, the urethra is capable of actively changing its resistance to voiding (Tanagho et al, 1969b). The presence of this high-pressure zone has not been confirmed in other species. The mucosal cells of the distal urethra are covered with microvilli, which function in urethral peristalsis. Urethral peristalsis may play a role in defense by maintaining unidirectional urine flow (Tanagho, 1969a; Hosgood and Hedlund, 1993), thereby preventing ascent of bacteria that may be facilitated by turbulent or retrograde flow.

Although no true, anatomic valves are present at the vesicoureteral junction, the angle and course of the ureters within the bladder wall create an anti-reflux effect during distension and voiding (Bjorling and Peterson, 1990; Agace et al, 1996). When coupled with ureteral peristalsis, these physiologic valves ensure unidirectional flow of urine in the normal animal, assisting in the prevention of ascending infection.

In order for bacterial pathogens to colonize and invade the urinary tract, adherence to the epithelium is necessary. Mucosal defense mechanisms include the negative charge of the urethral epithelium, which repels the negatively-charged bacterial walls and inhibits colonization (Parsons et al, 1975; Hosgood and Hedlund, 1993). The

urinary mucosa does not possess direct antibacterial defenses, but a surface layer of glycosaminoglycans provides a mechanical barrier to bacterial adherence (Hosgood and Hedlund, 1993). Secretion of immunoglobulin A (IgA) by the urethral epithelium provides further inhibition of bacterial ascent and adherence by neutralizing and opsonizing bacteria (Parsons et al, 1975; Hosgood and Hedlund, 1993; Agace et al, 1996). Immunoglobulin levels, primarily IgG levels, are known to increase in response to infection; this production can be enhanced by immunization prior to inoculation with bacteria, resulting in more rapid recovery from infection (Uehling and Wolf, 1969; Parsons et al, 1975). Bacterial adherence may be further reduced by normal exfoliation of urethral mucosal cells (Hosgood and Hedlund, 1993). Direct killing of invading bacteria by the bladder mucosa may also occur, but is believed to play a minor and secondary role in bacterial elimination (Parsons et al, 1975).

The role of antibacterial properties of urine in host defense mechanisms has been debated (Lees and Osborne, 1979; Agace et al, 1996). Urine pH above 7.0 or below 6.0 is inhibitory for bacterial growth, and bactericidal activity is increased with increased deviation from this range. In addition, urine osmolality outside of a narrow range also appears to inhibit bacterial growth (Lees and Osborne, 1979; Agace et al, 1996). Urea has been shown to have antibacterial effects, and its high concentration in urine may provide protection from ascending infection (Lees and Osborne, 1979). Urine contains molecules that are analogous to mucosal-bound adhesion factors, and competitively inhibit bacterial attachment (Agace et al, 1996). Uromodulin, also called Tamm-Horsfall glycoprotein, is produced by luminal cells of the thick ascending loop of Henle and the distal tubule. This glycoprotein is secreted into the urine and inhibits bacterial

attachment to urinary epithelial cells (Agace et al, 1996). Uromodulin also enhances phagocytosis by neutrophils, increases complement activation, and increases arachidonic acid metabolism (Agace et al, 1996).

URINARY SYSTEM RESPONSE TO INJURY

Wound healing in the urinary system is very similar to that of the other tissues of the body, but several adaptations are unique to the system. Wound strength returns more quickly following urinary bladder trauma (Painter et al, 1971; Peacock and van Winkle, 1976; Bellah, 1989a, 1989b; Degner, 1996), allowing earlier restoration of storage capacity and resistance to distension.

Inflammatory urethritis can result from irritation by a urolith, catheter, or catheterization technique, and can result in scar formation and stricture (Webster and Sihelnik, 1985; Bellah, 1989a, 1989b). Frequently, the normal healing response results in the formation of an urethral stricture that may partially or completely obstruct the urethral lumen, predisposing the animal to urinary outflow obstruction and its sequelae (Painter et al, 1971; Peacock and van Winkle, 1976; Layton et al, 1987; Bellah, 1989a, 1989b). Stricture formation is enhanced by the presence of periurethral urine leakage and infection, which delay wound healing and increase fibrosis (Peacock and van Winkle, 1976; Layton et al, 1987; Bellah, 1989a, 1989b). The submucosal tissues contribute little to the fibrotic proliferation; instead, the corpus spongiosum and other periurethral tissues are responsible for the excessive fibrous synthesis (Peacock and van Winkle, 1976). Remodeling of periurethral fibrosis can result in luminal stenosis (Peacock and van Winkle, 1976; Bellah, 1989a, 1989b). During closure of urethral incisions, apposition of the incised edges is critical to avoid the development of excessive fibrous tissue that results from absence of urothelial continuity (Peacock and van Winkle, 1976; Webster and Sihelnik, 1985; Bellah, 1989a, 1989b; Scavelli, 1989).

The presence of urine in periurethral tissues results in cellulitis, edema, pain, delayed wound healing, and infection. Urethral fistulae may form and enlarge with time (Peacock and van Winkle, 1976; Bellah, 1989a, 1989b). Adequate hemostasis during urethral surgery is also imperative, because the presence of a hematoma provides medium for bacterial infection and creates a physical separation of the wound edges, favoring delayed wound healing and complications (Bellah, 1989a, 1989b). Singh and Blandy (1976) simulated urethral injuries in rats by 5 different techniques: 15 seconds of crushing with hemostats, hemostatic crushing with bacterial inoculation, hemostatic crushing with intraurethral injection of nitric acid, creation of a window in the urethra, or complete transection. The authors observed that even severe urethral injury did not induce periurethral reaction except when urine extravasation occurred. These observations led to the conclusion that urine extravasation is vital to the development of urethral stricture.

Urethral regeneration can occur if a longitudinal piece of intact urethra remains that connects the urethra proximal and distal to the site of injury (Peacock and van Winkle, 1976; Bellah, 1989a, 1989b). The urethral mucosa can regenerate in 7 days, but the corpus spongiosum requires three to five weeks for regeneration (Bellah, 1989a, 1989b; Degner, 1996). If the urethra is completely transected, urethral muscular contraction results in retraction of the urethral mucosa into the periurethral tissues proximal and distal to the wound, and the gap is filled by fibrous tissue (Peacock and van Winkle, 1976; Layton et al, 1987; Bellah, 1989a, 1989b). Remodeling and contraction of the fibrous scar results in urethral luminal restriction and subsequent partial or complete urethral obstruction (Peacock and van Winkle, 1976; Layton et al, 1987; Bellah, 1989a,

1989b). Similarly, failure to relieve excessive tension during urethral anastomosis has the same consequence (Bellah, 1989a, 1989b).

Bladder injuries in small ruminants are usually associated with traumatic bladder rupture (secondary to external trauma or, more commonly, secondary to urinary outflow obstruction) or iatrogenic damage. As with other tissues, the phases of wound healing of the urinary bladder include a lag phase (approximately 5 days), a fibroblastic phase (approximately 14 days), and a remodeling phase (Bellah, 1989b). However, unlike other tissues, the urinary bladder is capable of regaining 100% of its normal, pre-wound strength in 14-21 days (Bellah, 1989b; Degner, 1996). Collagenase activity during the fibroblastic phase is minimal when compared to the integument (Degner, 1996). Following injury, the detrusor muscle initially spasms around the site to form a watertight seal (Degner, 1996).

Another remarkable feature of the bladder is its ability to completely restore the entire bladder mucosa within 30 days of injury, with adjacent uninjured mucosa in the bladder, ureters, and urethra serving as cell reservoirs to resurface the exposed submucosa (Peacock and van Winkle, 1976; Bellah, 1989b; Wishnow et al, 1989; Degner, 1996). Unlike other epithelial tissues of the body, which rely on basal cell layer proliferation, all cell layers of the bladder mucosa proliferate and migrate during the healing process (Bellah, 1989b; Wishnow et al, 1989).

In addition to extensive mucosal regenerative capacity, the bladder is capable of regaining near-complete function within 3 months following resection of up to 75% of its total mass (Peacock and van Winkle, 1976; Bellah, 1989b). In contrast, permanent urinary diversion may result in loss of functional storage capacity due to contraction of

the bladder remnant (Bellah, 1989b). These findings suggest that some degree of stretching tension of the bladder wall is required to preserve or restore bladder storage capacity.

The ureters respond to injury in a similar manner to that of the urethra. As little as 25% of the ureteral diameter is required intact for complete regeneration of the ureter following injury (Bellah, 1989b). Urinary diversion enhances ureteral healing and decreases fibrotic stricture formation (Peacock and van Winkle, 1976; Bellah, 1989b). A watertight seal can be formed within 48 hours after injury (Peacock and van Winkle, 1976). The return of ureteral peristalsis is required for normal urine movement and kidney drainage, and will usually occur within 7-21 days after injury (Peacock and van Winkle, 1976; Bellah, 1989b).

Osteogenesis of retroperitoneal or abdominal tissues during wound healing of the bladder or ureter has been reported in humans and in dogs, and is believed to result from an interaction between the urothelium and urine during the healing process (Peacock and van Winkle, 1976). This has not been reported or clinically observed in small ruminants.

As with healing of any other tissues, wound healing of the urinary tract can be detrimentally affected by negative nitrogen balance (as with malnutrition), anemia, monocyte depletion, and corticosteroid administration (Degner, 1996). As stated previously, continued contact with urine will delay healing and enhance fibrosis and contracture (Degner, 1996).

It has been clinically observed and reported that the ruminant species exhibit an increased fibrotic response to injury (McSporran and Russell, 1978; Trent and Bailey, 1986; Moll et al, 1992). It has been speculated that this is due to decreased fibrinolytic

activity, primarily deficiencies in plasminogen activator (McSporran and Russell, 1978; Trent and Bailey, 1986). Trent and Bailey (1986) found that bovine peritoneal plasminogen activator activity was low or absent compared to that of the dog, but that fibrinolytic inhibitor activity was exaggerated when compared to that of the dog. Although this study was performed as an investigation into the pathogenesis of intraabdominal adhesions, it is possible that the differences observed may also affect wound healing of other organs. McSporran et al (1978) observed that sheep pleural mesothelium exhibited low fibrinolytic activity relative to that observed in rats. Pugatch et al (1970) found that levels of plasminogen activator were lower in sheep than in cattle, but that the levels of fibrinolytic inhibitors were comparable between the species. These studies suggest that ruminant species, and more specifically small ruminants, exhibit decreased ability to resolve or attenuate fibrinous and fibrous proliferative responses, predisposing them to the formation of excessive fibrous tissue in response to injury.

BASIC PRINCIPLES OF URINARY SURGERY

The basic principles of aseptic surgery should be followed regardless of the tissue or system of interest. However, urinary tract surgery also necessitates additional precision and comprehension of urinary physiology to reduce detrimental consequences.

Important preoperative considerations include attention to the patient's cardiovascular and renal status, careful selection of antibiotics and/or anti-inflammatory medications, and awareness of potential postoperative complications. Systemic hypotension and catecholamine-induced decreases in renal blood flow will affect the patient's response to anesthetic agents and other medications (Christie and Bjorling, 1993). Because renal excretion is a major route of elimination of many antibiotics, potentially nephrotoxic medications should be used with caution in animals with preexisting urinary tract disease. The benefits of the administration of antibiotics such as amikacin, cephaloridine, gentamicin, kanamycin, neomycin, polymyxin, streptomycin, and tobramycin should be weighed against the disadvantages in each case (Christie and Bjorling, 1993). The nephrotoxic effects of non-steroidal anti-inflammatory agents have been well described, and should also be considered prior to administration.

Primary repair of urethral lacerations may result in potentially damaging inflammation. Everingham et al (1973) compared histologic sections of canine urethrae incised then sutured with interrupted everting sutures, interrupted inverting sutures, or a continuous suture pattern that did not enter the urethral lumen (continuous extraluminal pattern). The continuous extraluminal suture pattern was superior to the other two

patterns, resulted in minimal periurethral inflammation or cyst formation, and produced no urethral distortion when healed.

As previously described (see “Urinary System Response to Injury” section), the use of an indwelling urinary catheter is associated with an increased risk of infection. Indwelling urethral catheterization is also associated with an increased risk of urethritis and subsequent urethral stricture. Ureteral splinting with a catheter has been recommended as a component of treatment for ureteral transection because ureteral mucosa is capable of regeneration over a splint (Christie and Bjorling, 1993). Urethral splinting following transection is recommended when primary anastomosis is performed, but is of little benefit if primary repair is not performed and there is not an intact segment of urethral mucosa bridging the defect (Christie and Bjorling, 1993). The primary benefit offered by urethral splinting in conjunction with anastomosis is that of urine diversion away from the healing anastomosis site, decreasing urine extravasation and its associated detrimental effects (Layton et al, 1987; Christie and Bjorling, 1993).

Factors that contribute to urethral stricture following urethrostomy include traumatic tissue handling, failure to appose urethral mucosa to the skin, excessive tension, and the use of large suture material (Christie and Bjorling, 1993; Degner, 1996; Hooper, 1998). Christie and Bjorling (1993) advocate gentle and minimal tissue handling and the use of delicate instruments for urinary surgery.

It is widely accepted that luminal exposure of suture material predisposes animals to urolith formation (Christie and Bjorling, 1993; Hooper, 1998; Baird, 1999). Polyglycolic acid and polyglactin 910, both synthetic multifilament materials, are commonly used for urinary tract surgery. Both suture materials induce minimal tissue

reaction, lose their strength after 28 days, and provide adequate strength for urinary tract surgery (Christie and Bjorling, 1993). Polyglactin 910 is more stable over a wider range of pH (Christie and Bjorling, 1993). Polyglycolic acid may lose its strength rapidly in the presence of bacteriuria and alkaline environments, compromising its applicability for use in alkaline urine-containing small ruminant urinary bladders; this has been the subject of contradictory reports (Christie and Bjorling, 1993; Boothe, 1998; Hooper, 1998). Although most suture materials act as nidi for formation of urinary calculi when in contact with urine, polyglycolic acid has been associated with an increased degree of calculus formation (Christie and Bjorling, 1993). However, since most surgeons advocate that suture material should not enter the lumen, this characteristic of polyglycolic acid is not likely to cause a problem (Boothe, 1998).

Polydioxanone (PDS-II , Ethicon, Inc., Union, NJ) and modified polyglycolic acid (Maxon , Sherwood Davis & Geck, Wayne, NJ) are synthetic monofilament absorbable materials that are also acceptable for use in urinary tract surgery (Christie and Bjorling, 1993; Boothe, 1998). Reported advantages to the use of monofilament materials include decreased tissue drag, increased strength compared to comparably-sized multifilament material, and adequate knot security (Christie and Bjorling, 1993; Boothe, 1998). Polydioxanone has an added benefit of reduced bacterial adhesion on its surface (Christie and Bjorling, 1993; Boothe, 1998). The primary disadvantage of this monofilament material is prolonged absorption (Christie and Bjorling, 1993).

Poliglecaprone (Monocryl , Ethicon, Inc., Union, NJ) is a synthetic monofilament suture material with high initial tensile strength and low tissue reactivity

(Boothe, 1998). This material may also be suitable for urinary surgery, but its use in this manner has not been reported.

Radasch et al (1990) investigated three techniques of cystotomy closure in dogs. Comparisons were made between interrupted single-layer appositional, interrupted double-layer appositional, and continuous double-layer inverting suture patterns (Cushing oversewn with Lembert) for cystotomy closure in normal urinary bladders. Polyglactin 910 was used for cystotomy closure, and none of the suture patterns used entered the bladder lumen. Except when bursting strength was determined immediately after surgery (time 0), all three suture patterns provided comparable wound strength. Although the two double-layer suture patterns were stronger at time 0, this was attributed to an increased amount of suture material and two layers of tissue closure. The authors also concluded that the comparable wound strength of the single-layer appositional pattern at later time intervals may have been due to a lesser degree of tissue trauma, vascular compromise, and anatomic disruption by one layer of suture material. Incisional ruptures occurred in 11 of 12 (91.6%) bladders tested to bursting pressures at time 0 regardless of technique, and the number of incisional ruptures decreased steadily from time 0 to 24 postoperative hours. Urine leakage always occurred at suture holes instead of incision lines, and incisional strength was greater than surrounding bladder wall strength by 24 hours after surgery. All nonincisional ruptures involved the formation of multiple mucosal tears and dissection of urine through the bladder wall until it extruded through the serosa, leading the authors to speculate that incision of the bladder wall or the initial phases of wound healing may weaken the entire urinary bladder wall.

ETIOLOGY OF UROLITHIASIS

Uroliths are composed of a central nidus (or nucleus) surrounded by sequential laminations of inorganic crystalloids and organic matrix. The three-dimensional physical shape of uroliths is determined by its crystalline structure, contact with other uroliths, the mobility of the stone, the luminal diameter and shape of the organ affected, and the flow characteristics of the urine (Osborne et al, 1985).

The exact process of urolith formation (calculogenesis) is unknown and unpredictable. Initiation of calculogenesis begins with nucleation, or the formation of a nidus. This phase is dependent upon the presence of urine supersaturation with crystalloids, which in turn is dependent upon renal excretion, urine pH, and the presence of crystallization inhibitors (Osborne et al, 1985; Bojrab, 1993; Wolfe, 1999). Several theories exist regarding the mechanism of this phase, and differ in the proposed critical factor. The precipitation-crystallization theory states that nucleation occurs as a direct result of supersaturation and precipitation of crystalloids and is independent of the presence of organic matrix or crystallization inhibitors. The matrix-crystallization theory suggests that organic matrix acts as a “scaffold” for nucleation. The crystallization-inhibition theory proposes that nucleation occurs as a result of impairment or absence of crystallization inhibitors (Osborne et al, 1985; Hosgood and Hedlund, 1993; Grauer, 1993; Wolfe, 1999).

Once established, the urolith may grow in size by one or more of three methods: crystal growth, epitaxial growth, and crystal aggregation (Osborne et al, 1985; Bojrab, 1993; Grauer, 1993). Crystal growth is dependent upon continued supersaturation of the

urine, and favors radial growth of existing uroliths of a single type. Epitaxial growth refers to the growth of one type of crystal on the surface of another type, and results in mixed-type uroliths. Crystal aggregation refers to the theory that crystal aggregation inhibitors are normally present in urine; these factors are believed to inhibit radial urolith growth and limit their size. If these factors are absent, deficient, or have altered or impaired function, crystalline growth occurs. The process of urolith growth can be rapid; experimentally-induced struvite uroliths have been reported to occur within two weeks in dogs, and can resolve within the same time period (Osborne et al, 1985).

The etiology of urolithiasis is multifactorial. Infection and pharmacologic factors have sometimes been implicated, but nutritional factors appear to be the most significant determinants of the disease (McIntosh, 1978). Genetic variations in water metabolism and phosphorus absorption and excretion have also been incriminated (Hay, 1990).

Urine pH affects the incidence of urolithiasis by altering the solubility of the components (McIntosh, 1978; Smith, 1994). Water intake is also important in determining the concentration of the solutes, and dehydration and water shortages have been associated with outbreaks of urolithiasis (McIntosh, 1978).

The presence of bacterial infection is considered an important factor in urolith formation in humans (Osborne et al, 1985). Bacteria may alter production or concentration of urine citrates, which may inhibit crystal aggregation. In addition, the presence of infection may favor the production of calculogenic matrix factors. Bacteria may also serve as nidi for urolith formation (Ling et al, 1998c). Infection with urea-producing bacteria in association with increased ammonium and carbonate ions and an alkaline urine environment favors the formation of struvite (magnesium ammonium

phosphate, MgNH_4PO_4), calcium apatite $[\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2]$, or carbonate-apatite $[\text{Ca}_{10}(\text{PO}_4)_6\text{CO}_3]$ uroliths (Stone, 1981; Osborne et al, 1985). Bacterial infections are reportedly common in humans and dogs with struvite urolithiasis (Ling et al, 1998c, 1998d), but is less common in cats with uroliths (Osborne et al, 1985). Non-struvite uroliths are not commonly associated with urinary tract infections (Osborne et al, 1985). Ling et al (1998b, 1998c) observed that urolithiasis was associated with bacterial infection in 65% of female dogs and 44% of male dogs, and female dogs were more likely to develop mixed infection. Struvite uroliths were more commonly associated with *Staphylococcus* cultures in females (Ling et al, 1998c, 1998d). Also observed was an increase in incidence of *Escherichia coli* and *Proteus* spp. in association with ureteroliths and nephroliths (Ling et al, 1998c). However, it should be noted that the association of bacterial infection with urolithiasis may instead be that of opportunistic infection whose persistence is favored by the presence of a foreign body (the urolith) (Ling et al, 1998c).

Nutrition plays a vital role in the etiopathogenesis of urolithiasis. Diets low in roughage content and with poorly balanced mineral levels are often incriminated (McIntosh, 1978; Kimberling and Arnold, 1983; van Weeren et al, 1987; Haven, 1992; Hooper, 1998). Siliceous calculi are more common in ruminants grazing in range conditions, and their formation is favored by high urinary alkalinity, low urinary phosphorus, and a high dietary calcium to phosphorus ratio (Stewart et al, 1991; Smith, 1994). Some grasses, such as Canadian prairie grass, contain higher amounts of silica, and surface waters may significantly contribute to silica intake as well (McIntosh, 1978). Feedlot animals fed concentrated rations are more commonly affected with phosphatic salt uroliths, such as struvite (Hawkins, 1965; Smith, 1994; Hooper and Taylor, 1995;

van Metre et al, 1996b; Hooper, 1998). Urinary excretion of phosphorus, which favors struvite formation, is increased by diets containing a low calcium to phosphorus ratio (McIntosh, 1978; Hooper and Taylor, 1995; Hooper, 1998). Oxalate uroliths are reported to occur in the western United States, and can result from vitamin B₆ deficiency or ingestion of *Halogeton* plants (McIntosh, 1978; Smith, 1994).

Pet goats are frequently fed grain in excess of their metabolic demands (Smith, 1994). Although it is consistently recommended to cease grain supplementation to affected animals, owner compliance is usually poor. Higher concentrate-to-roughage ratios favor the incidence of urolithiasis (Hawkins, 1965; McIntosh, 1978).

Urine output and concentration are directly related to water intake. Decreased water intake (and consequent increased urinary concentration) due to dietary or environmental conditions is an important factor in the development of urolithiasis (Hay, 1990; Smith, 1994; Wolfe, 1999). Water intake is also reported to decrease dramatically during stressful situations, such as weaning (Hay, 1990).

Schmidt (1941) speculated that Vitamin A deficiency resulted in the desquamation of bladder epithelial cells, which served as nidi for siliceous urolith formation. However, field studies were not able to confirm that animals fed vitamin A-deficient diets had a higher incidence of urolithiasis compared to animals fed adequate diets (Hawkins, 1965; McIntosh, 1978).

