CHAPTER 55

Rhinoceridae (Rhinoceroses)

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BIOLOGY

Rhinoceroses (rhinos) are among the most primitive of the world's large mammals, and in prehistoric times were common large herbivores in North America. Five extant species exist in four genera; in Africa, the white (*Ceratotherium simum*) and black (*Diceros bicornis*) rhinos and in Asia, the Sumatran (*Dicerorhinus sumatrensis*), Indian (*Rhinoceros unicornis*), and Javan (*Rhinoceros sondaicus*) rhinos. The Sumatran rhino is the most primitive and predates the extinct woolly rhino (*Coelodonta antiqitatis*), which inhabited in northern Europe and Asia during the last Ice Age.

African rhinos live in habitats that are related to dietary requirements; the white rhino requires relatively flat terrain with areas of short grass, whereas the black rhino prefers areas with shrubs and young trees. The Javan rhino inhabits lowland coastal forests rather than the more mountainous inland areas. Sumatran rhinos are found in areas of dense primary rain forest. Indian rhinos exist across a wide range of habitats, including marshes, alluvial plains, grasslands, and aril forests. All species of rhino require regular access to water. They need to drink daily or every second day, as they are hindgut fermenters with relatively fast gut transit times which reduce the time for water resorption from the feces.[35] Rhinos wallow in mud pools to cool off during the heat of the day and to help keep their skin free of external parasites.

Habitat loss and severe poaching has led to the devastation of rhino populations. Current worldwide population estimates (2012) are 35 to 44 Javan, 152 to 199 Sumatran, 3270 Indian, 4837 black, and 20,143 white rhinos (International Rhino Foundation; www.rhinos.org). In contrast, in the 1970s, the black rhino population in Africa was approximately 65,000.[22]

UNIQUE ANATOMY

Rhinos have a barrel-shaped torso; short, thick legs and broad feet with three weight-bearing digits; and an elongated, bulky skull. The soles of the feet have a large pad to cushion the weight of the animal. The most distinctive feature of rhino is the presence of a single horn (*Rhinoceros* sp.) or a pair of horns (*Ceratotherium*, *Diceros*, and *Dicerorhinus*) that are composed of tubular hairlike keratin filaments, which are outgrowths of the skin. The horn is relatively easy to displace, since it is not attached to the skull but rather set on bony protuberances.[35] African rhinos lack both incisors and canine teeth: incisors (I) 0, canines (C) 0, premolars (P) 3–4, molars (M) 3); in contrast, the Asian species have incisors: I 1/1–2, C 0, P 3–4, M 3).[28] Hypertrophied and tusklike lateral lower incisors are characteristic of the three Asian rhinos, with only members of the genus *Rhinoceros* having a pair of small central incisors. The black, Sumatran, and Javan rhinos are browsers with prehensile upper lips, which assist in grasping the plants that they consume. White and Indian rhinos have wide, flat lips for grazing.

The rhino's skin is thick, and in the white rhino, it may reach a thickness of 5 centimeters (cm), with a thick vascular dermis covered by an epidermis 1 millimeter (mm) thick.[35] All rhinos have skinfolds, although these are more pronounced in the Asian species, with the Indian rhino being best known for the exaggerated armorlike plates. The Sumatran rhino is unique, having a distinctive shaggy coat of hair with particularly hairy ears and a tuft of long hair at the tip of the tail.

SPECIAL HOUSING REQUIREMENTS

Enclosures for all rhino species need to be sturdy and constructed of concrete, large-diameter wooden or metal poles or of more natural

materials such as rocks; however, it should ensure that the animals cannot climb over or become entrapped within the enclosures. Spacing between vertical bars should be 0.5 m. If calves are present, chains or cables should be added. When horizontal poles are used, a potential for horn avulsion or climbing exists.

Substrate should be textured to minimize slipping yet not abrade foot pads. Most indoor stalls are made of concrete to facilitate cleaning. Natural substrate used in outdoor enclosures or pens should allow adequate drainage and cleaning or be periodically changed to prevent buildup of parasites, pathogens, or excessive moisture.

All rhinos must be provided with access to pools and wallows. The access and depth of the pool should be sufficient to encourage use and allow complete submersion, especially for Indian and Sumatran rhinos. Enclosure items such as scratching posts, rocks, and vegetation promote natural, species-specific behaviors but should be designed to avoid head or limb entrapment or horn rubbing or avulsion.