Calcium carbonate urolith formation is favored by low phosphorus, high calcium-to-magnesium ratio, and alkaline urine (McIntosh, 1978). These uroliths are more commonly formed in animals grazing lush pastures rich in oxalate but low in phosphorus (McIntosh, 1978). Calcium carbonate uroliths are the most common type of urolith in

horses (Osborne et al, 1989; Howard et al, 1998; Sertich et al, 1998). Ingestion of subterranean clover, high in calcium, has been associated with the formation of calcium carbonate uroliths in small ruminants (Kimberling and Arnold, 1983; Smith, 1994).

Cystine uroliths are more common in small animals than in small ruminants or in humans, and are associated with high dietary protein levels (McIntosh, 1978; Osborne et al, 1989). Uric acid urates and xanthine uroliths are much less common, and have been observed in humans and in Dalmation dogs (McIntosh, 1978; Osborne et al, 1989). Outbreaks in sheep have been reported, and may have resulted from molybdenum deficiency (McIntosh, 1978).

Suture material in contact with urine in the urinary bladder may favor the formation of cystic calculi (Stone, 1981; Hooper, 1998). Stone (1981) stated that calculi may be induced in alkaline urine by the luminal exposure of synthetic nonabsorbable suture. In addition, the same author stated that polyglycolic acid and chromic gut will similarly favor crystallization despite their absorbable nature.

Diethylstilbestrol administration appears to increase the incidence of urolithiasis (Hawkins, 1965; Hooper and Taylor, 1995). This may be due to hypertrophy of the seminal vesicles, ampullae, urethrae, and bulbourethral glands (Hawkins, 1965) or due to increased mucoprotein production (McIntosh, 1978; Hooper and Taylor, 1995; Wolfe, 1999). The administration of the diuretic acetazolamide, which alters urinary calcium excretion, has been associated with an increased risk of urolithiasis (McIntosh, 1978).

Castrated male goats are reportedly predisposed to obstructive urolithiasis (van Metre et al, 1996a, 1996b; Hooper, 1998). However, the overrepresentation of castrated male goats may be confounding because male goats are frequently castrated at an early

age to limit odor and libido to increase their desirability as pets. Male goats are able to produce mature spermatozoa at 3.5 months of age, and develop libido earlier (Smith, 1986). Bailey (1984) stated that urolithiasis is less common in the goat than in rams and wethers. However, due to the increasing trend in the number of goats kept as pets (personal communication, National Pygmy Goat Association, 1996), the majority of cases referred for treatment of urolithiasis are pets or valuable breeding animals, and goats predominate the hospitalized population of small ruminants at the Veterinary Teaching Hospital at the Virginia-Maryland Regional College of Veterinary Medicine. Calculi are reported to occur in equal incidence in females and males; males appear predisposed to obstruction due to increased urethral length, decreased urethral diameter, and slower urine flow (Aldridge and Garry, 1992; Wolfe, 1999). It is generally accepted that the female urethra is larger in diameter and more distensible, facilitating passage of uroliths; therefore, males are much more likely to develop clinical signs of urinary tract obstruction (Wolfe, 1999).

The urethrae of castrated male ruminants have been reported to be significantly smaller than those of partially castrated or uncastrated males (Marsh and Safford, 1957; Bailey, 1975; Kumar et al, 1982; Kimberling and Arnold, 1983; Smith, 1994; Hooper and Taylor, 1995). Marsh and Safford (1957) performed latex casts of the urethra in bulls, steers castrated at 7 months of age, and steers castrated at less than 1 month of age. The minimum mean diameter of the urethra (at the distal sigmoid flexure) were 5.08mm, 4.0mm, and 3.53mm for the bulls, late castrates, and early castrates, respectively. Based on measurement of the diameter of transected urethrae in slaughtered animals, Bailey (1975) calculated that bulls, partially castrated males, and males castrated at 6 months of

age were able to eliminate calculi that were 100%, 50%, and 14% heavier than those that could be eliminated by males castrated at 2 months of age, respectively. Kumar et al (1982) observed decreased urethral length and diameter in castrated and partially castrated goats compared to intact male goats. Frank et al (1961) did not observe significant differences in the urethral luminal diameters of steers affected or not affected with cystic calculi; however, none of the animals in the study experienced urethral obstruction.

In dogs, the most common site of obstruction by uroliths is in the urethra and immediately proximal to the os penis (Stone, 1981; Smith, 1993). Female dogs do not commonly develop urethral obstruction, due to shorter urethral length and larger urethral lumen size (Ling et al, 1998b). Urinary calculi are more commonly located in the urinary bladder than the upper urinary tract (Ling et al, 1998b; Franti et al, 1999). Sex, age, breed, and geographical predispositions to urolith type and occurrence have been observed in dogs (Ling et al, 1998a, 1998b, 1998c, 1998d; Franti et al, 1999).

In cats, potential risk factors associated with struvite urolith formation include excess dietary magnesium, phosphorus, and/or protein, urinary tract infection with urease-producing organisms, alkaline urine, decreased urine volume, and hypokalemia (Bartges et al, 1996). These factors influence struvite formation by increasing the excretion or supersaturation of precursors such as ammonium, magnesium, or phosphate ions. The most common site of urethral obstruction in male cats is at the junction of the pelvic and penile urethra, where an abrupt decrease in lumen size occurs (Scavelli, 1989).

The development of uroliths in the kidneys of ruminants has been reported, and Bailey (1975) observed more than ten times as many siliceous calculi in the kidneys

than in the bladders of slaughtered male cattle. However, none of the animals with renal calculi were reported to have clinically evident renal compromise, and the only fatalities during the study were animals that developed urethral obstruction. It was concluded that the site of calculus formation plays an important role in the manifestation of clinical disease.

The urethral process is considered the most common site of urinary tract obstruction by uroliths in small ruminants, and amputation of the urethral process does not appear to impair breeding function (Smith, 1986). Cattle are more commonly affected by obstruction by a single calculus lodged in the distal sigmoid flexure (Oehme and Tillmann, 1965; Wolfe, 1999). In contrast, sheep and goats are more commonly affected by multiple calculi (Oehme and Tillman, 1965; Haven et al, 1992; van Metre et al, 1996a; Wolfe, 1999). Sutherland (1958) reported up to 200 uroliths in sheep affected with urinary calculi. The distal sigmoid flexure is a common site of obstruction because of the sharp bend associated with the flexure and the abrupt decrease in the size of the cavernous space that permits urethral expansion (Hooper and Taylor, 1995; Hooper, 1998; Wolfe, 1999). Twenty of 21 cases of urolithiasis in small ruminants were affected by multiple calculi (Haven et al, 1992).

CLINICAL MANIFESTATIONS OF UROLITHIASIS AND URINARY OBSTRUCTION

In the early phases of urinary obstruction, animals exhibit restlessness, tail flagging, and straining to urinate (Oehme and Tillmann, 1965; Kimberling and Arnold, 1983; Smith, 1994; Hooper and Taylor, 1995; van Metre et al, 1996a; Baird, 1999; Wolfe, 1999). Blood-stained urine may be observed, and crystals may be present on the preputial hairs (Oehme and Tillmann, 1965; Kimberling and Arnold, 1983; Smith, 1994; Baird, 1999; Wolfe, 1999). Elevation in temperature, heart rate, and respiratory rate may reflect pain and/or inflammation (Hooper and Taylor, 1995; Hooper, 1998). As the disease progresses and bladder distention increases, signs of abdominal pain are observed (Oehme and Tillmann, 1965; van Metre et al, 1996a). Excessive vocalization is commonly observed in goats (van Metre et al, 1996a). As the animal strains, rectal prolapse may occur (Oehme and Tillmann, 1965; Smith, 1994). A prominent urethral pulsation may be palpable when a finger is inserted rectally (Oehme and Tillmann, 1965; Smith, 1994; Wolfe, 1999). Affected animals may become anorectic, and ruminal motility is often reduced (Prasad et al, 1978).

As with any systemic condition in animals, a complete physical examination can provide numerous clues for diagnosis. Because intact male goats will often urinate on the long hairs under their mandibles to attract females, examination of this area can reveal the presence of grit and suggest that crystalluria is present (Wenzel, 1999). Animals with ruptured urethrae may present with a warm, painful swelling of the ventral abdomen and prepuce and preputial prolapse; as the subcutaneous urine produces tissue necrosis, the

affected tissues become cold and often become infected (Oehme and Tillmann, 1965; Hooper and Taylor, 1995; Hooper, 1998; Wolfe, 1999). If bladder rupture has occurred, the signs of abdominal pain will often abate until peritonitis and abdominal distention occur (Oehme and Tillmann, 1965; Smith, 1994; Wolfe, 1999). Ballotment of the abdomen of an animal with a ruptured bladder may reveal fluid waves (Prasad et al, 1978; Baird, 1999; Wolfe, 1999). Heating of aspirated abdominal or subcutaneous fluid may produce a urine odor (Hooper, 1998).

Abdominocentesis can provide confirmation of suspected uroperitoneum. Abdominal fluid creatinine concentration is at least twice that of the serum in animals with uroperitoneum (van Metre and Smith, 1991; Hooper, 1998; Baird, 1999). Marked elevation of abdominal fluid urea or potassium levels has also supported the diagnosis of uroperitoneum in a small number of cases (van Metre and Smith, 1991).

Frequently, bacterial cystitis accompanies urinary obstruction. Incomplete urine voiding and traumatic disruption of the epithelial lining of the bladder are important risk factors in the development of ascending urinary tract infection (Lulich et al, 1996). A self-perpetuating cycle can occur between urolithiasis and urease-producing bacterial urinary tract infection: infection with these organisms favors the deposition of magnesium ammonium phosphate (MgNH_4PO_3 , or struvite) crystals in the urine, which may then result in obstructive urolithiasis and resultant urine retention and predisposition to infection (Lulich et al, 1996).

Urinary outflow obstruction results in the inability to eliminate metabolic waste products. Postrenal azotemia occurs, and renal azotemia may develop if the obstruction is not relieved and hydronephrosis and renal compromise occur. Postrenal azotemia will

resolve upon resolution of the obstruction. In a retrospective study of 19 male goats with urolithiasis, postrenal azotemia was present in 10 of 11 goats tested (May et al, 1998). Blood urea nitrogen (BUN) levels up to 373 mg/dl and creatinine concentrations of up to 16.7 mg/dl were observed (May et al, 1998). Within one week following resolution of urinary tract obstruction, creatinine and BUN levels were normal in 4 of 5 goats tested and were markedly reduced in the fifth goat.

Ruminant species are capable of eliminating urea and phosphorus through the saliva and rumen, and are therefore better able to tolerate azotemia (Houpt, 1959; Brobst et al, 1978; Sockett, 1984; Hooper, 1998). Ruminal recycling of urea allows the species to reuse the nitrogen source when protein intake is reduced, and allows for removal of excess blood urea in the presence of urinary obstruction and impairment of the ability to eliminate waste products (Sockett, 1984; Hooper, 1998). Urea, formed in the liver during protein breakdown, is then transported to the rumen via the blood, and is utilized by rumen microbes for protein synthesis. The formed protein is digested and absorbed by more distal segments of the gastrointestinal tract, completing the regeneration cycle (Houpt, 1959; Hooper, 1998). The rumen is capable of reabsorbing up to 99% of circulating urea (Brobst et al, 1978). Due to ruminal recycling, substantial increases in blood urea nitrogen (BUN) and creatinine levels may not become evident for several days (Hooper, 1998). Anorexia or alterations of ruminal microflora results in impairment of ruminal urea cycling (Sockett, 1984; Hooper, 1998).

Because the peritoneum is a semi-permeable membrane, fluids and electrolytes may readily diffuse across the membrane to equilibrate their concentrations (Sockett, 1984; Donecker and Bellamy, 1982; Hooper, 1998). If urinary tract rupture occurs, the

presence of hyperosmolar urine in the abdomen causes influx of water into the abdomen from the vessels and tissues, resulting in dehydration (Donecker and Bellamy, 1982; Sockett, 1984; Hooper, 1998). Similarly, since urine is high in potassium and low in chloride and sodium, these ions diffuse in response to their respective concentration gradients; this results in hyperkalemia, hypochloremia, and hyponatremia (Donecker and Bellamy, 1982; Sockett, 1984; Hooper, 1998). However, because ruminants are able to secrete large amounts of potassium in the saliva, hyperkalemia is less likely to develop, and normokalemia or hypokalemia occurs (Ward, 1966; Sockett, 1984). The development of hyponatremia results in aldosterone release from the adrenal cortex, which then results in increased loss of potassium in the saliva and decreased potassium absorption by the small intestine (Sockett, 1984; Hooper, 1998). Anorexia may contribute to hypokalemia by decreasing the intake of potassium (Sockett, 1984). Urea and creatinine also move across the peritoneum according to their concentration gradients; however, due to its molecular size, creatinine movement is more restricted (Sullivan et al, 1972; Burrows and Bovee, 1974; Donecker and Bellamy, 1982; Sockett, 1984; Hooper, 1998). Hypocalcemia may occur, possibly due to decreased absorption, phosphorus-mediated inhibition of calcium absorption, or as a result of an unidentified uremic factor (Sockett, 1984). In a retrospective review of 21 azotemic cattle, Brobst et al (1978) observed hypocalcemia in 16 cases.

Hyperphosphatemia results from decreased glomerular filtration rate in nonruminant species (Sockett, 1984). Renal excretion of phosphorus in ruminants is believed to play a minimal role in phosphorus elimination, and the hyperphosphatemia that results from urinary obstruction may originate from the breakdown of high-energy

organic phosphate compounds during dehydration-induced tissue hypoxemia (Brobst et al, 1978; Sockett, 1984). Donecker and Bellamy (1982) reported that urinary bladder rupture should be suspected when serum sodium is below 135 mmol/L, serum chloride is below 85 mmol/L, and the phosphorus to BUN ratio is below 0.1:1.

Metabolic alkalosis commonly develops in azotemic ruminants secondary to abomasal atony (Sockett, 1984). The sequestration of hydrochloric acid in the abomasum results in loss of chloride ions into the abomasum and increased levels of bicarbonate ions in the bloodstream (Sockett, 1984). Metabolic alkalosis was observed in most azotemic cattle that did not have diarrhea (Brobst et al, 1978). As a result of metabolic alkalosis, intracellular shifting of potassium occurs and hypokalemia is observed (Brobst et al, 1978). The development of metabolic alkalosis differs from the metabolic acidosis that may develop in dogs (Brobst et al, 1978).

Complete blood count (CBC) abnormalities commonly observed in ruminants with urinary tract obstruction include stress neutrophilia and increased plasma fibrinogen levels (Sockett, 1984). Inflammatory leukograms may be present in chronic cases of urinary obstruction, urethral rupture, or urinary bladder rupture, and will reflect the severity of infection and/or the inflammatory response.

Abdominal ultrasonography is a noninvasive technique that may be used to evaluate the integrity of the bladder by direct visualization as well as detection of free fluid in the abdomen (Nyland et al, 1995). The presence of free fluid in the abdomen coupled with other historical or clinical signs of urolithiasis is suggestive of uroperitoneum. Prasad et al (1978) observed that bladder rupture occurred on the ventral aspect of the bladder in 15 of 20 bullocks; the remaining cases ruptured on the dorsal

aspect of the bladder. Ultrasonographic assessment of both kidneys for hydroureter, hydronephrosis, or renoliths may be beneficial in the determination of therapeutic choice and prognosis (Morin and Badertshcer, 1990; Nyland et al, 1995).

Abdominal survey radiographs are of limited benefit in the evaluation of urinary problems in ruminants due to poor visualization of the urinary system (van Weeren et al, 1987B). Excretory urographic studies may provide important information regarding the presence of hydronephrosis and/or hydroureter, but do not often allow complete visualization of the urinary bladder or urethra (Palmer et al, 1998). Several reports describe difficulty in obtaining adequate visualization of the ureters by this technique (Cegarra and Lewis, 1977; Singh et al, 1983). Singh et al (1983) reported that pneumoperitoneum in conjunction with excretory urography (2-3 ml/kg sodium iothalamate administered intravenously) allowed adequate visualization of the upper urinary tract. Pneumoperitoneum aids in visualization by causing ventral displacement of the rumen and abdominal viscera, creating a negative contrast background, and decreasing the necessary exposure (Singh et al, 1983). The use of oxygen or room air for the creation of pneumoperitoneum did not produce any adverse effects (Singh et al, 1983). Positive contrast retrograde urethrography may allow visualization of urethral calculi, sites of urethral rupture, and many conditions of the urinary bladder (Ticer et al, 1980). However, urethral catheterization of the small ruminant species is difficult due to the presence of the urethral recess (Hinkle et al, 1978; Garrett, 1987; Wenzel, 1999), and contrast imaging of the pelvic urethra may be difficult. In addition, catheterization attempts may induce additional urethral damage or rupture (Palmer et al, 1998).

Positive-contrast cystography facilitates assessment of the urinary tract in small animals. If rupture of the urinary bladder or intra-abdominal portion of the ureters has occurred, contrast medium is observed in the abdominal cavity (Waldron, 1993). In small ruminants, the previously mentioned difficulties with catheterization make cystographic studies less applicable.

Abdominal exploration may reveal uroperitoneum without gross evidence of urinary bladder rupture. Radasch et al (1990) observed that nonincisional rupture of urinary bladders occurred by mucosal tearing followed by dissection of urine through the bladder wall until it extruded through the serosa. Similar findings were observed during abdominal exploration of several clinical cases at the Virginia-Maryland Regional College of Veterinary Medicine Veterinary Teaching Hospital, in which uroperitoneum was discovered despite the presence of an intact urinary bladder. The urinary bladders in these cases were grossly distended and discolored, and would “sweat” urine through the serosa.

PREVENTION OF UROLITHIASIS

Urolithiasis is a multifactorial disease; therefore, preventive measures are centered on changes in nutrition, management, and environment. Recurrence of urolithiasis is common in all species studied (Osborne et al, 1985; van Metre et al, 1996b). An understanding of the etiologic factors is critical to success in prevention of initial occurrence as well as recurrence of urolithiasis.

Encouraging adequate water intake may prevent urolithiasis by diluting the concentration of solutes in the urine (Sockett, 1984; Hay, 1990; Smith, 1994; van Metre et al, 1996b). This can be accomplished by ensuring unlimited access to potable water (water may need to be warmed during cold weather and placed in the shade during hot weather) and making salt available. Addition of salt to the ration also produces elevation of chloride levels in the urine and favors the production of magnesium chloride, which is more soluble than magnesium ammonium phosphate (van Metre et al, 1996b; Hooper, 1998a). The palatability of feed is reduced if the concentration of salt is greater than 4-5% (Kimberling and Arnold, 1983; Sockett, 1984; Hay, 1990; Hooper and Taylor, 1995); therefore, salt addition should be practiced in moderation.

Roughage-based diets require more mastication, thereby increasing saliva production. Because saliva is an important route of phosphorus excretion in ruminant species, ad libitum roughage rations result in increased salivary and fecal loss of phosphorus and decrease urinary excretion of phosphorus (Hay, 1990). This results in lower urinary concentrations of phosphorus, decreasing the risk of phosphatic calculi. In addition, roughage diets result in increased water intake, increased urinary output, and

lower production of urinary mucoproteins compared to concentrate diets (Hay, 1990). The decrease in water intake associated with intermittent feeding of concentrate rations results in activation of the renin-angiotensin system. This results in decreased urine production, which in turn results in increased urinary solute concentration and increased risk of precipitation of these solutes into urolith form (Hay, 1990). Ad libitum roughage-based diets do not result in activation of this system, thereby preventing the abrupt changes in urinary output and concentration that are considered risk factors for urolithiasis (Hay, 1990). It is obviously important that the roughage diet be palatable. One detrimental effect of roughage-based diets is the production of alkaline urine (pH 7.8 to 8.5), which is reported to favor the precipitation of salts such as magnesium ammonium phosphate (Hay, 1990). Concentrate diets result in acidic urine (pH 5.2 to 7.0) (Hay, 1990).

Addition of calcium carbonate, calcium chloride, or calcium sulfate to the diet to ensure a calcium:phosphorus ratio of greater than 2:1 has been reported to reduce the absorption of excess dietary phosphorus (Kimberling and Arnold, 1983; Hay, 1990; Smith, 1994; Hooper and Taylor, 1995; Hooper, 1998). Phosphorus levels should not exceed 0.6% in the diet (Hay, 1990).

Stressful situations, such as weaning or management changes, can produce dramatic reduction in water and feed intake (Hay, 1990). Avoidance of sudden changes in management or diet may help prevent abrupt changes in water intake. Gradual weaning and bucket feeding during artificial rearing may reduce stress and help reduce the risk of urolithiasis (Hay, 1990).

Addition of ammonium chloride to the diet of small ruminants to acidify the urine has been the subject of contradictory reports (Bailey, 1976; Stewart et al, 1990; Stewart et al 1991; Smith, 1994; Hooper and Taylor, 1995; van Metre et al, 1996b; Hooper, 1998). Dosages of 40-300 mg/kg/day, or to 0.5-2% of the ration, have been reported (Hooper, 1998). Bailey (1984) recommended the addition of 2% ammonium chloride to the ration in flocks where urolithiasis was prevalent. Higher doses of ammonium chloride reduce the palatability of the ration, and Hooper (1998) recommended a maximum dose of 100 mg/kg/day to avoid toxicity. Stewart et al (1991) documented that the addition of approximately 12 grams of ammonium chloride per lamb per day resulted in increased urine volume, urinary silica excretion, urinary calcium excretion, and a significant decrease in urolith formation when compared to control animals. Although urinary calcium excretion was increased, the increase in urine volume resulted in dilution of the calcium and lower calcium and silica concentrations in the urine (Stewart et al, 1991). Hooper (1998) recommended making a paste of ammonium chloride (30-40 mg/kg/day) and syrup and administering the paste with an oral syringe for the prevention of urolithiasis in small ruminants. Excessive consumption or administration of ammonium chloride may result in inappetance and diarrhea (Hooper, 1998).

Inactive pet goats are commonly fed grain rations in excess of their metabolic needs (Smith, 1994). Therefore, it is usually recommended to cease grain supplementation in these animals; however, owner compliance is generally disappointing.

Because urethral size has been reported to be a critical factor in the development of urolithiasis, many authors advocate delayed castration or rearing of intact lambs and kids (Marsh and Safford, 1957; Bailey, 1975; Hay, 1990; Smith, 1994). This practice

may be acceptable in production facilities, but early appearance of libido, sperm production, and odor in male small ruminants (especially goats) often necessitates early castration of animals intended as pets.

TREATMENT AND PROGNOSIS OF UROLITHIASIS AND URINARY OBSTRUCTION

Treatment goals for urolithiasis and urinary obstruction include the relief of outflow obstruction, correction and stabilization of metabolic and systemic abnormalities, elimination of existing uroliths, control and resolution of urinary tract infection, and prevention of recurrence (Osborne et al, 1985). Mortality due to urolithiasis and urinary obstruction can be due to inability to relieve the obstruction, recurrence of obstruction, loss of renal function, inability to correct severe metabolic abnormalities, and euthanasia due to expected loss of reproductive function in breeding animals (Craig et al, 1987).

Urinary tract obstruction due to urolithiasis is the most common reason for urinary surgery in small ruminants and swine (Palmer et al, 1998). Because urethral obstruction is more common in males than in females, the majority of the surgical treatments described below address the procedure in males. Factors that determine the choice of procedure include the severity and extent of tissue trauma or necrosis, the animal's value, surgeon's experience, economic constraints, available facilities, and the owner's expectations for the animal's future use (van Metre, 1996a, 1996b; Wolfe, 1999). The prognosis for life for animals with ruptured urethrae is reportedly better than that for animals with ruptured bladders, and reflects the degree of electrolyte and water imbalance resulting from uroperitoneum (Donecker and Bellamy, 1982). However, the prognosis for breeding ability is poor following urethral surgery or urethral rupture (Hay, 1990; Noordsy, 1994; van Metre et al, 1996a, 1996b; Hooper, 1998).

Supportive care

Initial assessment and early institution of therapy for azotemia, electrolyte abnormalities, and acid-base derangements are vital to the success of treatment of urinary obstruction due to urolithiasis. Stabilization of the patient is crucial to survival during surgery and recovery from anesthesia. Therapy for correction of metabolic abnormalities should be instituted, if not completed, prior to surgery (Burrows and Bovee, 1974; Bellah, 1989a; Hooper and Taylor, 1995; Hooper, 1998). Postoperative continuation of intravenous fluid therapy and medications should be based upon the animal's clinical condition and cardiovascular and renal status (van Metre et al, 1996a, 1996b; Hooper, 1998; May et al, 1998).

Most azotemic small ruminants are also hyponatremic, hypochloremic, hypokalemic, hyperphosphatemic, dehydrated, and have metabolic alkalosis (Sockett, 1984; Hooper and Taylor, 1995; Hooper, 1998). Therefore, intravenous administration of normal saline is indicated to rehydrate and diurese the animal. Supplementation with potassium chloride should be performed only when clinicopathologic confirmation of hypokalemia is present (Hooper and Taylor, 1995; Hooper, 1998).

If urinary tract rupture and uroperitoneum or subcutaneous urine leakage has occurred, establishment of urine drainage is vital. Removal of urine from the abdomen prevents further solute and water losses, reduces urine-induced chemical peritonitis, and allows stabilization of the patient prior to surgery (Burrows and Bovee, 1974; Sockett, 1984; Hooper and Taylor, 1995; Wolfe, 1999). It has been recommended that urine or other abdominal effusions be removed slowly to prevent circulatory shock and venous pooling that may result from rapid fluid removal (Sockett, 1984; Hooper and Taylor,

1995; Baird, 1999; Wolfe, 1999). Prasad et al (1978) observed no complications associated with rapid removal of urine from the peritoneum of standing bullocks with ruptured bladders. Maintenance of an intraabdominal drain may be difficult in ruminants due to occlusion of the drain by the omentum or fibrin (Wolfe, 1999). Placement of a one-way valve, fashioned from a finger of a latex glove, has been recommended to decrease air aspiration into the abdomen and bladder and decrease associated irritation and distention (Wolfe, 1999).

Cystocentesis has been advocated as a potentially life-saving technique in small animals (Smith, 1993). Due to the presence of the rumen, percutaneous cystocentesis is difficult in standing large ruminants. Small ruminants may be placed in dorsal or lateral recumbency for cystocentesis via an ultrasound-guided ventral percutaneous approach; this is relatively simple in animals with distended bladders. Gera and Nigam (1979) performed cystocentesis per rectum, but did not report on the results or complications of the technique. Despite cleansing of the rectum, perforation of the bladder through the rectal wall would likely be associated with an increased risk of cystitis and perirectal abscessation.

Parenteral administration of antibiotics and anti-inflammatories is indicated in the therapy for urinary tract rupture and abdominal or subcutaneous urine leakage (Burrows and Bovee, 1974; Sockett, 1984). Choice of antibiotics and anti-inflammatory agents administered should be based upon efficacy, activity against likely pathogens, convenience of administration, and the renal-sparing properties of the medication (Lees and Rogers, 1986).

The presence of sequelae or concurrent illness should also be evaluated, and may have a marked influence on the prognosis. Sharma et al (1983) reported on the complications and outcome of urolithiasis and urinary retention in 36 bullocks; a poor prognosis was associated with recumbency, ruminal tympany, severe anemia, rectal prolapse, subcutaneous urine leakage, and atonic bladder. Mortality was proportionately related to the chronicity and severity of bladder serosal adhesions to the body wall, omentum, intestines, or rumen.

Medical therapy

The administration of smooth muscle relaxants and anxiolytics may aid the passage of obstructing uroliths by relieving local spasm (Hay, 1990). An early report advocated the administration of smooth muscle antispasmodics and tranquilizers, with a reported success rate of 73% (Oehme and Tillmann, 1965). Diazepam may be used intravenously at a dose of 0.1-0.5 mg/kg, or acepromazine may be administered intravenously at a dose of 0.1 mg/kg (Smith, 1994). However, more recent contradictory manuscripts (vanMetre and Smith, 1991) indicate little to no success with medical therapy. The infusion of lidocaine into the urethra may relieve spasm and pain, facilitating the passage of urethral calculi (Hooper, 1998).

Urethral catheterization may relieve the urethral obstruction. However, retrograde passage of a catheter into the bladder is very difficult in ruminants due to the previously described urethral recess (see “Anatomy of the Urinary System of the Male Small Ruminant”), and catheterization attempts may induce traumatic urethritis that facilitates bacterial ascent and colonization. Poor technique during catheterization

attempts may also induce urethral rupture (Hooper, 1998). The use of intravenous xylazine (0.025 mg/kg) has been reported to facilitate exteriorization of the penis for catheterization, but induces diuresis and must be used with caution in an animal with urinary obstruction (Gasthuys et al, 1993; Hooper, 1998).

Antibiotic therapy is indicated when urinary tract infection is present. However, control of urinary tract infection is not likely if uroliths are present; elimination of the primary disease process is vital to success (Lees and Rogers, 1986; Bellah, 1989a). Elimination of infectious cystitis is a vital component of treatment of struvite urolithiasis (Grauer, 1993). Administration of antibiotics while an indwelling urinary catheter is in place may decrease the incidence of clinical urinary tract infection (Lees et al, 1981). However, antibiotic administration does not prevent infection in all animals, and appears to select for resistant organisms (Lees et al, 1981).

Nutritional management plays an important role in the management of urolithiasis, either alone or in conjunction with surgical therapy. Acidification of the urine increases solubility of struvite and enhances its dissolution; a change of only 0.6 units of pH can increase the solubility of struvite by 75% (Osborne et al, 1985). Frank et al (1961) reported a significantly lower urine pH in steers without urinary calculi (mean, 7.63) when compared to steers with cystic calculi (mean, 7.74). In horses, urinary acidification can be achieved by the administration of ammonium chloride, methionine, or ascorbic acid and water. Superior results were obtained with one-time administration of 1 kg ascorbic acid per horse via nasogastric tube, which produced urinary acidification 4 to 6 hours after administration (Wood et al, 1990). Disadvantages to medical dissolution of uroliths include the possibility of prolonged treatment and the requirement

for a high degree of owner compliance during therapy (Grauer, 1993). Medical therapy is not indicated for dissolution of urethroliths, because urethral calculi are not continuously exposed to urine (Osborne et al, 1993).

Urine acidification can also be achieved through direct cystic infusion of a 3% acetic acid/sterile saline solution. This solution was used for repeated bladder lavage of a stallion with cystic calculi (Sertich et al, 1998). Although clinical improvement and preservation of breeding function were obtained, small urinary calculi remained present despite treatment, and required periodic lavage for removal.

Urethral process amputation

Because many consider the urethral process to be the most common site of obstruction in small ruminants, amputation of the urethral process has been advocated for the treatment of urethral obstruction in ruminants. However, urethral process amputation is only successful when the offending calculus is lodged within the urethral process. Amputation of the urethral process does not adversely affect breeding function (Tayal et al, 1984; Smith, 1986; van Metre et al, 1996a). Although this procedure may immediately restore urinary outflow, reobstruction is common (vanMetre and Smith, 1991; Hooper, 1998; Moll and May, 1999). Urethral process amputation was performed in 22 goats, resulting in resolution of obstruction in 14 (63.4%); reobstruction occurred in 6 goats (42.9%) within three days and within one year in 4 (50%) of the remaining 8 goats (van Metre and Smith, 1991). Reobstruction is most likely the result of the presence of additional calculi (van Metre and Smith, 1991). One report indicated that in 16 cases (62.5%) of urethral obstruction 10 were not relieved by urethral process

amputation. Outflow obstruction was relieved in 4 of 16 animals (25%), but obstruction recurred in all 4 within 36 hours (Haven et al, 1992).

Exteriorization of the penis may be difficult in small ruminants, but can be facilitated by sedation or lumbosacral epidural anesthesia (van Metre, 1996a). Administration of acepromazine (0.05-0.1 mg/kg IM or IV) or diazepam (0.1 mg/kg IV) will produce sedation to facilitate the procedure. Lumbosacral epidural administration of 0.5-1 ml/kg of 2% lidocaine, the total dose not exceeding 15 ml, can be used.

Urethral surgery

Urethrotomy techniques differ primarily in the portion of the urethra that is incised. These procedures can often be performed in the conscious animal with epidural and/or local anesthesia. The ischial urethrotomy procedure requires placement of the incision at the level of the tuber ischii. Perineal urethrotomy entails a skin incision and deep dissection immediately below the tuber ischii. The urethra, located ventrally in the penis, is incised. Scrotal urethrotomy involves castration of the animal and incision into the urethra at the level of the scrotum. Alternatively, a urethrotomy incision can be centered over an obstructive urolith (Hooper and Taylor, 1995). Crushing of uroliths by extraluminal compression with towel forceps and urethral lavage following urethral exposure has been reported (Hooper and Taylor, 1995; Wolfe, 1999); although this avoids a urethral incision, avoidance of potentially severe urethral damage is almost impossible, and this technique cannot be recommended in pets or breeding stock.

Ischial urethrotomy requires incision into the bulbospongiosus muscle, resulting in increased hemorrhage (Hooper and Taylor, 1995). This procedure may be performed to

allow placement of an indwelling cystic or urethral catheter; however, passage of the catheter is usually difficult due to the presence of the urethral recess (Hooper and Taylor, 1995).

Oehme and Tillmann (1965) reported a 95% recovery rate following urethrotomy in cattle. The success rate was considered lower in small ruminants due to the presence of multiple calculi. However, the authors recommended that the animal not be kept for longer than 3-4 months due to increased likelihood of complications. Van Weeren et al (1987a) reported on the results of perineal urethrostomy in 18 small ruminants. Although all 18 animals survived to be discharged from the hospital, only 3 of the 18 animals (16.7%) of the animals had acceptable long-term results (≥ 1 year after surgery). Recurrence of obstruction was common, and death due to urolithiasis or slaughter due to recurrence of obstruction resulted in a 27.8% mortality rate within one year after surgery. Several animals required more than one procedure to obtain acceptable long-term results. Van Metre and Smith (1991) reported a 58.3% rate of recurrence following urethrostomy, with a mean of 1.1 months until recurrence. The results of Haven et al (1992) were similarly disappointing. Ten of 11 animals (90.9%) that were discharged after perineal urethrostomy experienced short-term complications (hemorrhage, dehiscence, or subcutaneous urine leakage), and urethral stricture occurred in 7 of 9 animals (77.7%), and long-term survival was 55%. A median time from surgery to reobstruction of 104 days was observed. At the time of follow-up, only 5 of 9 animals (55.5%) were still alive; 2 of these 5 animals (40%) had undergone more than one urethrostomy, and 4 of the 5 (80%) survivors had undergone concurrent cystotomy (Haven et al, 1992).

Urethrostomy techniques involve suturing of the urethral mucosa to the skin. The surgical approaches are identical to urethrotomy approaches in the same region, but careful apposition of the urethral mucosa and skin is important for the creation of a stoma. Prepubic urethrostomy involves the relocation of the pelvic urethra to the ventral abdominal wall cranial to the pubis.

Prepubic urethrostomy is an accepted technique of urinary tract diversion in small animals (Bradley, 1989; Bjorling and Peterson, 1990). Urinary continence can be maintained if the urethral sphincter is intact (Bjorling and Peterson, 1990). Important surgical principles include avoidance of an overly acute angle between the bladder and urethra and avoidance of excessive tension on the anastomotic site (Scavelli, 1989; Bjorling and Peterson, 1990). Prepubic urethrostomy was performed in one male goat and one wether lamb that had experienced recurrent obstruction after one or more urethrostomies (Stone et al, 1997). The technique required pubic and ischiatic osteotomy in the wether lamb, which resulted in incisional complications that necessitated mesh repair of an incisional hernia. The lamb exhibited recurrent urinary tract infections after surgery, and died of renal failure three years after surgery. Osteotomy was not required in the goat. Stomal stricture and partial urinary outflow obstruction occurred two months after surgery, and euthanasia was performed. Although both animals maintained urinary continence, the outcome of the procedure in these two animals was unacceptable. Further investigation of this technique is necessary before its use in additional clinical cases can be recommended.

Leon et al (1997) reported success with prepubic urethrostomy in two Vietnamese pot-bellied pigs with urethral abnormalities. The pelvic urethra was more accessible in

the pigs than in small ruminants, and pelvic osteotomy was not necessary. Follow-up information was obtained at 6 weeks in one pig and 18 months in the second pig. No complications were reported.

Extrapelvic anastomosis of the neck of the bladder to the penile urethra has been performed in male dogs, with less favorable results than prepubic urethrostomy (Bjorling and Peterson, 1990). Potential complications include stricture of the anastomotic site and loss of urethral sphincter continence. Extrapelvic urethral or urethropheputial anastomosis was performed in two Vietnamese pot-bellied pigs (Mann et al, 1994) with acceptable outcome at follow-up (13 and 12 months, respectively). Following extrapelvic anastomosis of the pelvic urethra and penile urethra, urinary continence was preserved and no complications were reported. Extrapelvic urethropheputial anastomosis resulted in urine pooling in the prepuce, hematuria, and nocturia that necessitated antibiotic therapy; these conditions resolved with thorough cleansing of the prepuce and preputial diverticulum nine months after surgery.

With the exception of prepubic urethrostomy, advantages of urethral surgical procedures include low cost, preservation of urinary continence, and the avoidance of general anesthesia (van Metre and Smith, 1991; Osborne et al, 1993; Stone et al, 1997). Prepubic urethrostomy requires general anesthesia, and is therefore associated with higher cost. Disadvantages of urethral surgery include loss of breeding function, inability to remove additional calculi in the bladder, and relatively high risks for reobstruction and stricture (Smith and Schiller, 1978; Smith et al, 1981; van Weeren, 1987; Bellah, 1989a, 1989b; van Metre and Smith, 1991; Hooper, 1998). Perineal urethrostomy in cats is considered a risk factor for urinary tract infection, due to alteration of urethral defense

mechanisms (Gregory et al, 1984; Osborne et al, 1991; Griffin and Gregory, 1992; Osborne et al, 1996). The incidence of stomal stricture is reduced by careful apposition of the skin and urethral mucosa, thereby preventing suture gapping and mucosal trauma (Hooper, 1998). Urethral dilation may result in immediate resolution of the urethral stricture but is generally considered to increase the risk of stricture recurrence (Peacock and van Winkle, 1976; Webster and Sihelnik, 1985).

Although loss of breeding function is not often a concern in pets, it can be a vital factor when surgical intervention is required in male breeding stock. Other less commonly reported complications include fecal incontinence, urinary incontinence, rectourethral fistula formation, rectal prolapse, and perineal hernias (Scavelli, 1989). These complications are likely to result from traumatic dissection and poor attention to regional anatomy (Johnson et al, 1981; Gregory et al, 1984). Because of the high incidence of reobstruction following urethral surgery, the long-term prognosis for life is guarded (Winter et al, 1987; Noordsy, 1994; Hooper, 1998). Median time to recurrence of urinary obstruction following urethral surgery of 60-70 days has been reported (Haven et al, 1993; May et al, 1998). The likelihood of recurrence markedly limits the applicability of these procedures in pets.

Due to the close proximity to the anus, a perineal urethrotomy is potentially more likely to become infected (Bellah, 1989a, 1989b). Urethrotomies may be less likely to develop stricture than urethrostomies (van Metre and Smith, 1991).

Primary closure of the urethrotomy incision has been advocated to decrease postoperative hemorrhage from the corpus spongiosum and fibrosis in dogs. However, primary closure requires meticulous hemostasis, increased intraoperative time, gentle

tissue handling, and delicate suture material and instrumentation in order to decrease the risk of urethral stricture (Waldron et al, 1985; Stone, 1992; Smith, 1993). Postoperative hemorrhage from the corpus spongiosum during micturition is common following urethrostomy, and can be heavy during the first few postoperative days (Waldron et al, 1985; Bellah, 1989a, 1989b; Smith, 1993; Hooper and Taylor, 1995). In small animals, it has been reported that the bleeding may persist until the sutures are removed (Bellah, 1989a, 1989b).

Penile amputation

Penile amputation is performed in the same location as the perineal urethrotomy/ urethrostomy incision, but the penis is completely severed and the proximal portion is retroverted through the incision. The proximal stump of the penis is sutured to the skin of the perineum. The distal penis may be left intact or removed. If the dorsal artery and vein of the penis have undergone thrombosis or have been transected and/or ligated, it is recommended that the distal penis be resected (Haven et al, 1992; van Metre et al, 1996a). Penile amputation is indicated when the urethra has ruptured (Wolfe, 1999), and the severity of necrosis of the prepuce and distal penis may determine the need for resection. Hooper and Taylor (1995) advocate removal of the distal stump of the penis to allow superior exteriorization of the urethra, apposition of mucosa and skin, and elimination of the distal urethra as a source of infection. Potential complications of penile amputation include perineal urine scald, obstruction by clotted blood, recurrent obstruction by additional calculi or stricture, and trauma to the penile stump by the animal's tail (Gasthuys et al, 1993; van Metre et al, 1996a). The advantages and

disadvantages of penile amputation are similar to those for urethrotomy and urethrostomy. Gasthuys et al (1993) reported that 20 of 52 animals (38%) treated with penile amputation had successful outcomes.

Cystotomy

A cystotomy involves direct incision into the bladder through a celiotomy approach. The bladder lumen is directly visualized, and uroliths can be manually removed or lavaged from the lumen. This technique is often performed in conjunction with normograde and/or retrograde urethral flushing. If the bladder has ruptured, primary repair can be performed. Occasionally, a partial cystectomy may be necessary to repair the rupture (Hooper and Taylor, 1995).

Cystotomy remains the “gold standard” for treatment of urolithiasis in small ruminants (Moll and May, 1999). Advantages to this technique include the ability to remove cystic calculi and potentially flush the urethra, avoidance of urethral incision, reestablishment of normal anatomy, and preservation of breeding ability and urinary continence (van Metre and Smith, 1991; Hooper and Taylor, 1995; May et al, 1998).

Disadvantages of cystotomy include the necessity for general anesthesia, increased surgical time, recurrent cystitis, and increased difficulty compared to urethrostomy (van Metre and Smith, 1991; Hooper and Taylor, 1995). Repetitive and forceful flushing is often necessary to retropulse urethral calculi into the bladder or expel them in a normograde direction (van Metre and Smith, 1991). Stone et al (1997) stated that normograde flushing of the urethra may actually pack crystals more tightly in the distal urethra, resulting in severe obstruction and urethritis. In addition, the authors stated

that catheter manipulation during flushing may traumatize the urethra and result in stricture formation (Stone et al, 1997). Rakestraw et al (1995) observed urethral rupture in one of 15 small ruminants, and the authors believed that the rupture was precipitated by urethral flushing. Other studies have not observed this complication (Bailey, 1984; van Metre et al, 1991; Haven et al, 1992).

Bailey (1984) stated that cystotomy is the only technique that may result in resolution of the obstruction without resultant urethral stricture. However, this statement overlooks the possibility of urolith-induced urethral stricture. Van Metre (1991) reported reestablishment of urethral patency in 11 of 14 goats (79%) with cystotomy and urethral flushing, with a 27.3% recurrence rate a mean of 7.5 months after surgery. Haven et al (1992) reported that 7 of 8 animals that underwent cystotomy and urethral flushing were alive and had not developed complications at a median follow-up period of 23 months (range, 5 to 55 months). In 1996, van Metre et al (b) reported that 5 of 8 small ruminants (62.5%) treated with cystotomy developed recurrent urethral obstruction a mean of 15.3 months after surgery, and one animal died of acute renal failure after surgery. Although statistical analysis revealed no significant difference in the rates of reobstruction with cystotomy, tube cystostomy, or tube cystostomy with perineal urethrostomy, the time to reobstruction following cystotomy was significantly greater than that observed following the other two techniques.

Tube cystostomy

Permanent cystostomy catheters have been used in dogs with urinary outflow obstruction secondary to transitional cell carcinoma of the bladder (Smith, 1995). The

catheters allowed the owners to decompress the bladder several times daily. Median time from catheter placement to euthanasia was 106 days. The technique was advocated for use in cases where the clients did not wish to pursue aggressive surgical and chemotherapeutic treatment but were unwilling to euthanatize the animal at the time of diagnosis.

Placement of a percutaneous Foley or mushroom catheter into the bladder has been performed for therapy of obstructive urolithiasis in small ruminants. A routine cystotomy is performed, the bladder is repaired as necessary, and the catheter is secured through a small cystotomy site with a pursestring suture. The catheter exits the abdomen through a tunneled incision. This procedure is intended to allow bladder decompression while urethral inflammation resolves, the obstructing calculi are passed, urethral healing progresses, and dietary changes are implemented (Rakestraw et al, 1995). In addition, regular bladder lavage with antibacterial solutions or solutions intended to favor urolith dissolution is facilitated by the indwelling cystic catheter (Hooper and Taylor, 1995). Van Metre et al (1996b) performed daily infusions of 60-150 ml of an acetic acid-saline solution (pH 4.3 to 4.8) via the cystostomy catheter to encourage urolith dissolution. The catheter was occluded for 1 to 2 hours following infusion, and animals were fed ammonium chloride (1-2% dry matter intake per day); urethral patency was restored more quickly (within 10 days) in these animals compared to a mean of 14.4 days reported by Rakestraw et al (1995). Tube cystostomy allows decompression during healing of the cystorrhaphy in animals with ruptured bladders.