Rhinos may be acclimated to cold and inclement weather if provided access to shelter during wet and windy conditions. However, supplemental heat (to maintain 13°C) should be provided in cold climates when temperatures consistently drop below 10°C. Ill, old, or young animals may need higher temperatures.

Of the four rhino species, white rhinos are the most social and usually housed in groups. Black rhinos are more solitary but have been housed in small family groups in captivity. Indian rhinos are mostly solitary. Therefore they should be kept as individual animals except in very large spaces. Sumatran rhinos should also be housed individually and only introduced for breeding purposes. Significant aggression during breeding may occur with black, Indian, and Sumatran rhinos, and adequate space should exist to prevent cornering of animals.

FEEDING AND NUTRITION

Feeding strategies differ by species. Black and Sumatran rhinos are browsers, white rhinos are considered grazers, and Indian rhinos are classified as intermediate feeders. Studies on digestibility in captive rhinos show that the horse is a useful model for the development of diets for Indian and white rhinos but not for black rhinos.[7] Captive black rhinos appear to receive higher proportions of concentrates compared with other species and would benefit from higher proportion of browse.

In captivity, a rough guideline for diet quantity is 1% to 3% body weight as fed, with no more than one third of total calories obtained from pellets.[22] Energy management for weight control is especially important in Indian rhinos; 0.5% to 1.1% body weight in dry matter (DM) is adequate in this species.[9] Grass hay should be fed to white and Indian rhinos, whereas a grass–legume mixture or a legume–browse mixture is used for black and Sumatran rhinos. Alfalfa fed as exclusive forage may lead to mineral imbalances, colic, and diarrhea. Black rhinos appear to have higher calcium (Ca) and magnesium (Mg) absorption compared with horses and higher fecal losses of sodium (Na) and potassium (K).[7] Therefore, excessive mineral supplementation should be avoided. Natural browses appear to be limited in Na, phosphorus (P), zinc (Zn), and selenium (Se). Appropriate Ca:P ratio should also be monitored, since low phosphorus values have been associated with poorly defined syndromes, including dermatologic and hematologic disorders, in black rhinos.[10]

Vitamin E deficiency has been linked to health issues, especially in black rhinos. Therefore, the contents of the diet should be analyzed to ensure a sufficient concentration (150–200 international units per kilogram [IU/kg] DM).[22]

Black and Sumatran rhinos are susceptible to iron storage disorder in captivity, so a high-fiber, low-iron diet should be provided to these species. A low-iron herbivore pellet, in addition to browse, fed to captive black rhinos resulted in a decrease in serum ferritin levels.[27] Low-iron diets consisting of browse, long-stem forage, and low-iron pellets (iron [Fe] ≤350 parts per million [ppm]) is recommended for browser rhino species (Valdes E, personal communication). Citrus and other produce containing vitamin C may enhance iron absorption and should be minimized in the diets of these species.

Analyses of rhino milk have shown that it is lower in total solids compared with the milk of most ungulates (8.2%–8.8%). The relative composition is high in sugars (63%–82% of total solids), with 14%–28% protein and low fat (2.6%–6.8% of total solids).[3] Formulas used for hand-raising rhinos are based on cows' milk or commercial formulas (ZooLogic Milk Matrix 20/14, PetAg, Inc., Hampshire, IL). Lactase (Lactaid) has been useful as a milk additive in these cases. Indian, white, and black rhino calves have been successfully hand-reared with these formulas.

In neonates that have not received colostrum, bovine or equine colostrum should be fed at a 50% dilution to provide immunoglobulins if rhino colostrum is unavailable. Addition of 10% colostrum to the formula for up to 1 month is recommended for local gastrointestinal (GI) immunity.[3] Feeding for the first 3 days is 10% of body weight (kg) divided into seven feedings, with an increase to 15%–20% on day 4 through 6 months of age. At 6 months through the start of weaning at 1 year, a constant volume of 11 kg of formula at each of three feedings should be offered. Calves should be weighed regularly and gain 1 to 2 kg/day.