The cystostomy catheter is left in place and periodically occluded to assess urethral patency. Placement of an Elizabethan collar may be necessary to prevent the

animal from chewing on or dislodging the catheter (van Metre et al, 1996b). When urethral patency is re-established, the catheter is removed. Potential complications of tube cystostomy include failure of resolution of obstruction, obstruction or dislodging of the catheter, uroperitoneum, peritonitis, ascending infection, iatrogenic trauma to the bowel during percutaneous placement, cystitis, and incisional herniation.

Advantages to tube cystostomy include immediate relief of outflow obstruction; the ability to provide urinary decompression when a urethral catheter cannot be placed or maintained; avoidance of urethral incision or iatrogenic urethral trauma during catheterization techniques; avoidance of complications associated with indwelling urethral catheters; improved long-term outcome compared to urethral surgery; and the ability to assess urinary tract patency by occlusion of the catheter (Botte, 1983; van Metre et al, 1996b; Hooper, 1998). Rakestraw et al (1995) reported that seven animals of 12 (58.3%) were alive 1-27 months after surgery and had not experienced recurrence of obstruction. One of 8 small ruminants (12.5%) experienced recurrence of urethral obstruction at 6.5 months after surgery (van Metre et al, 1996b).

An additional benefit of tube cystostomy is the ability to use the indwelling cystostomy tube to instill contrast media in a normograde fashion to perform cystourethrography. This allows visualization of the urethra and assessment of urethral patency, thereby determining when the cystostomy catheter can be removed (Palmer et al, 1998).

This technique may also be advantageous in cases with prolonged and/or severe bladder distension. As previously described (see “Urinary System Response to Injury”), the tight junctions between smooth muscle cells of the bladder may become damaged

during distension. Maintaining urinary bladder decompression may allow restoration or reestablishment of function of these tight junctions, facilitating return to normal function and tone (Scavelli, 1989).

Disadvantages to tube cystostomy include prolonged hospitalization, increased duration of antibiotic administration, increased risk of bacteriuria, and a higher incidence of short-term postoperative complications than other procedures (Warren et al, 1981; Barsanti et al, 1985; Bjorling and Peterson, 1990; Rakestraw et al, 1995). Complications that necessitated additional surgery were observed in 3 of 15 animals, and included catheter obstruction and dislodging of the balloon. Mild incisional complications occurred in 3 animals, and incisional herniation occurred in another. Other complications observed included uroperitoneum following catheter removal in one animal, peritonitis in another, and cystitis in one animal (Rakestraw et al, 1995).

Rakestraw et al (1995) recommended that antibiotic administration be continued until 5-7 days following catheter removal; this resulted in a minimum duration of antibiotic therapy of 19 days. The presence of an indwelling urinary catheter in dogs and cats resulted in development of bacteriuria in 11 of 21 animals (52%) with prior sterile urine after a mean of 4 days (range, 1-10 days) of catheterization (Barsanti et al, 1985). As observed in human patients, the risk of infection increased with the duration of catheterization (Warren et al, 1981; Barsanti et al, 1985). In the same study, the type of bacteria present changed during the catheterization period in 6 of 10 animals with bacteriuria prior to catheterization. Although antibiotic therapy tended to decrease the overall incidence of catheter-associated infection, a trend toward antibiotic resistance was observed when antibiotics were administered during the catheterization period. The

administration of antibiotics while a cystostomy tube is in place is contraindicated in small animals because it favors the growth of resistant bacteria (Bjorling and Peterson, 1990). In small animal patients, connection to a closed urinary drainage system is recommended (Stone, 1992). This is rarely possible in small ruminants due to housing requirements and environment. Attachment of a one-way valve prevents air from entering the bladder, and may decrease bacterial ascent (Hooper, 1998).

Tube cystostomy was used in conjunction with daily dietary supplementation with 10g ammonium chloride and intravesicular infusion of 200ml of a 1:10 mixture of sodium acetate/acetic acid buffer (solution pH 4.5) solution and saline in a ram with urethral obstruction due to urolithiasis (Cockcroft, 1993). The cystostomy catheter was occluded for one hour following infusion. The ram was able to urinate normally 14 days postoperatively, and was normal at follow-up one month later (Cockcroft, 1993).

Cystorrhaphy

Urinary bladder rupture is a potential sequela of prolonged or severe distension. Although a dorsally ruptured urinary bladder may spontaneously heal if bladder distension is prevented (van Metre, 1996; van Metre et al, 1996b; Wolfe, 1999), cystorrhaphy is often performed in animals not intended for slaughter. The difficulties in catheterization and the detrimental aspects of indwelling catheters in ruminant species have been previously described.

Prasad et al (1978) performed urethrotomy and cystorrhaphy on twenty bullocks with ruptured bladders secondary to urethral obstruction. The procedures were performed in the standing animal with local anesthesia. The authors reported that

surgical access to the bladder was facilitated by a caudodorsal to cranioventral oblique incision in the dorsal region of the paralumbar fossa. Despite the improved access, the cystorrhaphy was performed blindly. Nine of the animals (45%) died between 2 and 19 days after the procedures, seven of which were attributed to uremia. The poor outcome reported was possibly related to duration of obstruction and severity of uremia on presentation. No clinicopathologic data was presented, but the clinical signs reported were consistent with moderate to severe dehydration and peritonitis.

Gera and Nigam (1979) reported a 71% survival rate following cystorrhaphy in 108 bovine males. Cystorrhaphy was performed via paralumbar, paramedian, or pararectal approach. Success rates for each technique were not reported, but the authors expressed difficulty in obtaining adequate surgical access with the pararectal approach.

Gasthuys et al (1993) reported a successful outcome in 8 of 24 (33%) animals that underwent cystorrhaphy. The poor outcome in this study was attributed to damage to the remaining urinary tract.

Urinary diversion techniques

Urinary tract diversion techniques involve the translocation of urine outflow to an abnormal location, usually the abdominal wall or the gastrointestinal tract (Bjorling and Peterson, 1990). These procedures are indicated in cases where urinary tract outflow has been destroyed or has lost its function (Bjorling and Howard, 1989; Bjorling and Peterson, 1990). This may occur due to neoplasia, iatrogenic injury, severe inflammation or infection, obstruction, traumatic injury, or rupture (Bjorling and Howard, 1989; Bjorling and Peterson, 1990).

The originally developed gastrointestinal urinary diversion techniques involved the creation of an isolated, blind-ended portion of bowel (stomach, small intestine, or large bowel). The urinary system (usually the ureters) was connected to the bowel conduit, and the bowel was then anchored to the skin to form the stoma and allow urinary elimination (Bjorling and Peterson, 1990; Waldron, 1993). Attempts to create continent diversion techniques by routing the bowel segment through the external anal sphincter or by utilizing the rectum as the conduit have been unsuccessful (Bjorling and Peterson, 1990).

Ureterosigmoidostomy (ureterocolonic anastomosis) is the standard procedure for urinary diversion in human patients (Bjorling and Peterson, 1990). This technique allows complete removal of the bladder (cystectomy) in cases of severe neoplasia or bladder compromise.

Trigonal-colonic anastomosis involves connection of the bladder trigone to the terminal colon. One reported advantage to this technique over ureterocolonic anastomosis is the preservation of the ureterovesical junction; this theoretically would decrease the incidence of ascending infection by preserving an anatomic barrier (Bjorling and Peterson, 1990).

Advantages to urinary diversion techniques include the ability for complete cystectomy and the possible preservation of urinary continence (Bjorling and Howard, 1989). The disadvantages of these techniques are numerous, and include increased risk of bacterial colonization of the urinary tract, and increased morbidity and mortality (Masih et al, 1970; Bjorling and Peterson, 1990; Maiti and Mogha, 1990; Waldron, 1993). Abnormal absorption of urinary waste products by the intestinal segment may

occur. This may result in azotemia, hyperchloremia, hypokalemia, and metabolic acidosis (Bjorling and Peterson, 1990; Maiti and Mogha, 1990; Waldron, 1993). Hyperammonemia and neurologic dysfunction has been reported in dogs following ureterocolonic anastomosis (Bjorling and Peterson, 1990). The degree of resorption of these products is related to the duration of contact with the intestinal tract, and is increased by dehydration, decreased gastrointestinal motility, and reduced volume of ingesta (Bjorling and Peterson, 1990).

URINARY BLADDER MARSUPIALIZATION

Urinary bladder marsupialization refers to the cutaneous diversion of the urinary bladder to the body wall. Other terms to describe the procedure include vesicostomy, urinary bladder fistulization, and cystostomy.

Cutaneous vesicostomy, or “tubeless cystostomy”, was first reported by Blocksom in 1957. The procedure has been advocated for use for temporary urinary diversion in children with congenital or developmental urinary tract diseases (Bruce and Gonzales, 1980). Reported advantages to its use include ease of surgical technique, avoidance of an indwelling catheter, ease of reversal, and a low complication rate (Lapides et al, 1960; Ross et al, 1965; Allen, 1980; Bruce and Gonzales, 1980; Hurwitz and Ehrlich, 1983; Krahn and Johnson, 1993). Indications for its use in children include bladder dysfunction or infravesical obstruction in conjunction with chronic infection, vesicoureteral reflux, hydronephrosis, or renal functional impairment (Bruce and Gonzales, 1980). Vesicostomy allows urinary tract decompression until the patient’s condition is stabilized, infection is cleared, and/or the patient has grown to a size that is more appropriate for reconstructive procedures (Duckett, 1974; Hurwitz and Ehrlich, 1983).

Several techniques have been described for cutaneous vesicostomy. The first described technique, or Blocksom technique, was reported in 1957. This technique remains the simplest and most commonly used vesicostomy technique in humans (Hurwitz and Ehrlich, 1983). Modifications of the Blocksom technique were made by Lapides (1960), Ross (1965), Duckett (1974), Allen (1980), Bruce and Gonzales (1980),

Krahn and Johnson (1993), and Krstic (1995) in attempt to decrease peristomal herniation and stomal stricture and preserve some degree of urinary continence. Choice of vesicostomy technique varies among authors, and is based upon personal preference and experience.

Cohen et al (1978) observed pyelographic improvement in 100% of 12 cases with presurgical hydronephrosis. Similar results were obtained by Sonda and Solomon (1980) during 20-year follow-up of cutaneous vesicostomy, when 40 of 58 renal units (69%) stabilized or improved with cutaneous vesicostomy alone. Agarwal et al (1997) reported complete resolution of vesicoureteral reflux and bladder wall changes in 14 of 23 patients (61%).

Hoffer (1962) reported a successful outcome in a canine patient that underwent transplantation of the bladder neck to the abdominal wall. The dog's urethra was necrotic and avulsed from the neck of the bladder, necessitating stoma formation. The dog retained urinary continence and no evidence of cystitis or systemic disease was observed during a four-month follow-up period.

Gasthuys et al (1993) performed fistulization of the urinary bladder in 9 male cattle with urethral obstruction. Seven of the 9 animals (77.8%) died within 14 days of surgery, and the remaining two animals were slaughtered or sold within 3 months after surgery. However, the authors recommended that the poor outcome of this study be interpreted with caution, because fistulization of the urinary bladder was performed when no other alternatives were available, and damage to the urinary tract was severe. In addition, the technique performed in cattle may be more likely to fail due to increased body size and difficulty in preventing excessive tension on the stoma. Permanent

cystostomy was performed in 2 buck goats and 2 rams, with successful outcome in 3 of 4 cases (Helminen, 1986).

The procedure of urinary bladder marsupialization in small ruminants was initially developed as a salvage technique in animals with ruptured urethras. In a retrospective study of urinary bladder marsupialization in 19 male goats presented for urolithiasis, eight goats had previously undergone one or more urethral surgeries or penile amputation (May et al, 1998). However, the procedure evolved into a primary procedure in many cases due to the improved long-term outcome when compared to other surgical procedures or when financial considerations dictated the selection of a more economical procedure. With the currently increasing trend in the number of goats and sheep kept as pets, the emphasis of treatment of urolithiasis has shifted from short-term survival until slaughter to long-term survival and prevention of recurrence. Although urethrotomy, urethrostomy, and penile amputation may produce favorable short-term results, successful long-term outcome is rare in pets and breeding stock (Hooper and Taylor, 1995; May et al, 1998). These procedures should be viewed strictly as salvage techniques, intended to resolve clinical signs of urinary tract obstruction until the animal can be slaughtered, and are unacceptable for use in pets or breeding stock (Hooper and Taylor, 1995).

In small animals, a minimum stomal diameter of 2cm has been suggested for permanent cystostomy (Bjorling and Howard, 1989; Bjorling and Peterson, 1990). Two centimeter stomal diameter is also recommended in human patients with vesicostomy (Hurwitz and Ehrlich, 1983). Increased stomal diameter is believed to increase the risk of

stomal prolapse (Hurwitz and Ehrlich, 1983). Stomal diameter is controlled by the size of the fascial opening (Hurwitz and Ehrlich, 1983).

De Badiola et al (1996) reported that insertion of a low-profile gastrostomy device (the Bard Button[®], CR Bard Inc., Murray Hill, NJ) into the bladder via the vesicostomy stoma facilitated urodynamic evaluation prior to stomal closure. The application of a stomal device is poorly tolerated by small animal patients (Bjorling and Peterson, 1990). In one clinical case at the Virginia-Maryland Regional College of Veterinary Medicine, a Bard low-profile gastrostomy device was inserted into the stoma; however, the device was removed and ingested by the goat within four hours of insertion. Further investigation is necessary before stomal devices can be recommended in clinical cases.

Advantages of the urinary bladder marsupialization procedure include simplicity of technique, minimized surgical instrumentation, decreased hospitalization and duration of medical treatment, decreased incidence of postoperative complications, and improved long-term outcome in clinical cases. Basic surgical skills are necessary for proper technique and tissue handling, but additional expertise is not required for the marsupialization procedure. Animals appear to tolerate the procedure with minimal discomfort.

Complications that may be observed following urinary bladder marsupialization include cystitis, bladder mucosal prolapse, urine scald dermatitis, and stomal stricture. Due to the cutaneous communication, the bladder is considered to be at an increased risk of bacterial colonization (Bjorling and Howard, 1989; Bjorling and Peterson, 1990; Stone, 1992). However, it is generally considered that urine retention and the presence of bacteria are required for the development of urinary tract infection (Lulich et al, 1997),

and significant urinary tract infections are believed to result from high intravesical pressures (Sonda and Solomon, 1980; Hurwitz and Ehrlich, 1983). Urinary bladder marsupialization eliminates the storage of urine and decreases intravesical pressure, decreasing the likelihood of cystitis and infection (May et al, 1998). Clinical cystitis was reported in one of 19 goats, and responded to medical therapy; however, the incidence of subclinical cystitis may have been higher (May et al, 1998). Cohen et al (1978) did not observe any clinical urinary tract infections in 12 cases of cutaneous vesicostomy in children, and no patient with preoperative sterile urine developed bacteriuria following surgery. Krahn and Johnson (1993) reported significant urinary tract infections requiring hospitalization with or without antibiotic therapy in 5 of 50 human patients with cutaneous vesicostomy. Hurwitz and Ehrlich (1983) stated that persistent or intermittent bacteriuria occurs in most human patients with vesicostomy, but that low intravesical pressures prevent the development of clinical cystitis.

Partial bladder mucosal prolapse was observed in one goat within 24 hours following the procedure, and did not recur after resection of the prolapsed mucosa (May et al, 1998). Hurwitz and Ehrlich (1983) differentiated mucosal eversion from true stomal prolapse; mucosal eversion may mimic prolapse, but does not interfere with urine drainage. Squamous metaplasia of the exposed mucosa may occur. True prolapse involves protrusion of the bladder wall through the stoma, and usually requires stomal revision. Cohen et al (1978) reported a 25% incidence of prolapse, but surgical revision was only necessary in 8% of cases.

Stomal stricture occurred in one of 19 animals three months after urinary bladder marsupialization, but the animal was able to urinate normally through its urethra (May et

al, 1998). A second goat developed stomal stricture 4 months after the procedure, and stomal revision was performed. Partial obstruction occurred 7 months later, and a second stomal revision was performed. No recurrence of obstruction had been observed in the subsequent four years. Ross et al (1965) observed stomal stricture in 7 of 36 human patients (19.4%) that underwent the Lapidus technique of cutaneous vesicostomy; stricture was attributed to insufficient stomal diameter in the majority of cases. Acute pyelonephritis developed as a consequence of stomal stricture in 2 patients (Ross et al, 1965). Stomal stricture occurred in 4 of 24 human patients (17%) that underwent cutaneous vesicostomy (Bruce and Gonzales, 1980). Only one of the 4 patients required surgical revision of the stoma; the other 3 patients were managed with infrequent, periodic dilations of the stoma. Krahn and Johnson (1993) observed stomal stricture in 4 of 50 patients and stomal prolapse in 6 of 50 patients, resulting in a stomal revision rate of 20%. Stomal stricture may be caused by excessive tension on the vesicostomy or by an undersized fascial opening (Duckett, 1974; Hurwitz and Ehrlich, 1983). Hurwitz and Ehrlich (1983) recommended calibrating the stoma to 24 French and ensuring proper mobilization of the bladder in order to decrease the likelihood of stomal stricture. The role of urine scald dermatitis in stomal stricture was addressed by Cohen et al (1978), who attributed stomal stricture to severe diaper dermatitis in 2 cases.

Reobstruction due to formation of additional uroliths was not reported in 19 cases following urinary bladder marsupialization in male goats (May et al, 1998). Despite the absence of reobstruction due to urolithiasis, the formation of additional stones cannot be ruled out; subsequent uroliths would be likely to exit the stoma instead of entering the

urethra, preventing urethral obstruction. Nephrolith formation was observed following vesicostomy in 3 of 40 patients (7.5%) by Sonda and Solomon (1980).

Although surgical reversal of urinary bladder marsupialization has not been attempted in goats, several reports describe the results in humans. The degree of success observed following primary or secondary closure of vesicostomy appears to be dependent on the original uropathy that necessitated vesicostomy (Bruce and Gonzales, 1980). Vesicostomy closure occurred in 34 of 50 human patients after a mean of 25 months, and follow-up obtained at a mean of 23 postoperative months revealed continued improvement or stabilization of the upper urinary tract in over 80 per cent of the patients. Impairment of urinary bladder function or storage capacity was not observed in any of the patients (Krahn and Johnson, 1993). Jayanthi et al (1995) performed surgical reversal of 55 vesicostomies. The majority (45/55) of the patients that underwent direct reversal without bladder augmentation became continent and had normal or clinically acceptable bladder capacities. It was concluded that normal function of the urinary bladder may be regained following reversal of vesicostomy, but that the initial pathologic process may limit the degree of return to normal function. These conclusions were supported by Khoury et al (1990) based upon cystometric studies following reversal of vesicostomy.

Postoperative concerns following urinary bladder marsupialization include urinary incontinence and, consequently, urine scald of the ventral abdomen (Bjorling and Peterson, 1990; May et al, 1998). It is largely due to these problems that cutaneous vesicostomy has been nearly abandoned in human adults, where social influences play a greater role (Cohen et al, 1978; Sheerin, 1997). Preservation of breeding ability following urinary bladder marsupialization has not been assessed; however, if the urethra

is not permanently damaged as a result of the obstruction, the marsupialization procedure should not adversely affect breeding ability. Because the currently described technique does not preserve urinary continence, marsupialized animals are not suitable house pets. The severity of urine odor and scald can be greatly reduced by maintenance of a clean stoma site. Urine scald dermatitis was reported in all 19 goats at the time of follow-up after urinary bladder marsupialization, but 15 of 17 owners indicated that they were satisfied with the outcome of the procedure (May et al, 1998). This can be achieved by keeping the area clipped and by judicious application of ointments to the peristomal skin.

Perioperative care

Presurgical stabilization of the patient, if possible, is critical to the success of the procedure. Administration of intravenous fluids for cardiovascular support and treatment of azotemia and acid-base and electrolyte disturbances should be instituted as soon as possible, and continued until resolution of the abnormalities is achieved.

Antibiotic administration (procaine penicillin G, potassium penicillin G, ceftiofur, or ampicillin) should begin preoperatively and continue postoperatively for a total of 5-7 days. Prolonged antibiotic administration is not recommended for this procedure.

Non-steroidal anti-inflammatory medications should be administered postoperatively for the management of pain and inflammation. Flunixin meglumine (0.5 to 1.1 mg/kg) has been most commonly used. Additional analgesia may be achieved by the administration of butorphanol.

AIMS OF STUDY

Although the currently available methods of treatment for obstructive urolithiasis in small ruminants provide adequate short-term relief of urinary outflow obstruction, recurrence of obstruction is common. Many of the techniques were developed to achieve short-term resolution until the animal could be slaughtered; however, the temporary results obtained are not acceptable to owners of small ruminants kept as pets or breeding animals.

The technique of urinary bladder marsupialization was developed in an attempt to improve long-term outcome in small ruminants with urinary outflow obstruction as well as animals in which the urethra had ruptured. Its use in clinical cases has resulted in acceptable long-term outcome with low morbidity (May et al, 1998). However, clinicopathologic and histopathologic assessment of clinical cases has not been performed, and the subclinical effects of urinary bladder marsupialization on the urinary tract is uncertain.

The aims of this study were as follows:

- 1) to subjectively evaluate the short- and long-term clinical effects of urinary bladder marsupialization on the urinary system of normal male goats
- 2) to determine if urinary bladder marsupialization produces histopathologic alteration of the organs of the urinary system of normal male goats
- 3) to determine if urinary bladder marsupialization results in clinicopathologic or renal ultrasonographic abnormalities in normal male goats

MATERIALS AND METHODS

Six male, crossbred, 3-6 month old goats free from systemic disease based on physical examination and historical data were studied. Initial body weight measurements were recorded. Following induction of general anesthesia with diazepam (0.5 mg/kg IV, diazepam injection, USP, Elkins-Sinn, Inc., Cherry Hill, NJ) and ketamine (10 mg/kg IV, Ketaset[®], Fort Dodge Animal Health, Fort Dodge, IA), all animals were castrated, dewormed with ivermectin (0.2 mg/kg SC, Ivomec[®], Merial Limited, Iselin, NJ), ear-tagged, and vaccinated with a clostridial vaccine (Vision[®] CDT with Spur[®], Bayer Corp., Shawnee Mission, KS). One postoperative dose of flunixin meglumine (1.1 mg/kg IV, Banamine[®], Schering-Plough Animal Health Corp., Union, NJ) was administered. Animals were allowed to recover for 10 or more days following castration before entering the marsupialization study. All animals were offered free choice grass hay and water.

Approximately 12 hours prior to urinary bladder marsupialization, each goat was anesthetized with diazepam and ketamine at the previously mentioned dosages. The urethral process was ligated with nonabsorbable suture material to simulate urinary outflow obstruction. Goats were held off of feed for 12 hours prior to surgery. Presurgical complete blood count (CBC) and chemistry profile samples were collected and submitted, and body weights were measured and recorded. Physical examination was performed and recorded. Ultrasonographic examination of the kidneys was performed. Clinical signs of urinary tract obstruction were noted if observed.

On the day of surgery, a jugular catheter was placed and perioperative intramuscular administration of ceftiofur sodium (2.2 mg/kg IM BID, Naxcel , Pharmacia & Upjohn Co., Kalamazoo, MI) and flunixin meglumine (1.1 mg/kg IM BID) was initiated. Butorphanol (0.2 mg/kg IV, Torbugesic , Fort Dodge Animal Health, Fort Dodge, IA) was administered for anesthetic premedication. Ten minutes later, a combination of diazepam (0.2 mg/kg) and ketamine (0.5 mg/kg) was administered intravenously to induce general anesthesia. An orotracheal tube was placed and connected to a nonrebreathing system delivering isoflurane (Isoflo , Abbott Laboratories, North Chicago, IL) in oxygen. The goat was placed on the operating table in dorsal recumbency and the ventral abdomen was clipped and prepared for sterile surgery.

The surgical procedures were performed using routine aseptic techniques. One primary surgeon (May) and one assistant surgeon (Moll) participated in each procedure. Surgical times were recorded, and subjective assessment of ease with which the procedure was performed was obtained. A 6 cm paramedian celiotomy was performed parallel to and 3 cm to the right of the prepuce. The sites of incision are indicated in Figure 1. The apex of the urinary bladder was identified and exteriorized from the incision (Figure 2). The urinary bladder and abdomen were evaluated for abnormalities in the bladder wall, bladder rupture, or the presence of uroperitoneum. If necessary, decompression was performed using an 18-gauge needle attached to a suction device. Two stay sutures of 2-0 polydioxanone (PDS-II , Ethicon, Inc., Union, NJ) were placed approximately 1.5 cm lateral to the apex of the bladder. A 3 cm cystotomy incision was made between the two stay sutures (Figure 3).



Figure 1. Photograph of the ventral abdomen of a goat in dorsal recumbency. This image and subsequent images that follow are oriented with the head to the top of the page. The locations of the paramedian celiotomy incisions are indicated. The dashed line represents the initial paramedian celiotomy, and the solid line represents the stomal celiotomy incision.

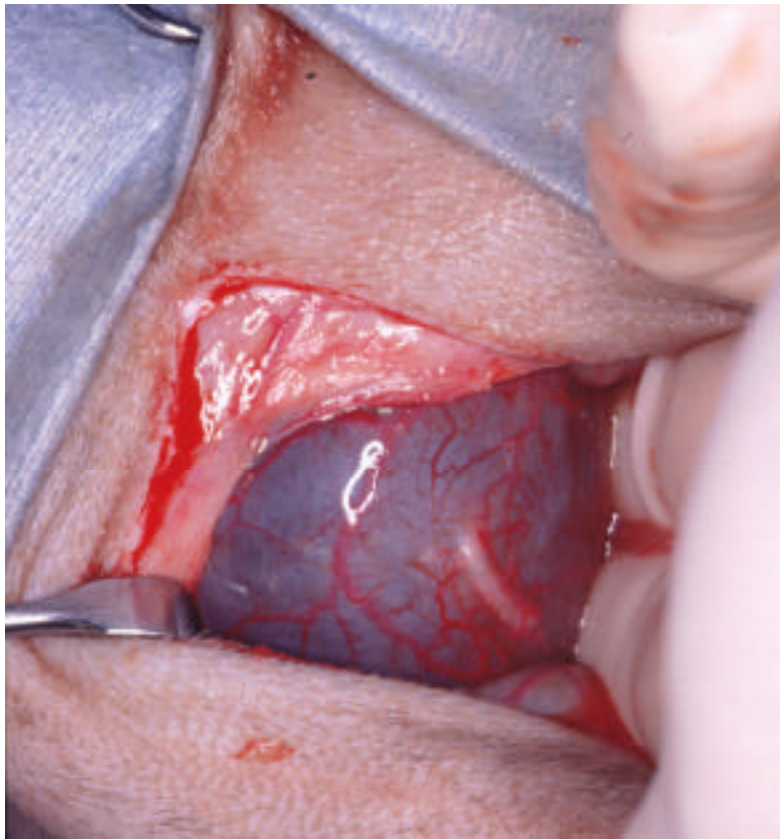


Figure 2. Visualization of the distended urinary bladder through the initial paramedian celiotomy. The remnant of the urachus can be observed as a protuberance on the urinary bladder apex. The goat's head is to the top left of the page.

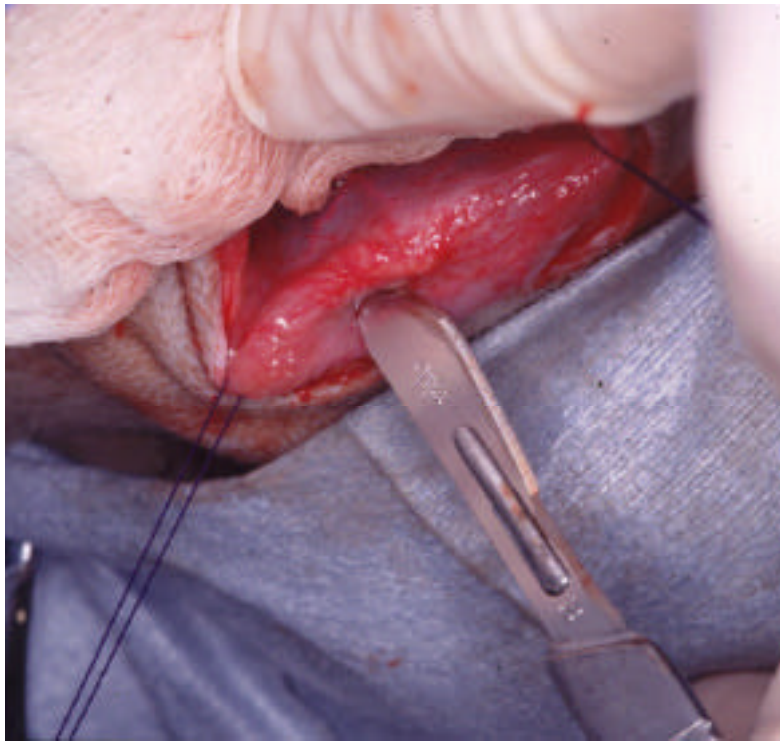


Figure 3. Photograph depicting the creation of the cystotomy between two preplaced 2-0 polydioxanone stay sutures. A saline-soaked gauze sponge has been placed in the cranial aspect of the incision to prevent urine contamination of the abdomen.

A 4 cm celiotomy was made contralateral to the first celiotomy. The location of the second celiotomy was determined by manipulation of the urinary bladder to the most cranial position that could be achieved without placing the bladder under excessive tension. A Kelly hemostatic forcep was passed through the second celiotomy, through the abdomen, and exited the initial celiotomy. The stay sutures were grasped within the jaws of the forceps (Figure 4), and gentle traction was applied until the urinary bladder was exteriorized through the second celiotomy. The seromuscular layer of the urinary bladder was circumferentially sutured to the rectus abdominis fascia with interrupted horizontal mattress sutures of 2-0 polydioxanone following placement of anchoring sutures at the most lateral, medial, cranial, and caudal aspects of the bladder (Figure 5). The margins of the cystotomy incision were sutured to the abdominal skin with simple interrupted sutures of 3-0 polydioxanone. The initial celiotomy was closed in three layers. The rectus abdominis fascia was apposed with 0 polyglycolic acid (Dexon , Sherwood Davis & Geck, Wayne, NJ) in a simple continuous pattern. The subcutaneous tissues were apposed with 2-0 polydioxanone in a simple continuous pattern, and the skin was closed with 2-0 nylon (Dermalon , Sherwood Davis & Geck, Wayne, NJ) in a continuous horizontal mattress pattern. The lengths of all incisions and the final operative stomal diameter were measured and recorded for each goat (Appendix 1). Intraoperative measurements were made with a ruled scalpel handle. Postoperative measurements were made with a metric ruler. The final appearance of the sutured stoma and celiotomy is shown in Figure 6.

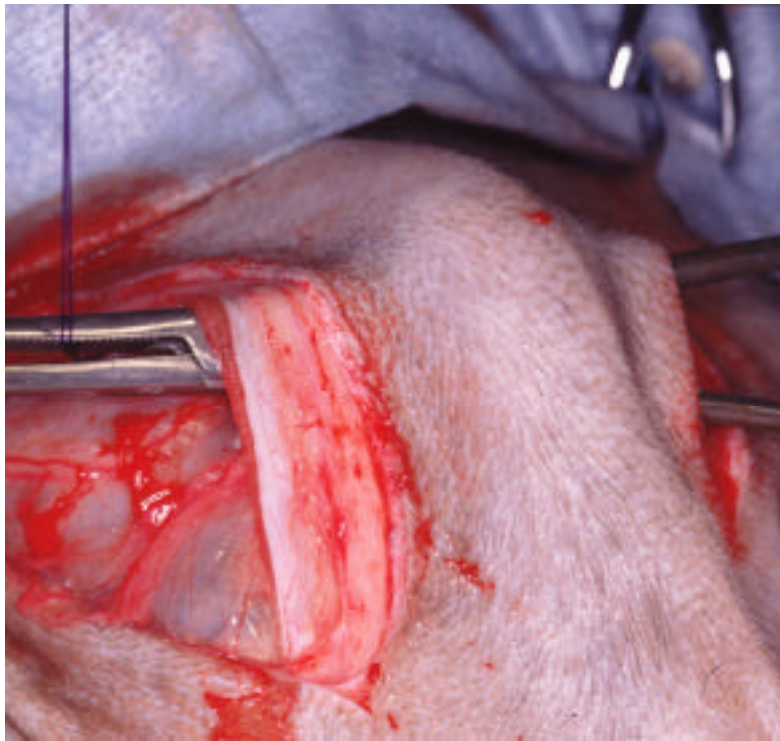


Figure 4. A Kelly forcep is passed from the initial celiotomy and used to grasp the stay sutures placed in the urinary bladder. The forceps are then withdrawn through the second incision to exteriorize the cystotomy.

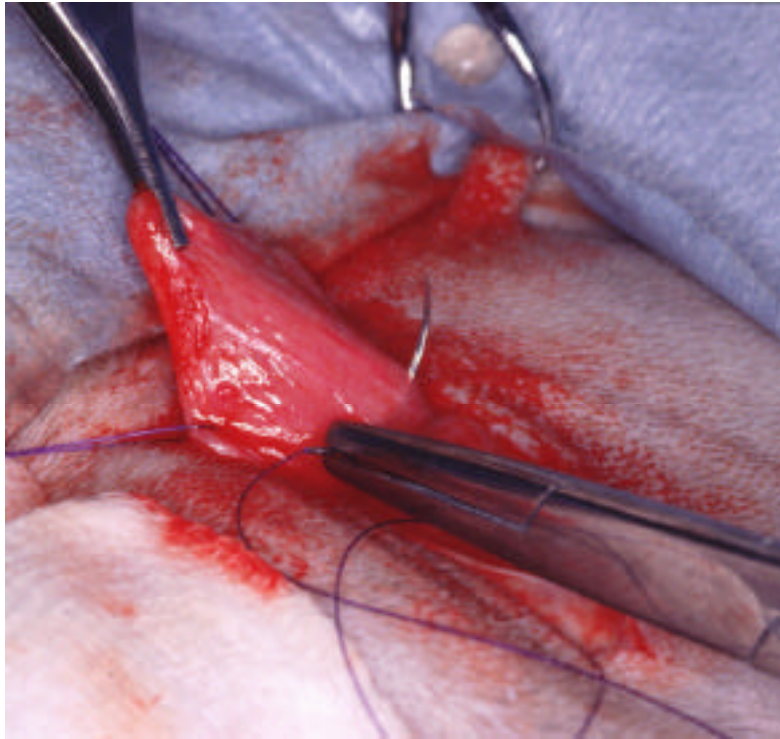


Figure 5. The seromuscular layer of the exteriorized urinary bladder is anchored to the abdominal fascia with 2-0 polydioxanone in a simple horizontal mattress pattern. A saline-soaked gauze sponge has been placed over the initial celiotomy to decrease contamination.



Figure 6. Photograph of the appearance of the sutured marsupialization stoma and initial celiotomy. The urinary bladder wall has been sutured to the skin of the ventral abdomen with 3-0 polydioxanone in a simple interrupted pattern. The initial paramedian celiotomy has been closed in three layers.

Perioperative administration of ceftiofur sodium was continued for a total of 5 days, and administration of flunixin meglumine was continued for a total of 3 days. Physical examination was performed daily for 7 postoperative days. Postoperative ultrasonographic exam of the kidneys of each goat was performed beginning at 30 postoperative days and at 30-day intervals thereafter; the ultrasonographic appearance and the measured dimensions of each kidney were recorded. CBC and chemistry profiles were obtained one week after surgery. Ten days after urinary bladder marsupialization, the goats were moved to an outside pen. Goats were fed free choice grass hay when pasture grass was not sufficient. Unlimited access to potable water was provided by a heated, automatic watering system. All goats were allowed access to a covered shed at all times for protection from weather conditions.

At weekly intervals, all marsupialization stomas were observed. If necessary, clipping of the ventral abdomen was performed. Zinc oxide (Desitin[®], Pfizer Inc., New York, NY) ointment was applied to the ventral abdomen weekly. Foot trimming was performed as necessary. All goats were dewormed with ivermectin (0.2 mg/kg, PO) approximately 3 months after surgery.

At monthly intervals, the goats were transported to the Veterinary Teaching Hospital for evaluation. Body weight was measured and recorded. Physical examination was performed, and parameters were recorded (Appendix 2). Blood samples were obtained and submitted for CBC and chemistry profile analysis. Results of CBC and chemistry profiles are reported in Appendices 3-8. Ultrasonographic examination of both kidneys was performed. Stomal diameter measurements were obtained and recorded (Appendix 9), photographs of the stomas were taken, and a subjective score for urine

scald dermatitis was assigned to each goat. The scald score was recorded as 0 = none, 1 = mild, 2 = moderate, 3 = marked, 4 = severe (Appendix 10). The ventral abdomen of each goat was clipped, and zinc oxide ointment was applied. As necessary, animals were bathed.

After six postoperative months or at complete stomal closure, animals were euthanized with pentobarbital sodium (1ml/5kg IV, Fatal-Plus , Vortech Pharmaceuticals, Dearborn, MI) following the final monthly evaluation. Necropsy examination was performed shortly after death. The celiotomy incisions were evaluated for herniation or adhesion formation. The urinary tract was photographed in situ prior to removal. All urinary tract organs were observed grossly for signs of devitalization, infection, or inflammation, including discoloration and thickening of the mucosal lining. Cultures of the urine, bladder and kidneys were obtained. The entire urinary tract and a 1 cm margin of skin surrounding the stoma were removed and preserved in 10% formalin for histopathologic examination following gross pathologic evaluation. Histopathologic examination of the peristomal skin, urinary bladder adjacent to the stoma and trigone region, proximal and distal ureters, and both kidneys was performed, and tissue sections were evaluated for inflammation, fibrosis, suppuration, and hemorrhage. The scoring system used was based on 0 = normal, 1 = mild, 2 = moderate, 3 = marked, and 4 = severe (Appendix 11).

Variables measured and recorded at each interval were temperature, pulse rate, respiratory rate, body weight, stomal diameter, change in stomal diameter from previous measurement, per cent change in stomal diameter from previous measurement, total white blood cell count, total serum protein, blood urea nitrogen, serum creatinine,

calcium, phosphorus, sodium, potassium, and chloride concentrations. Data was entered into a Microsoft Excel spreadsheet and imported into the SAS program for general linear regression modeling procedure, and polynomial trend analysis of each reported variable across time at each 30-day interval was performed. A student's t-test was performed to analyze statistical differences between mean values on Day 0 (day of surgery) and postoperative Day 7 and also between mean values on Day 0 and postoperative Day 180. Determination of significance was based upon a p-value of less than 0.05. Significant and nonsignificant trends were recorded for analysis.

RESULTS

Goats obtained ranged from approximately 3 to 6 months of age. Initial body weights ranged from 7.7 to 16.8 kg (mean, 13.3 kg; median, 14.3 kg). Body weights at completion of the study ranged from 11.4 to 19.5 kg (mean, 15 kg; median, 14.5 kg), reflecting a mean net weight gain of 1.7 kg over the study period (Appendix 12).

Ligation of the urethral process was successful for simulating short-term urinary outflow obstruction. No clinical signs of urinary tract obstruction were observed in any of the goats prior to surgery. At surgery, all goats had distended bladders. Goat 8 also had uroperitoneum due to dorsal mucosal tearing and transmural leakage of urine; no gross bladder rupture was observed.

Total surgery time ranged from 40 to 70 minutes (mean, 56 minutes; median, 57 minutes). Subjectively, the procedure was simple, and required minimal surgical instrumentation.

Stomal diameter at the conclusion of surgery ranged from 3 to 4 cm (mean, 3.25 cm; median, 3 cm). The length of the incision in the abdominal fascia during creation of the stoma ranged from 2 to 2.5 cm, with a median and mean of 2.25 cm. The distance from the stoma to the level of the preputial orifice ranged from 4.5 to 8 cm, with a mean of 6.6 cm and a median of 6.5 cm. All stomas were caudal to the preputial orifice (Appendix 1).

No differences were observed in the ultrasonographic appearance of the kidneys during preoperative examination and examination at 30, 60, 90, 120, 150, and 180 postoperative days for Goats 1, 2, 3, 6, and 8. Moderate hydroureter and hydronephrosis secondary to urinary outflow obstruction was observed in Goat 5 prior to urinary bladder

marsupialization. The severity of the hydroureter was reduced by 7 postoperative days, and was completely resolved by 30 postoperative days. No recurrence of hydroureter or hydronephrosis was observed during the study period. Although measurements of each kidney were obtained at each interval, difficulties in obtaining consistent images in the same sagittal plane prevented accurate interpretation of the measurements. As the goats increased in weight and body size, ultrasonographic imaging of both kidneys became increasingly difficult from the right paralumbar fossa.

All but 2 goats survived the 6-month study period. One goat (Goat 1) was found dead 150 days after surgery, and another (Goat 5) developed complete stomal stricture at 120 postoperative days. Goat 5 was observed to urinate through his penis at the time that stomal stricture was identified. Necropsy examinations were performed at that time. Although the specific cause of death of Goat 1 was unknown, necropsy examination revealed severe, suppurative, fibrinonecrotic cystitis; this was assumed to be secondary to stomal occlusion with hair and fecal material (Figure 7).

Subjectively, the severity of urine scald appeared to be directly related to the size of the stoma; animals with larger stomas had a larger area of scald, whereas those with smaller stomas had less urine scald. Urine scald dermatitis resolved when stomal stricture occurred, and subjectively improved in score (see “Materials and Methods”) as the stomas decreased in size. Stomal diameter decreased by a mean of 0.24 cm per month during the study (Appendix 9). Mean stomal diameter at 6 postoperative months was 0.53 cm.

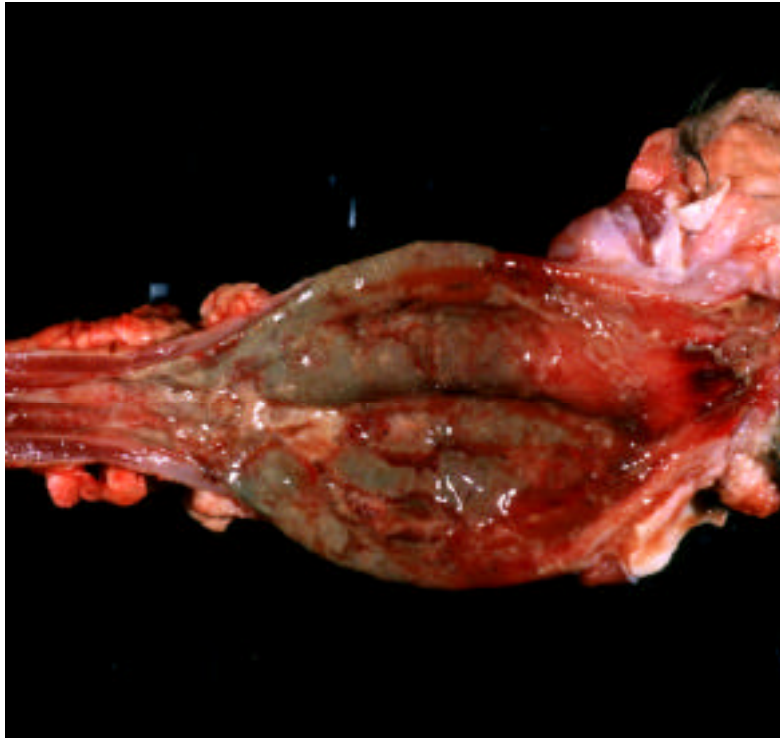


Figure 7. Urinary bladder of Goat 1 at necropsy following spontaneous death at 4 postoperative months. The photograph is oriented so that the marsupialization stoma is at the right of the image, and the pelvic urethra is at the left of the picture. Note the severe hyperemia and the presence of a fibrinonecrotic pseudomembrane.

Stomal stricture occurred in one (Goat 5) of six goats, producing a stricture rate of 16.7%. Mucosal prolapse was not observed in any of the goats. Clinical signs of cystitis or ascending urinary tract infection were not observed in Goats 2, 3, 5, 6, and 8 throughout the study period. No clinical signs of cystitis or ascending infection were observed in Goat 1 prior to spontaneous death.

Necropsy examination revealed that the bladder had become tubular in shape in all animals (Figure 8). The serosal and mucosal surfaces appeared grossly normal in all except Goat 1. Several adhesions of the omentum to the urinary bladder, internal margin of the stoma, and celiotomy were observed in all goats, but did not appear to result in obstruction or vascular compromise of the structures. The stoma was patent in all animals except Goat 5 at necropsy, and the urethrae were patent in all except Goat 6. The urethral process was absent in all animals. All urinary tract organs were grossly normal, with the exception of the shape of the bladder, in Goats 2, 3, 5, 6, and 8. A reducible, incisional hernia associated with the initial celiotomy was present in Goat 5; although small intestine was found within the hernia sac, no incarceration or compromise was present.

Histopathologic examination of the skin adjacent to the stoma revealed chronic, superficial, proliferative, perivascular dermatitis with orthokeratotic hyperkeratosis in all goats. Chronic, lymphoplasmacytic infiltration was present in the bladder mucosa of all goats, and ranged from minimal to moderate in Goats 2, 3, 5, 6, and 8 (Appendix 11). Lymphoplasmacytic infiltration was more pronounced in bladder tissue adjacent to the stoma, and became less evident with increased distance from the stoma. Histopathologic evaluation of the bladder of Goat 1 revealed severe necroulcerative and suppurative

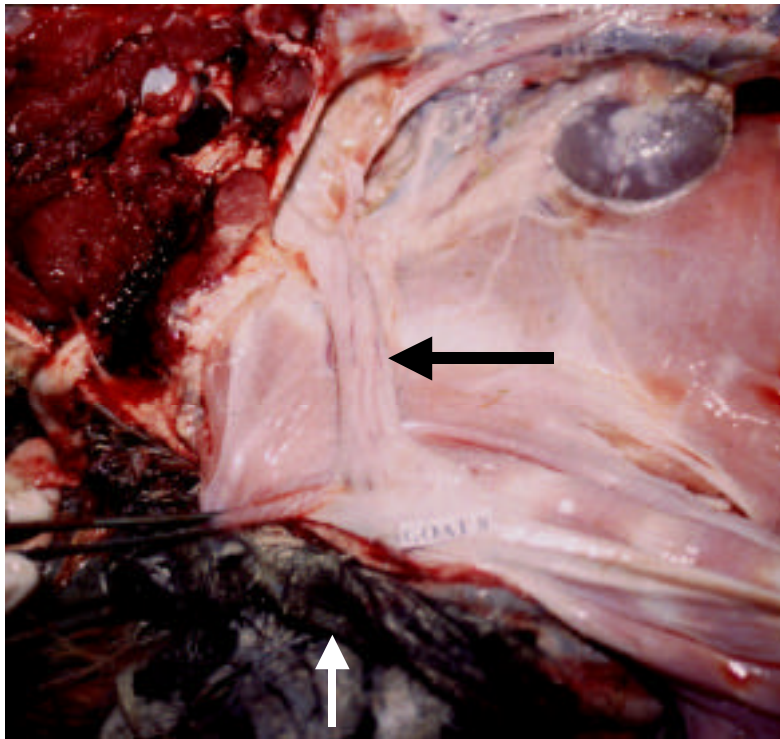


Figure 8. The appearance of the urinary organs in situ at necropsy 6 months after urinary bladder marsupialization. The photograph is oriented so that the head is toward the right of the image. The urinary bladder is tubelike in shape (indicated by the black arrow). The marsupialization stoma is indicated by the white arrow.

cystitis with visible bacterial adherence to the bladder mucosa. No significant findings were observed on evaluation of the ureters of Goats 2, 3, and 6. Minimal infiltration of lymphocytes and plasma cells was observed in the ureters of Goat 5 and Goat 8, and mild perivascular infiltration of lymphocytes and plasma cells was observed in Goat 1. The kidneys of Goat 1 were histopathologically normal, whereas those of all other goats exhibited mild lymphoplasmacytic pyelitis with occasional lymphoid aggregation. The histopathologic scores are reported in Appendix 11.

Urine culture yielded bacterial growth in all goats for which urine samples were submitted. No urine was present in the urinary bladder of Goat 8 at necropsy. Three of five samples yielded heavy growth of *Enterococcus* species and group D *Streptococci*. Urine culture from Goat 1 yielded heavy growth of *Actinomyces pyogenes* and urine culture from Goat 5 yielded *Staphylococcus xylosus*. Culture from a swab of the urinary bladder mucosa yielded bacterial species identical to those obtained from urine in Goats 2,3, 5, and 6. Renal culture yielded bacterial growth in 3 of 6 goats. The bacterial species obtained were identical to those obtained from culture of the urinary bladder, but were present in lower numbers than those in the bladder. Results of bacteriologic cultures are reported in Appendix 12.

Student's t-test analysis of Day 0 versus Day 180 yielded significant decreases in stomal diameter ($p < 0.0001$), total WBC ($p = 0.0454$), and serum creatinine concentration ($p = 0.0060$). Mean stomal diameter was 3.25 cm on Day 0 and 0.53 cm on Day 180. Mean total WBC on Day 0 was 24,683 cells/ μ L and 18,500 cells/ μ L on Day 180 (normal range, 3700-12900 cells/ μ L). The median total WBC decreased from Day 0 to Day 90, then increased from Day 90 to Day 180 (Appendix 14). However, the mean total WBC

did not reflect this trend (Appendix 14). Serum creatinine concentration decreased from a mean of 0.93 mg/dL on Day 0 to a mean of 0.73 mg/dL on Day 180 (normal range, 0.8-1.8 mg/dl) (Appendix 15). Statistically significant increases were observed in serum sodium concentration ($p=0.0024$), and serum chloride concentration ($p=0.0006$). Sodium concentration increased from a mean of 141.83 mmol/L on Day 0 to a mean of 147 mmol/L on Day 180 (normal range, 145-155 mmol/L). Mean serum chloride concentration increased from 102.67 mmol/L on Day 0 to 108 mmol/L on Day 180 (normal range, 102-114 mmol/L) (Appendix 16). Although not statistically significant, there was a trend toward increase in serum phosphorus and calcium levels from Day 90 to Day 180 (Appendix 17).

CONCLUSIONS AND DISCUSSION

Urinary bladder marsupialization did not appear to negatively impact the overall health of goats in the study, with the exception of one goat (Goat 1) that developed suppurative, necrotic cystitis secondary to stomal occlusion. All goats ate normally and gained weight, and no clinical signs of illness were observed during the study period. Weight loss observed from surgery to 30 postoperative days and from 60 to 90 postoperative days was attributed to inclement weather conditions affecting the availability of feed resources; all goats gained weight when supplemented with additional grain (Appendix 13).

Subjectively, stomal management was accomplished with minimal to moderate investments of time and effort. Stomal care included periodic visualization, clipping of ventral abdominal hair, application of zinc oxide ointment, and bathing as necessary. These procedures were required more frequently during inclement weather conditions such as ice and snow, and increased frequency of care subjectively decreased the time investment at each subsequent assessment period. Periodic bathing decreased the severity of urine odor as well as the effort required for subsequent stomal care. The application of zinc oxide ointment subjectively improved urine scald dermatitis, possibly due to prevention of urine contact with the skin as well as its astringent function. These observations are similar to those of May et al (1998), who reported 87.5% owner satisfaction with the procedure. Owners that were dissatisfied cited urine odor as the primary reason for dissatisfaction; the quality of owner compliance with recommended

care was unknown for these animals, and urine odor may have been less distasteful to those owners if additional stomal care efforts were made.

During the first 3 months of the study, all goats were incontinent; however, all goats except Goat 8 exhibited an apparent, gradual increase in the amount of urine storage and intermittent urine voiding during the second half of the study. Although a statistical correlation was not present, the volume of urine storage and frequency of urination appeared to correlate with decreased stomal diameter. This was most pronounced in Goat 5, which developed stomal stricture at 5 postoperative months.

Although ligation of the urethral process produced adequate short-term urinary outflow obstruction, urethral patency was reestablished when the urethral process sloughed. It was not known when the urethral process sloughed in each animal. Urination through the penile urethra was not observed in any of the goats except Goat 5. In Goat 5, the reestablishment of urethral patency and urinary outflow may have served a protective function when the marsupialization stoma strictured. On the other hand, the reestablishment of normal urinary outflow when the marsupialization stoma was patent but small in diameter may have decreased the physiologic stimulus for maintaining stomal patency.

Preoperative ultrasonographic examination revealed hydroureter and hydronephrosis in Goat 5; this examination was performed immediately prior to surgery, approximately 12 hours after urethral process ligation. Ultrasonographic assessment of all other goats was performed prior to urethral process ligation. The hydroureter/hydronephrosis observed in Goat 5 had markedly improved within one postoperative week, and had resolved by 30 postoperative days. The observation of

hydroureter and hydronephrosis in association with urinary outflow obstruction was not unusual, and may also have been observed in other goats if ultrasonographic examination had been performed at the same time relative to urethral process ligation. The resolution of hydroureter/hydronephrosis in Goat 5 was similar to that observed in human patients following cutaneous vesicostomy (Cohen et al, 1978; Sonda and Solomon, 1980; Agarwal et al, 1997), and emphasizes that resolution of urinary outflow obstruction may produce resolution of the sequelae of these problems.

Renal ultrasonography allowed noninvasive assessment of renal morphology throughout the study period. Correlation of ultrasonographic appearance with histopathologic appearance could not be made due to a lack of variation in histologic score within the study population. However, the absence of ultrasonographically detectable lesions throughout the study appeared to correlate with the absence of gross lesions at necropsy. The difficulty of ultrasonographic imaging increased as the animals grew in size, and accurate, repeatable measurements of midsagittal renal length could not be obtained at each interval due to variations in renal position and increasing body size. Ruminal distention markedly impacted imaging ability by affecting the position of the left kidney relative to midline and the position of the right kidney relative to the paralumbar fossa.

Despite the statistically significant increase observed in serum sodium and chloride concentrations from Day 0 to Day 180, all values remained near or within normal range and may reflect normal variation (Appendix 16). The observed significant decrease in serum creatinine concentration was below normal range values by 0.07 mmol/L, and was of questionable clinical relevance (Appendix 15).

A nonsignificant increasing trend in phosphorus and calcium levels was observed during the second half of the study (Appendix 17). Phosphorus and calcium levels remained within normal limits during the study period, but these trends may have become statistically and/or clinically significant if the study duration was lengthened.

The total WBC for all goats was persistently elevated throughout the study period (Appendix 14). However, the WBC values and differential counts indicated physiologic neutrophilia and lymphocytosis, and physical examination parameters obtained at the time of sample collection supported the theory that excitement was the inciting factor of the observed values. Only Goat 6 began to show clinicopathologic evidence of inflammation from 4 months to the completion of the study period, characterized by an increase in immature neutrophils; no immature neutrophils were observed in any other goat throughout the study period. The elevated peripheral WBC values in Goat 6 did not correlate to increased severity of histopathologic score at necropsy. Bacterial growth from renal tissue was heaviest in Goat 6, and may correlate to the increased leukocyte values. Although no gross evidence of other causes of inflammation was identified at necropsy, the inflammation may have been unrelated to the urinary tract.

Other than the altered anatomic configuration of the urinary bladder and mild omental adhesions to the surgery sites at necropsy, the marsupialization procedure did not produce any detectable changes in the gross appearance of the urinary organs in Goats 2, 3, 5, 6, and 8. The presence of mild to moderate histologic lesions in the upper urinary tract of these goats correlates with the absence of gross abnormalities. The severe cystitis observed in Goat 1 produced severe gross abnormalities of the urinary bladder, but no detectable abnormalities of the upper urinary tract organs were observed. The complete

absence of renal histologic abnormalities in Goat 1 was surprising, especially when compared to renal histopathologic score of 1 in all other goats (Appendix 11). It is possible that the development of severe, suppurative cystitis produced septicemia and death prior to the development of renal compromise.

Urine culture yielded bacterial growth in all goats for which urine samples were submitted (Appendix 12). No urine was present in the urinary bladder of Goat 8 at necropsy. Three of five samples yielded heavy growth of *Enterococcus* species and group D *Streptococci*, indicating environmental contamination of the urine. Urine culture from Goat 1 yielded heavy growth of *Actinomyces pyogenes*, indicating that the skin flora was the source of urine contamination. The growth of *Staphylococcus xylosus* in Goat 5 likely indicates environmental sources of contamination of the urine.

Culture from a swab of the urinary bladder mucosa yielded bacterial species identical to those obtained from urine in Goats 2, 3, 5, and 6 (Appendix 12). This was expected, indicating that urine cultures were actual and not the result of iatrogenic contamination during urine sampling. However, only the urinary bladder of Goat 1 showed histologic evidence of bacterial adherence. The absence of bacterial mucosal adherence and infiltration in the organs of Goats 2, 3, 5, 6, and 8, coupled with the presence of submucosal and intersitital lymphoplasmacytic infiltration, may reflect that the urinary tract was able to clear bacteria but was not able to reestablish homeostasis.

Renal culture yielded bacterial growth in 3 of 6 goats (Appendix 12). The bacterial species obtained were identical to those obtained from culture of the urinary bladder, but yielded less growth than those from the bladder. These findings suggest that bacteria ascended the urinary tract. Despite bacterial growth from the kidneys, there was

no evidence of suppurative pyelonephritis at the time of necropsy. A longer study period may have resulted in clinical or histologic evidence of suppurative pyelonephritis; however, the absence of clinical signs of pyelonephritis in clinical cases with up to four years of follow-up suggests that the animals may be able to maintain normal renal function despite the presence of bacteria and inflammation (May et al, 1998).

It has been observed that unobstructed urine flow is required for the maintenance of bladder asepsis, and obstruction of flow and the presence of residual urine predispose the bladder and upper urinary tract to infection (Parsons et al, 1975; Hosgood and Hedlund, 1993). It is generally considered that urine retention and the presence of bacteria are required for the development of urinary tract infection (Lulich et al, 1997), and significant urinary tract infections are believed to result from high intravesical pressures (Sonda and Solomon, 1980; Hurwitz and Ehrlich, 1983). Although urinary bladder marsupialization prevents urine storage in the early postoperative period, decrease in stomal size over time correlated to an apparent trend toward urine storage and intermittent urine voiding from the stoma. Retention of urine may be more acceptable to owners of pet goats, but is likely to predispose the animals to urinary tract infection and decrease the postoperative longevity of affected animals.

The development of suppurative cystitis in Goat 1 was likely a sequela of stomal occlusion by fecal material and hair; this led to the retention of contaminated urine, and resulted in the development of severe infection. This occurred during inclement weather conditions (snow and ice), when all goats were observed to spend increased amounts of time within a small, covered enclosure. Because Goat 1 was the most dominant member of the study group, this goat was more likely to be found in the enclosure at any given

time; this may have predisposed this goat to fecal contamination of the stoma. In addition, reluctance to keep the abdomens shaved during inclement weather conditions (in order to afford the greatest warmth to the animals) may have increased the likelihood of urine scald dermatitis due to urine retention on the abdominal hair. The death of this goat emphasizes the need for owner compliance in the management of marsupialized goats, especially during inclement weather conditions. Maintenance of a clean environment, especially when animals are housed in smaller enclosures or at higher stocking rates, has a marked impact on the ease of stomal care and the contamination of the stomal site.

Stomal stricture occurred in one of six goats in this study. Although a smaller number of animals were included in this study, this observation was comparable to those previously observed. Stomal stricture rates reported in human studies range from 17-20%, and in the previously reported retrospective clinical study, stricture was observed in 2 of 19 goats (May et al, 1998). Factors that may have influenced the rate of stricture include the young age of goats in the study population, the presence of peristomal dermatitis, the size of the incision in the abdominal fascia, and the degree of tension placed on the bladder at the stoma. It is possible that the stomas of young animals may reduce in size or develop strictures at faster rates due to increased growth rates. In addition, reestablishment of urethral patency may favor stomal closure.

Because dermatitis reflects localized inflammatory processes, and inflammation affects the rate and severity of fibrosis, stomal stricture may be accelerated by severe peristomal urine scald dermatitis. This was supported by the observations of Cohen et al

(1978) and the subjective observations in this study that stomal diameter appeared to be proportional to urine scald dermatitis.

Undersized abdominal fascial openings have been incriminated in stomal stricture (Duckett, 1974; Hurwitz and Ehrlich, 1983), probably due to early restriction of stomal size. The abdominal fascial incisions (mean and median, 2.25 cm) in the study animals approximated the “ideal” length of 2 cm proposed for small animal and human patients (Hurwitz and Ehrlich, 1983; Bjorling and Howard, 1989; Bjorling and Peterson, 1990). Subjectively, this length corresponded to the ability to introduce an index finger to at least the depth of the first knuckle through the completed stoma. Further studies may be indicated to determine the ideal stomal size in male ruminants. The absence of stomal prolapse in any of the goats in this study may indicate that the abdominal fascial incision was not excessive.

Tension on the bladder at the junction with the abdominal wall may affect stomal stricture by increasing local inflammation and impairing normal wound healing processes. Excess tension can be reduced by attention to proper surgical technique and proper placement of the stoma.

The complete stomal stricture and reestablishment of normal urinary outflow observed in Goat 5 led to speculation regarding surgical closure, or reversal, of urinary bladder marsupialization stomas. This goat was able to void urine normally when stomal stricture was observed, indicating that detrusor function was preserved and/or restored. Reversal of urinary bladder marsupialization was not performed in this study. However, this procedure may be of benefit in animals that reestablish urethral patency during the postoperative period. Advantages of reversal may include reestablishment of normal

anatomy and urine outflow, minimization of postoperative care, and reduction of dermatitis. Potential disadvantages of reversal include recurrence of urinary obstruction (due to formation of additional calculi), ascending infection, surgical difficulty due to peristomal fibrosis, and the need for general anesthesia. The reversal procedure would involve en bloc resection of the stoma and primary closure of the urinary bladder. It is imperative that urethral patency be confirmed by urethrography or, at minimum, observation of normal urination prior to surgical reversal. In addition, pre- or intraoperative urine cultures should be obtained to direct postoperative antibiotic therapy.

The use of stomal devices for the preservation of stomal diameter was not evaluated in this study. However, experimental evaluation of the use of such devices may be an important component of further studies to improve the outcome of urinary bladder marsupialization. Benefits to the use of these devices may include maintenance of functional stomal diameter over longer periods, decreased urine scald, and facilitation of postoperative care. Perhaps the largest obstacle to overcome is the lack of dietary discretion observed in small ruminants, especially in goats; the ingestion of stomal devices would obviously nullify any benefit that could be gained by their use. In addition, the currently available devices are marketed for use in human patients, and the cost associated with their application in most animal patients is prohibitive.

The rate of complications observed in this study were similar or reduced when compared to reported complication rates associated with other procedures utilized in the treatment of urinary outflow obstruction. However, this statement should be interpreted with caution, because the animals used in this study were normal animals without urinary outflow obstruction or urinary tract disease. The primary goal of this study was not to

compare complication rates with other studies, but to assess the clinicopathologic and histopathologic effects of the procedure when performed on normal goats.

It is important to note that urinary bladder marsupialization is not an ideal technique for the treatment of urinary outflow obstruction in small ruminants. Although good clinical results are observed as long as stomal patency is maintained or urethral patency is reestablished, the presence of bacteria and histologic observation of inflammation in the upper urinary tract indicate that bacterial ascent and inflammation occur following urinary bladder marsupialization. The use of urinary bladder marsupialization may still offer the best clinical outcome in animals with ruptured urethrae or those that have undergone multiple prior procedures, but is recommended with caution as primary surgical intervention during the first episode of urinary outflow obstruction. Goals for improving the marsupialization procedure include the maintenance of adequate stomal size for longer duration, prevention of urine retention, and minimization of urine contamination of the ventral abdomen. Proper attention to prevention of urolithiasis is vital to improving the health of animal species, and no surgical technique is as valuable as measures that may prevent the necessity for surgical intervention.

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APPENDICES

Appendix 1

Surgical Data

Goat #	Skin incision length	Body wall incision length	Final stomal diameter	Distance from preputial orifice to stoma	Celiotomy length
	(cm)	(cm)	(cm)	(cm)	(cm)
1	4.0	2.0	3.0	8.0	6.0
2	4.0	2.5	4.0	7.0	6.0
3	3.5	2.5	3.0	4.5	5.0
5	4.0	2.0	3.0	5.0	6.0
6	3.5	2.5	3.5	7.0	7.0
8	4.0	2.0	3.0	6.0	5.5

Appendix 2

Goat Examination Data (page 1 of 2)

DATE	GOAT #	TEMP. (°F)	PULSE	RESP. RATE	WEIGHT (kg)	SCALD SCORE	STOMAL DIAMETER (cm)
DAY 0	1	102.8	120	20	15.5	n/a	n/a
	2	102.8	114	20	13.2	n/a	n/a
	3	102.8	128	24	7.7	n/a	n/a
	5	102.4	164	28	16.8	n/a	n/a
	6	101.4	136	20	15.5	n/a	n/a
	8	103.2	176	32	11.4	n/a	n/a
DAY 7	1	102.4	124	20	n/a	n/a	n/a
	2	102.3	132	20	n/a	n/a	n/a
	3	102.2	140	20	n/a	n/a	n/a
	5	102.3	160	28	n/a	n/a	n/a
	6	100.9	102	28	n/a	n/a	n/a
	8	101.9	142	20	n/a	n/a	n/a
DAY 30	1	102.8	104	20	14.5	1	1.5
	2	101.9	140	20	11.8	3	2.5
	3	102.2	138	24	8.9	2	2.0
	5	101.5	118	20	15.8	2	2.0
	6	102.3	100	16	14.5	2	2.0
	8	102.2	152	20	9.3	2	2.25
DAY 60	1	102.6	100	16	15.8	2	2.0
	2	104.2	116	20	14.8	3	2.0
	3	103.3	136	28	9.5	2	0.75
	5	102.2	120	20	18.2	2	1.25
	6	102.7	126	20	16.4	2	1.75
	8	103.5	148	20	12.4	2	1.5
DAY 90	1	101.7	96	16	14.1	2	1.25
	2	102.0	120	24	13.6	2	1.25
	3	102.0	124	20	8.2	2	1.0
	5	101.1	88	28	17.3	1	0.5
	6	101.9	96	32	15.5	2	1.25
	8	101.2	100	28	12.3	3	1.5

Appendix 2

Goat Examination Data (page 2 of 2)

DATE	GOAT #	TEMP. (°F)	PULSE	RESP. RATE	WEIGHT (kg)	SCALD SCORE	STOMAL DIAMETER (cm)
DAY 120	1	102.7	88	16	16	2	0.7
	2	102.1	96	24	13.7	2	1.0
	3	102.3	100	38	9.7	3	0.6
	5	101.2	86	24	16.9	0	0
	6	101.8	104	28	16.5	2	0.8
	8	101.6	104	24	11.5	3	0.7
DAY 150	1	n/a	n/a	n/a	n/a	n/a	n/a
	2	101.3	96	20	14.3	1	1.2
	3	101.7	110	24	10.2	2	0.4
	6	100.9	112	20	18.1	2	0.7
	8	101.5	136	20	13.7	3	0.8
DAY 180	1	n/a	n/a	n/a	n/a	n/a	n/a
	2	103.2	120	30	15	0	0.4
	3	103.0	156	32	11.4	2	0.4
	6	103.1	132	24	19.5	0	0.3
	8	102.9	142	26	14.1	1	1.0

Appendix 3

Goat 1 Clinical Pathology Data

PARAMETER	DAY 0	DAY 7	DAY 30	DAY 60	DAY 90	DAY 120	DAY 150	DAY 180
RBC (X 10 ³ /μl)	13.59	10.61	14.93	13.04	14.6	17.72	n/a	n/a
HGB (gm/dl)	10.5	8.2	10.5	9.3	10	11.4	n/a	n/a
HCT (%)	30	24	29	28	27.5	32.5	n/a	n/a
MCV (fl)	22	23	19	21	19	18	n/a	n/a
MCH (pg)	7.7	7.7	7	7.1	6.8	6.4	n/a	n/a
MCHC (%)	35	34.2	36.2	33.2	36.4	35.1	n/a	n/a
WBC (X 10 ³ /μl)	28.8	20.6	19.4	19	17.5	17.4	n/a	n/a
SEG (X 10 ³ /μl)	20.16	9.476	9.894	8.36	8.575	6.96	n/a	n/a
BAND (X 10 ³ /μl)	0	0	0	0	0	0	n/a	n/a
LYMPH (X 10 ³ /μl)	8.64	10.712	8.924	10.45	8.75	9.57	n/a	n/a
MONO (X 10 ³ /μl)	0	0	0.582	0.19	0.175	0.696	n/a	n/a
EOS (X 10 ³ /μl)	0	0.412	0	0	0	0	n/a	n/a
BASO (X 10 ³ /μl)	0	0	0	0	0	0.174	n/a	n/a
PLATELETS (X 10 ³ /μl)	CL	CL	CL	CL	CL	CL	n/a	n/a
FIBRINOGEN (mg/dl)	200	100	100	100	100	200	n/a	n/a
P. PROTEIN (g/dl)	6.6	6.1	6.5	6	6.3	6.3	n/a	n/a
T. PROTEIN (g/dl)	6.4	5.7	6.4	6.1	6.3	6.3	n/a	n/a
ALBUMIN (g/dl)	2.8	2.4	2.9	2.6	2.7	2.7	n/a	n/a
BUN (mg/dl)	18	19	23	18	22	14	n/a	n/a
CREATININE (mg/dl)	0.9	0.7	0.6	0.6	0.6	0.6	n/a	n/a
TOTAL BILI. (mg/dl)	0.4	0.3	0.3	0.3	0.3	0.3	n/a	n/a
GGT (U/l)	79	73	80	80	77	93	n/a	n/a
AST (U/l)	163	138	114	115	97	108	n/a	n/a
SODIUM (mmol/l)	139	142	142	142	143	142	n/a	n/a
POTASSIUM (mmol/l)	3.96	4.51	4.48	4.05	3.84	3.47	n/a	n/a
CHLORIDE (mmol/l)	101	105	102	103	104	101	n/a	n/a
CALCIUM (mg/dl)	9.42	9.59	9.62	8.75	8.27	9.01	n/a	n/a
PHOSPHORUS (mg/dl)	4.2	4.7	4.2	4.4	2.6	3.4	n/a	n/a
MAGNESIUM (mg/dl)	1.8	1.8	2.1	2.2	2.2	1.8	n/a	n/a

Appendix 4

Goat 2 Clinical Pathology Data

PARAMETER	DAY 0	DAY 7	DAY 30	DAY 60	DAY 90	DAY 120	DAY 150	DAY 180
RBC (X 10 ³ /μl)	11.65	10.67	13.36	11.16	13.2	17.73	14.27	17.33
HGB (gm/dl)	9	8.6	9.6	7.5	8.5	10.9	9.3	10.5
HCT (%)	27	24	29	22	23	32	31	30
MCV (fl)	23	22	22	20	17	18	22	17
MCH (pg)	7.7	8.1	7.2	6.7	6.4	6.1	6.5	6.1
MCHC (%)	33.3	35.8	33.1	34.1	37	34.1	30	35
WBC (X 10 ³ /μl)	20.8	32	20.1	17.7	22.3	26.7	24.4	18.8
SEG (X 10 ³ /μl)	9.776	25.92	14.07	11.51	12.488	18.423	11.712	7.896
BAND (X 10 ³ /μl)	0	0	0	0	0	0	0	0
LYMPH (X 10 ³ /μl)	9.984	5.76	4.623	5.841	9.366	7.209	11.468	9.4
MONO (X 10 ³ /μl)	0.208	0	1.005	0.177	0.223	0.801	0	0.564
EOS (X 10 ³ /μl)	0.832	0.32	0.201	0.531	0	0	0.976	0.752
BASO (X 10 ³ /μl)	0	0	0	0	0.223	0.267	0.244	0.188
PLATELETS (X 10 ³ /μl)	CL	CL	CL	CL	CL	CL	CL	CL
FIBRINOGEN (mg/dl)	100	300	100	200	100	100	200	100
P. PROTEIN (g/dl)	7.6	6.8	6.9	7.5	7.4	7.6	7.3	7.3
T. PROTEIN (g/dl)	7.5	6.6	6.8	7.6	7.3	7.6	7.3	7.3
ALBUMIN (g/dl)	2.9	2.6	2.8	2.8	2.8	3.1	2.9	3
BUN (mg/dl)	15	10	25	14	21	16	14	12
CREATININE (mg/dl)	0.8	0.6	0.6	0.6	0.6	0.6	0.6	0.7
TOTAL BILI. (mg/dl)	0.4	0.3	0.3	0.3	0.2	0.3	0.3	0.7
GGT (U/l)	128	114	77	83	75	90	76	86
AST (U/l)	132	468	124	128	113	128	126	111
SODIUM (mmol/l)	141	141	140	140	144	143	142	150
POTASSIUM (mmol/l)	4.75	4.49	3.8	3.93	3.59	3.84	3.94	3.97
CHLORIDE (mmol/l)	102	107	103	105	110	102	103	110
CALCIUM (mg/dl)	9.57	8.78	9.39	8.64	8.6	8.81	9.59	9.75
PHOSPHORUS (mg/dl)	8	4.8	3.5	4.5	4	4.3	7.4	7.8
MAGNESIUM (mg/dl)	1.9	1.9	2.1	2.3	2.2	2	2.1	2.2

Appendix 5

Goat 3 Clinical Pathology Data

PARAMETER	DAY 0	DAY 7	DAY 30	DAY 60	DAY 90	DAY 120	DAY 150	DAY 180
RBC (X 10 ³ /μl)	12.42	11.13	12.58	11.3	12.64	12.55	12.59	13.86
HGB (gm/dl)	9.6	9	9.6	8.7	9.6	9.1	9	9
HCT (%)	28.1	24	27.5	26	27	25.5	26.5	25
MCV (fl)	23	22	22	23	21	20	21	18
MCH (pg)	7.7	8.1	7.6	7.7	7.6	7.3	7.1	6.5
MCHC (%)	34.2	37.5	34.9	33.5	35.6	35.7	34	36
WBC (X 10 ³ /μl)	23.3	25.3	18.7	20.7	16.5	15.7	19.5	21.6
SEG (X 10 ³ /μl)	9.32	11.638	11.033	6.21	7.095	6.751	8.19	11.88
BAND (X 10 ³ /μl)	0	0	0	0	0	0	0	0
LYMPH (X 10 ³ /μl)	13.98	13.662	7.48	12.213	8.415	8.007	10.53	8.208
MONO (X 10 ³ /μl)	0	0	0.187	0	0.495	0.628	0.39	1.08
EOS (X 10 ³ /μl)	0	0	0	2.277	0.33	0.314	0.39	1.08
BASO (X 10 ³ /μl)	0	0	0	0	0.165	0	0	0.432
PLATELETS (X 10 ³ /μl)	CL	CL	CL	CL	CL	CL	CL	CL
FIBRINOGEN (mg/dl)	100	200	100	400	100	100	100	100
P. PROTEIN (g/dl)	6.5	6.2	6.4	7.2	7.3	6.8	7	6.2
T. PROTEIN (g/dl)	6.4	6	6.4	7.6	7.2	6.9	6.8	6.3
ALBUMIN (g/dl)	2.8	2.6	2.8	3	2.9	2.8	2.8	2.6
BUN (mg/dl)	15	13	29	20	22	16	17	14
CREATININE (mg/dl)	.09	0.9	0.7	0.6	0.7	0.7	0.6	0.7
TOTAL BILI. (mg/dl)	0.4	0.3	0.3	0.3	0.3	0.3	0.2	0.2
GGT (U/l)	87	85	73	74	72	71	69	66
AST (U/l)	129	183	133	131	106	102	117	109
SODIUM (mmol/l)	142	144	141	146	143	143	144	147
POTASSIUM (mmol/l)	4.06	4.27	4.51	3.69	4.39	3.23	3.98	3.69
CHLORIDE (mmol/l)	103	105	103	107	109	104	107	109
CALCIUM (mg/dl)	10.15	9.91	9.5	9.3	8.93	9.32	9.44	9.57
PHOSPHORUS (mg/dl)	4.6	4.8	6.1	4.4	3.6	2.8	5.5	6.3
MAGNESIUM (mg/dl)	2.3	2.8	2.2	2.5	2.3	2.2	2.2	2.4

Appendix 6

Goat 5 Clinical Pathology Data

PARAMETER	DAY 0	DAY 7	DAY 30	DAY 60	DAY 90	DAY 120	DAY 150	DAY 180
RBC (X 10 ³ /μl)	14.14	13.27	16.5	12.43	13.49	15.18	n/a	n/a
9.8	9.3	11	8.6	8.9	9.8	n/a	n/a	n/a
HCT (%)	30	30	31	27	26	27	n/a	n/a
MCV (fl)	21	23	19	22	19	18	n/a	n/a
MCH (pg)	6.9	7	6.7	6.9	6.6	6.5	n/a	n/a
MCHC (%)	32.7	31	35.5	31.9	34.2	36.3	n/a	n/a
WBC (X 10 ³ /μl)	30.7	30	19.1	20.6	24.4	16.2	n/a	n/a
SEG (X 10 ³ /μl)	9.824	22.5	13.752	10.094	12.2	7.776	n/a	n/a
BAND (X 10 ³ /μl)	0	0	0	0	0	0	n/a	n/a
LYMPH (X 10 ³ /μl)	19.648	22.5	13.752	10.094	12.2	7.776	n/a	n/a
MONO (X 10 ³ /μl)	0	0.3	0	0	0	0.162	n/a	n/a
EOS (X 10 ³ /μl)	1.228	0.6	0.191	2.06	0.488	0.648	n/a	n/a
BASO (X 10 ³ /μl)	0	0.3	0	0	0	0.162	n/a	n/a
PLATELETS (X 10 ³ /μl)	CL	CL	CL	CL	CL	CL	n/a	n/a
FIBRINOGEN (mg/dl)	200	300	200	100	100	200	n/a	n/a
P. PROTEIN (g/dl)	6.9	6.7	6.8	6.5	6.6	6.5	n/a	n/a
T. PROTEIN (g/dl)	6.7	6	6.6	6.6	6.9	6.5	n/a	n/a
ALBUMIN (g/dl)	2.7	2.4	3	2.6	2.8	2.7	n/a	n/a
BUN (mg/dl)	5	8	24	13	19	12	n/a	n/a
CREATININE (mg/dl)	1	1	0.8	0.8	0.9	0.8	n/a	n/a
TOTAL BILL. (mg/dl)	0.3	0.2	0.4	0.3	0.3	0.3	n/a	n/a
GGT (U/l)	78	65	72	77	71	78	n/a	n/a
AST (U/l)	141	106	136	138	134	113	n/a	n/a
SODIUM (mmol/l)	143	144	142	142	148	143	n/a	n/a
POTASSIUM (mmol/l)	4.21	3.39	4.7	3.3	4.1	3.31	n/a	n/a
CHLORIDE (mmol/l)	103	112	102	108	111	105	n/a	n/a
CALCIUM (mg/dl)	9.34	8.54	9.01	8.08	7.87	8.37	n/a	n/a
PHOSPHORUS (mg/dl)	5.3	3.8	4.7	4.3	2.8	3.6	n/a	n/a
MAGNESIUM (mg/dl)	2.4	2.2	2.3	2.5	2.5	2	n/a	n/a

Appendix 7

Goat 6 Clinical Pathology Data

PARAMETER	DAY 0	DAY 7	DAY 30	DAY 60	DAY 90	DAY 120	DAY 150	DAY 180
RBC (X 10 ³ /μl)	14.65	13.19	14.61	11.98	14.98	16.36	22.28	18.45
HGB (gm/dl)	10.3	9.7	10.4	8.5	9.8	10.5	11.8	11.8
HCT (%)	30	29	28	25	27	30	35	33
MCV (fl)	20	22	19	21	18	18	16	18
MCH (pg)	7	7.4	7.1	7.1	6.5	6.4	5.3	6.4
MCHC (%)	34.3	33.4	37.1	34	36.3	35	33.7	35.8
WBC (X 10 ³ /μl)	23.1	20.4	14.6	18.9	13.4	14.1	14.7	20.8
SEG (X 10 ³ /μl)	10.626	10.608	6.278	10.773	6.968	7.896	6.762	11.44
BAND (X 10 ³ /μl)	0	0	0	0	0	0	0	0.147
LYMPH (X 10 ³ /μl)	11.088	9.792	7.884	7.749	5.896	5.64	7.35	7.488
MONO (X 10 ³ /μl)	0.231	0	00.146	0.378	0.134	0.423	0.147	0.416
EOS (X 10 ³ /μl)	0.693	0	0.146	0	0.268	0.141	0.294	1.04
BASO (X 10 ³ /μl)	0.231	0	0.146	0	0.134	0	0	0.208
PLATELETS (X 10 ³ /μl)	CL	CL	CL	CL	CL	CL	CL	CL
FIBRINOGEN (mg/dl)	300	200	100	100	100	100	200	400
P. PROTEIN (g/dl)	7.1	6.8	7.2	6.6	7	6.6	7.2	6.4
T. PROTEIN (g/dl)	6.9	6.5	7.5	6.7	7.4	6.8	7.1	6.2
ALBUMIN (g/dl)	3.1	2.8	6.4	2.8	3.2	3	3.1	2.8
BUN (mg/dl)	10	29	31	16	18	12	14	17
CREATININE (mg/dl)	1.1	1.4	0.9	0.8	0.8	0.8	0.8	0.8
TOTAL BILL. (mg/dl)	0.4	0.3	0.4	0.3	0.3	0.3	0.3	0.4
GGT (U/l)	100	95	99	92	85	83	86	88
AST (U/l)	135	126	174	108	106	102	121	96
SODIUM (mmol/l)	143	142	142	142	145	143	143	146
POTASSIUM (mmol/l)	4.15	5.97	5.69	4.48	4.57	4.01	4.18	3.94
CHLORIDE (mmol/l)	104	108	103	109	112	106	105	105
CALCIUM (mg/dl)	9.5	9.71	10.24	8.8	8.93	8.96	9.25	9.22
PHOSPHORUS (mg/dl)	4.9	5.1	5	4.6	5.5	5.3	7.5	5
MAGNESIUM (mg/dl)	2.3	2.2	2.4	2.7	2.4	2.1	2.3	2.2

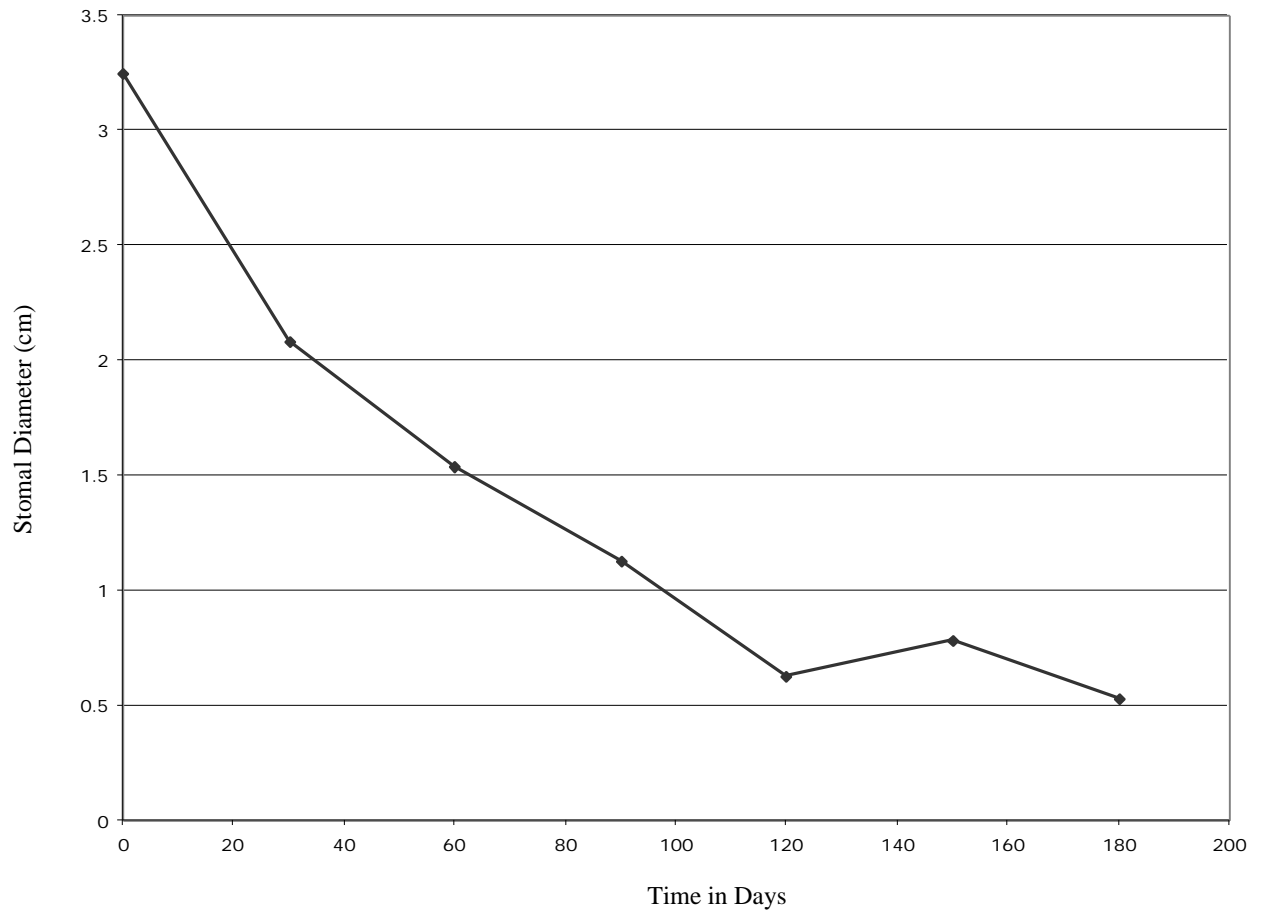
Appendix 8

Goat 8 Clinical Pathology Data

PARAMETER	DAY 0	DAY 7	DAY 30	DAY 60	DAY 90	DAY 120	DAY 150	DAY 180
RBC (X 10 ³ /μl)	12.38	11.35	16.57	10.82	11.73	11.28	10.34	10.65
HGB (gm/dl)	9	9.1	11.6	7.3	7.7	7.4	6.8	6.7
HCT (%)	29	25	34	19	22	20	20.5	18
MCV (fl)	23	22	21	18	19	18	20	17
MCH (pg)	7.3	8	7	6.7	6.6	6.6	6.6	6.3
MCHC (%)	31	36.4	34.1	38.4	35	37	33.2	37.2
WBC (X 10 ³ /μl)	21.4	34.1	23.5	19.3	15.8	17.9	17.4	12.8
SEG (X 10 ³ /μl)	6.206	21.483	14.1	10.8.8	9.164	10.74	11.136	7.808
BAND (X 10 ³ /μl)	0	0	0	0	0	0	0	0
LYMPH (X 10 ³ /μl)	14.552	12.276	8.93	8.106	5.372	6.444	5.916	3.456
MONO (X 10 ³ /μl)	0.428	0.341	0.47	0.386	0.632	0.537	0.174	1.28
EOS (X 10 ³ /μl)	0.214	0	0	0	0.316	0.179	0.174	0.256
BASO (X 10 ³ /μl)	0	0	0	0	0.316	0	0	0
PLATELETS (X 10 ³ /μl)	CL	CL	CL	CL	CL	CL	CL	CL
FIBRINOGEN (mg/dl)	100	300	300	200	200	100	100	100
P. PROTEIN (g/dl)	7.5	7.2	7.4	6.8	7.2	6.8	6.9	6
T. PROTEIN (g/dl)	7.3	7	7.4	7	7.2	6.9	7	6
ALBUMIN (g/dl)	3	3	3.4	2.6	2.4	2.5	2.6	2.3
BUN (mg/dl)	10	16	22	14	18	17	14	14
CREATININE (mg/dl)	0.9	0.9	0.6	0.7	0.7	0.7	0.6	0.7
TOTAL BILI. (mg/dl)	0.4	0.3	0.3	0.2	0.2	0.3	0.3	0.2
GGT (U/l)	79	77	72	88	62	73	98	57
AST (U/l)	130	143	132	142	141	145	155	136
SODIUM (mmol/l)	143	147	140	144	147	144	145	145
POTASSIUM (mmol/l)	4.94	4.6	5.36	4.56	3.75	3.95	4.39	4.68
CHLORIDE (mmol/l)	103	106	99	110	112	110	108	109
CALCIUM (mg/dl)	9.8	9.6	10	8.64	8.3	8.52	9.35	8.64
PHOSPHORUS (mg/dl)	5.8	5.4	5	4.5	5	5.3	6.2	6.1
MAGNESIUM (mg/dl)	2.5	2.4	2.4	2.4	2.4	2.2	2.1	2.3

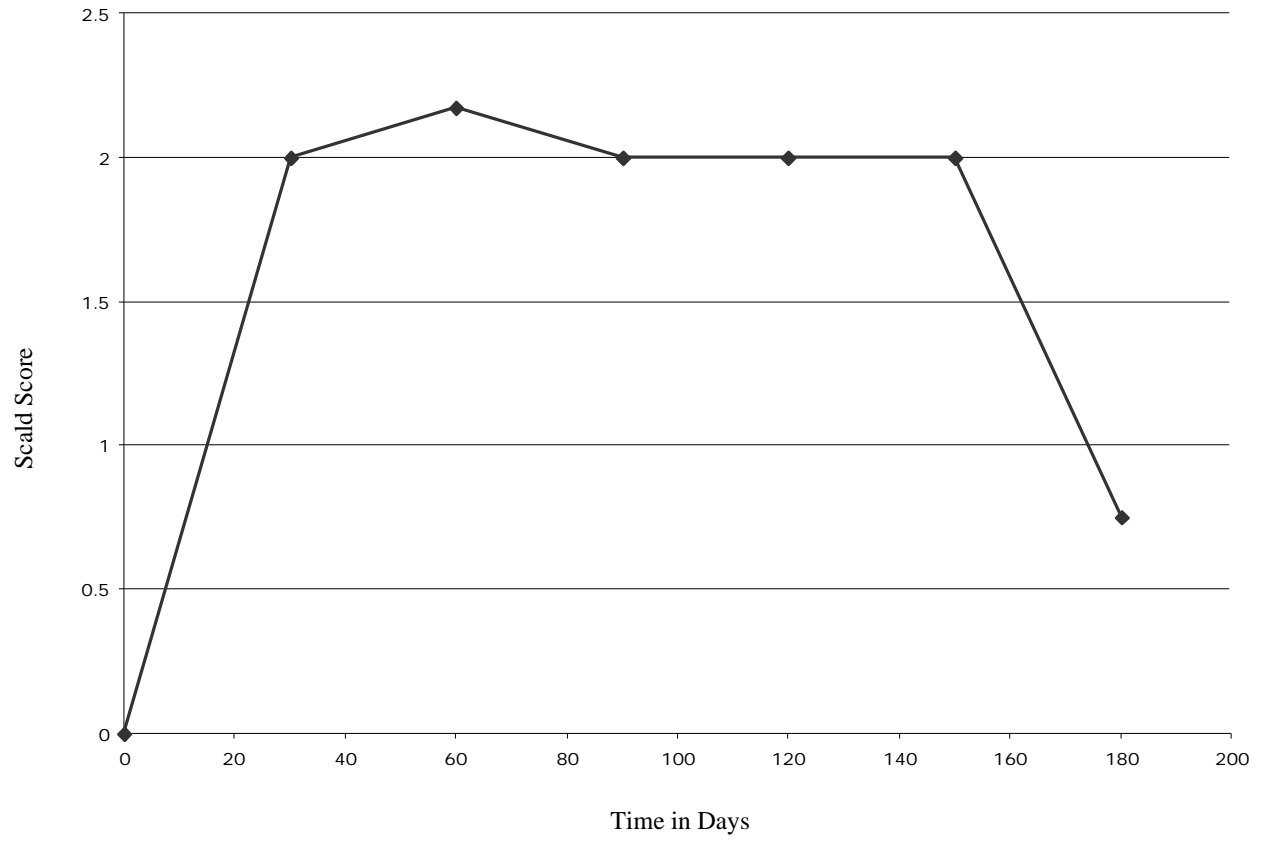
Appendix 9

Mean Stomal Diameter Over Time



Appendix 10

Mean Scald Score Over Time



Appendix 11

Histopathologic Scores

Organ	Goat 1	Goat 2	Goat 3	Goat 5	Goat 6	Goat 8	Mean	Median
Skin	3	2	2	1	2	2	2	2
Urinary bladder near stoma	4	0	2	3	2	2	2.2	2
Urinary bladder distant from stoma	4	1	1	1	1	1	1.5	1
Ureters	1	0	0	1	0	1	0.5	0.5
Kidneys	0	1	1	1	1	1	0.8	1

Scoring scale:

0 = Normal
 1 = Mild inflammation
 2 = Moderate inflammation
 3 = Marked inflammation
 4 = Severe inflammation

Appendix 12

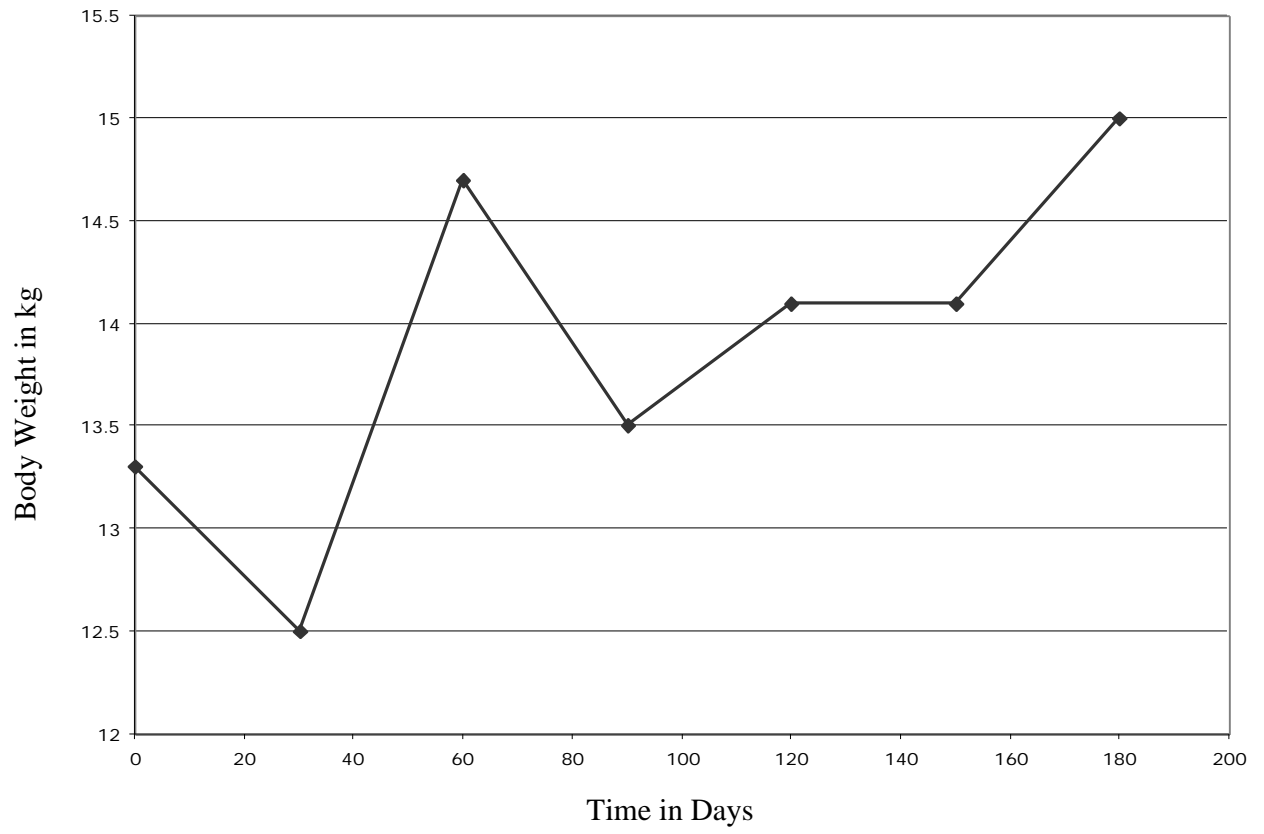
Bacteriologic Culture Results

Goat	Urine culture	Urinary bladder culture	Kidney culture
Goat 1	> 100,000 CFU/cc <i>Actinomyces pyogenes</i>	No result	No growth
Goat 2	1. 100,000 CFU/cc <i>Enterococcus</i> species 2. > 100,000 CFU/cc group D <i>Streptococci</i>	1. 4+ <i>Enterococcus</i> species 2. 4+ group D <i>Streptococci</i>	1. 2+ <i>Enterococcus</i> species 2. 2+ group D <i>Streptococci</i>
Goat 3	1. 300 CFU/cc <i>Enterococcus</i> species 2. 200 CFU/cc group D <i>Streptococci</i>	1. 4+ <i>Enterococcus</i> species 2. 4+ group D <i>Streptococci</i>	No growth
Goat 5	4+ <i>Staphylococcus xylosus</i>	4+ <i>Staphylococcus xylosus</i>	No growth
Goat 6	1. > 100,000 CFU/cc <i>Enterococcus</i> species 2. > 100,000 CFU/cc group D <i>Streptococci</i>	1) 4+ <i>Enterococcus</i> species 2) 4+ group D <i>Streptococci</i>	1) 3+ <i>Enterococcus</i> species 2) 3+ group D <i>Streptococci</i>
Goat 8	No urine obtained	1) 4+ <i>Enterococcus</i> species 2) 4+ group D <i>Streptococci</i>	1) 2+ <i>Enterococcus</i> species 2) 2+ group D <i>Streptococci</i>

CFU = Colony Forming Units

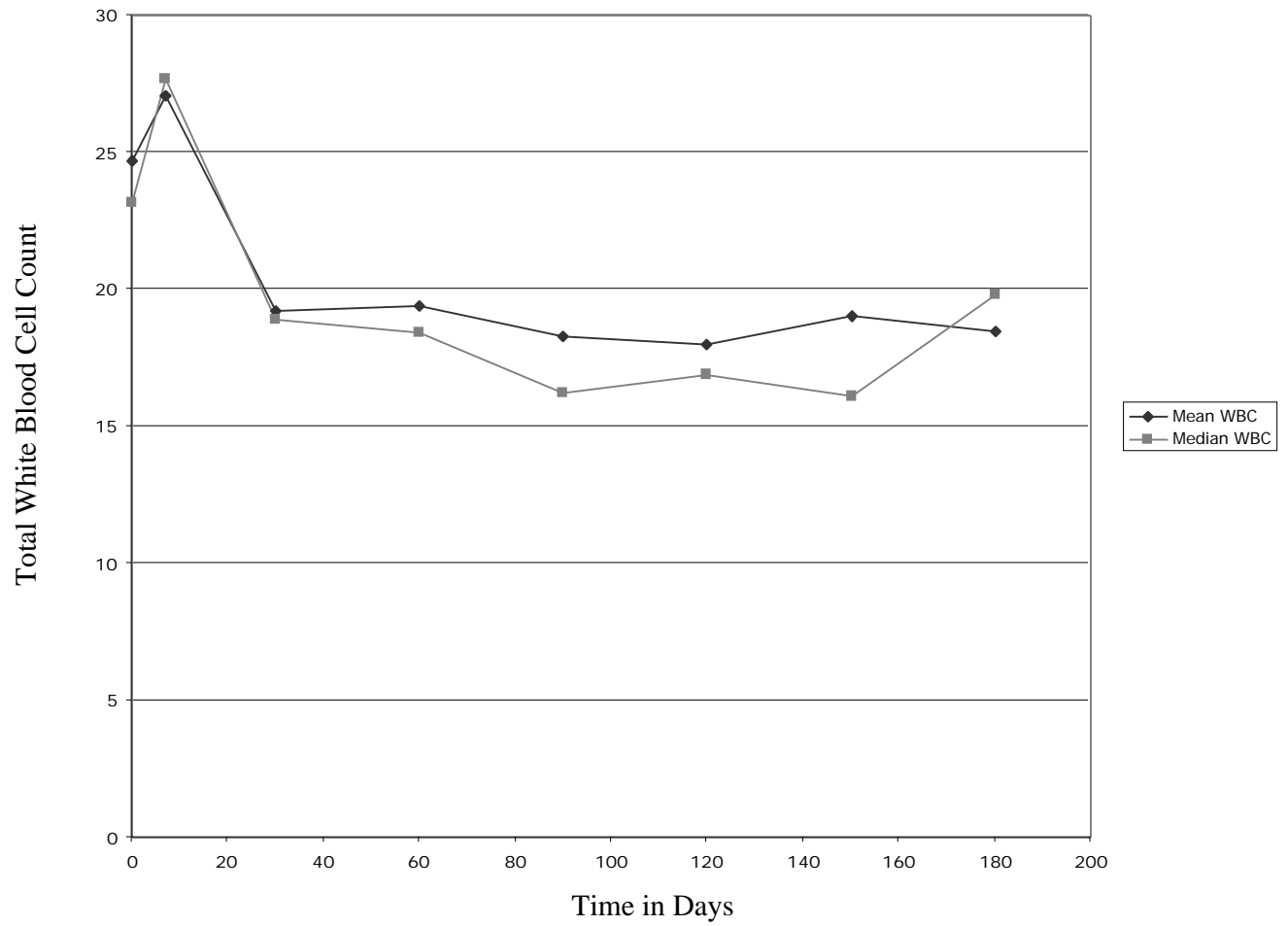
Appendix 13

Mean Body Weight Over Time



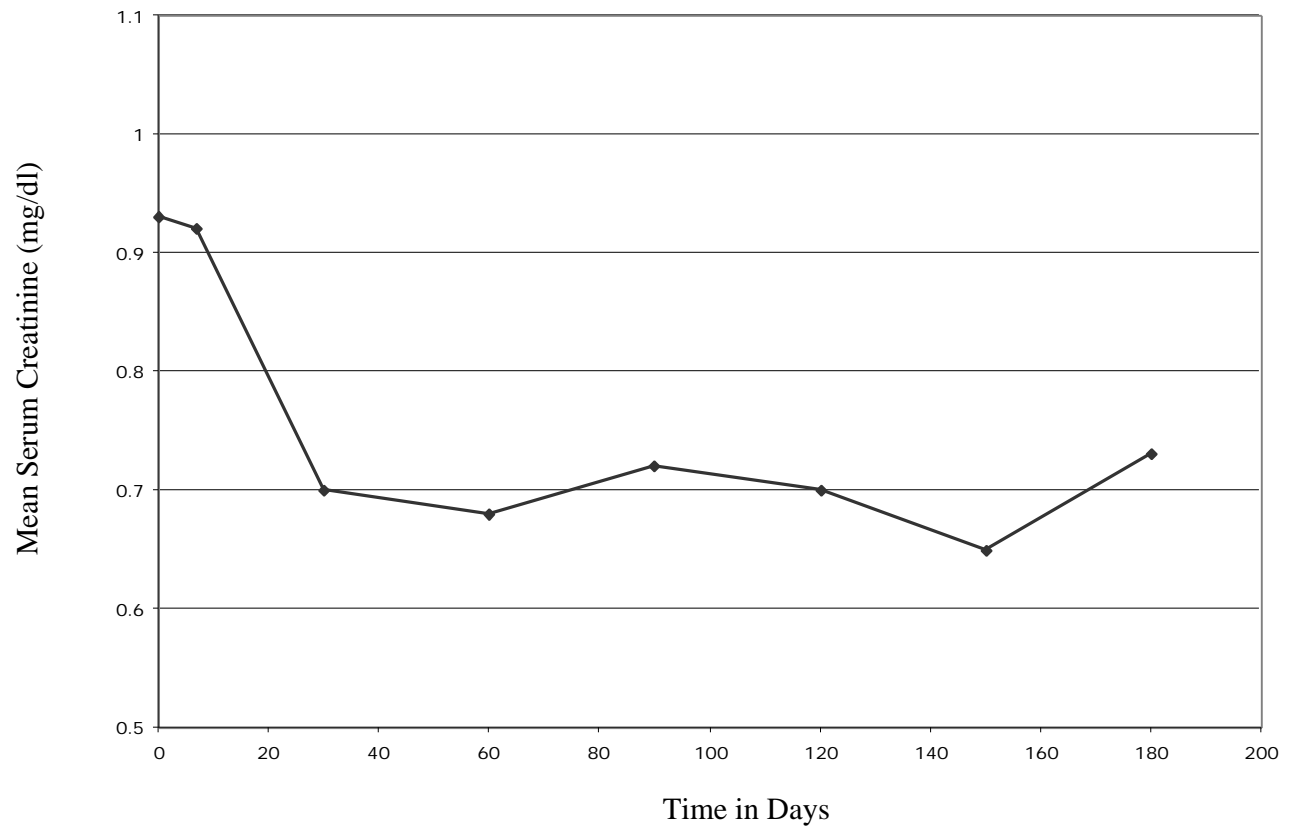
Appendix 14

Mean and Median White Blood Cell Counts Over Time



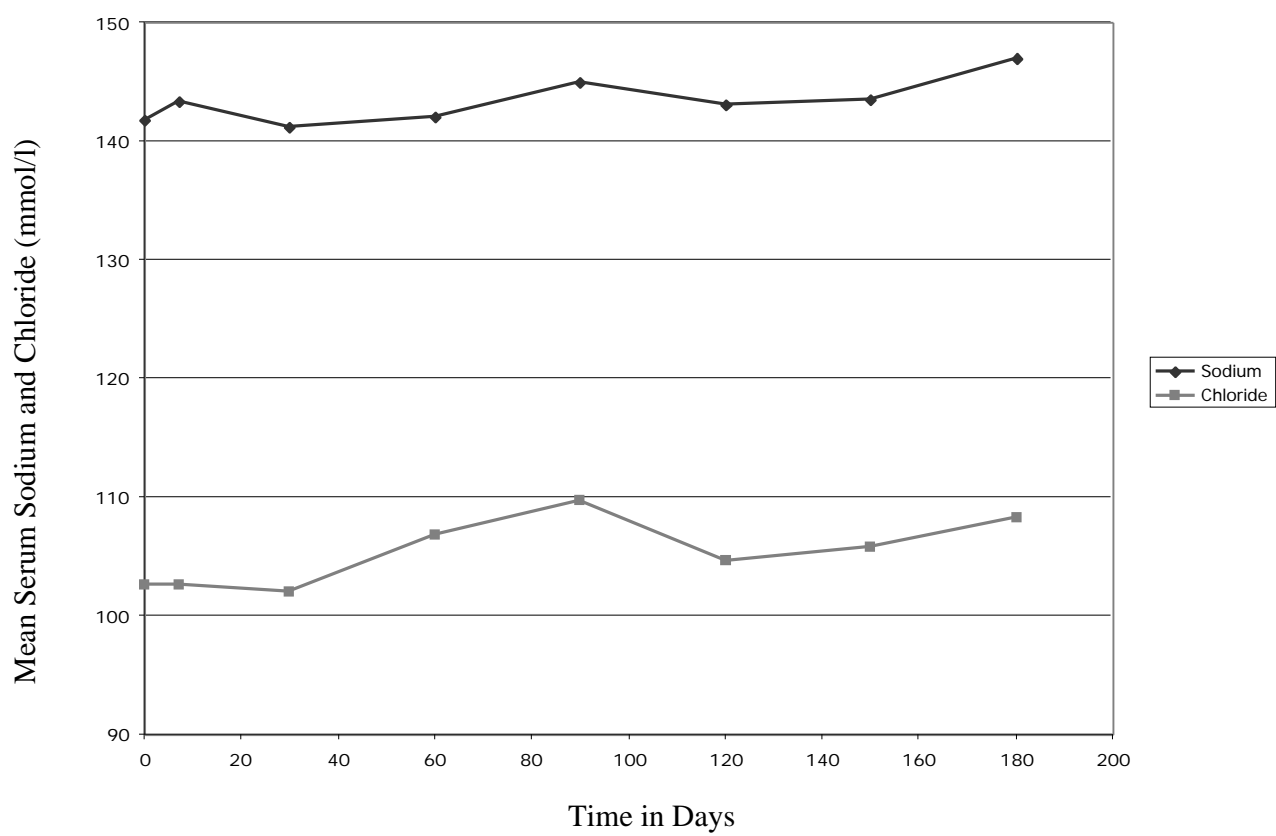
Appendix 15

Mean Serum Creatinine Values Over Time



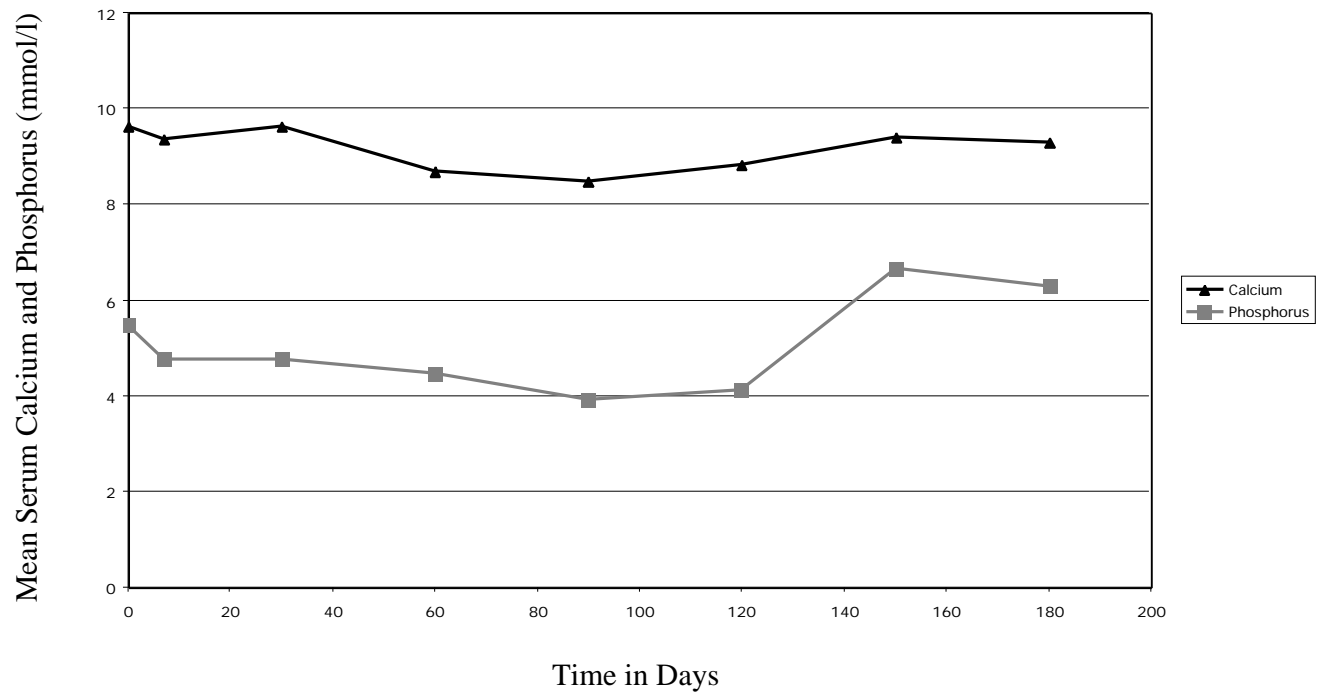
Appendix 16

Mean Sodium and Chloride Values Over Time



Appendix 17

Mean Serum Calcium and Phosphorus Values Over Time



VITA

Kimberly Anne May was born on June 17, 1969 in Homestead, Florida. At the age of two, she moved with her parents, Jon and Anne May, and her brother, Erik, to Gaithersburg, Maryland. She attended elementary, junior high, and high school in Gaithersburg.

Kimberly was admitted to Virginia Tech in 1987, where she majored in Animal Science with minors in Biology and English. She was accepted to the Virginia-Maryland College of Veterinary Medicine and graduated in 1994 as a Doctor of Veterinary Medicine.

Following an in-hospital internship in Equine Surgery and Medicine at Peterson, Smith, Matthews, Hahn, & Slone Equine Hospital from June 1994 to June 1995, Kimberly was hired as an associate veterinarian in a mixed animal clinic near Charlotte, North Carolina. She was accepted as a Large Animal Surgery Resident at the Virginia-Maryland College of Veterinary Medicine beginning in July 1996, and finished her residency program in July 1999.