RESTRAINT AND HANDLING

Physical Restraint

Chutes for rhinos may be simple free-stall designs, in which an individual animal is trained to enter voluntarily and poles or bollards allow protected contact. More sophisticated designs include hydraulic or movable walls, head restraints, and access doors for examinations and minor medical or reproduction-related procedures. Ideally, the design should be incorporated into the facility so that the animal has to pass through and it is not a dead-end. Successful use of these devices usually requires operant conditioning of the rhino, the use of tranquilization or sedation, or a combination.

Chemical Restraint

This section will focus on restraint of captive rhinos. Excellent sources of information on the immobilization of free-ranging rhinos are available.[5,35] The two techniques used are standing restraint and recumbent immobilization.

For captive rhinos, drug delivery usually requires use of darting equipment. Depending on the situation, pole syringes with robust needles or hand-injection in conditioned animals may be used. Most darting systems may be used in captive situations, as long as a robust needle (minimum 40–60 mm × 2 mm needle) is used to penetrate the thick skin and deliver the drug intramuscularly. Nylon darts (Teleinject, Daninject) are preferred in these situations, since they cause less trauma compared with metal darts. Ideal sites for drug injection are just caudal to the ear on the lateral cervical area, upper caudal hindlimb, shoulder, or nuchal hump in the white rhino. However, any site may be used if the dart is placed perpendicular to the skin and is adequate to penetrate muscle.

Depending on the animal's health status, the environment, and the procedure planned, food and water should be withheld prior to the procedure, at least overnight, although regurgitation and aspiration are infrequent complications in rhinos.

Procedures should be planned for the coolest time of day, preferably early mornings. Rapid induction and minimal excitement further decrease the risk of hyperthermia. Rectal enemas, evaporative cooling with sprayers and fans, or cold water baths for small individuals are warranted if the rectal temperature is greater than 39°C, although complete anesthetic reversal should be performed immediately if the temperature reaches 41°C or above.[5]

Rhinos are prone to developing myopathy and neuropathy after prolonged recumbency. Inflated truck inner tubes, heavy mats, or padding may be used under pressure points to prevent radial nerve paralysis and other neuropathies if the procedure is to take place on a hard surface.

The optimal body position is still being debated. Lateral recumbency is often preferred, since it provides optimal circulation to the limbs; however, sternal recumbency may allow better ventilation. If the animal needs to be in sternal recumbency for the procedure, it is ideal to roll the animal into lateral recumbency, whenever possible, to ensure adequate peripheral circulation. Limbs should be "pumped" about every 20 minutes to promote perfusion of muscles.[35]

It is imperative that immobilized rhinos be closely monitored to minimize complications. Hypoxia, hypercapnia, and hypertension are commonly associated with the use of potent opioids in rhinos.[22,33-] Accurate weight or estimated weight facilitates optimal drug calculation and prevents drug overdosing or underdosing and associated problems.

Pulse oximetry is useful for monitoring trends in hemoglobin oxygen saturation. Sites for placement include the ear pinnae (scraping of the skin surface may sometimes provide more accurate readings), mucosal folds of the prepuce, vulva, and rectum; and sensor pads placed side by side in the conjunctival sac, gingival mucosa, nasal mucosa, and inside the rectum, vagina, or prepuce. Ideally, readings should be greater than 90%, but interpretation must be made in conjunction with the color of the mucous membranes and blood and other clinical signs. Capnography may be used by placing a small-animal endotracheal tube inside a nostril in nonintubated rhinos. Direct and indirect blood pressure may be measured by using either an arterial catheter in the medial auricular artery or a blood pressure cuff at the base of the tail, respectively.

Standing sedation should only be attempted after proper consideration of animal and staff safety. In captive rhinos, standing chemical restraint has historically been performed using low doses of the potent opioid etorphine. However, variable responses may lead to recumbency. More recently, butorphanol, alone or in combination with azaperone or α_2-agonists (detomidine, medetomidine), has been successfully used. In the author's experience, a combination of etorphine (1 mg /1000 kg), butorphanol (10 mg butorphanol to 1 mg etorphine), and azaperone (20 mg standard dose, intramuscularly [IM]) has been effective in "walking" captive white rhino, with the use of a white flag as a target to bring the rhino from the holding facility to the crate. Once in the crate, the animal will remain standing and tolerate minor procedures. Chemical restraint may be partially to fully reversed with naltrexone (40–100 mg naltrexone:1 mg etorphine or 1–2.5 mg naltrexone:1 mg butorphanol) with or without atipamezole (5 mg atipamezole:1 mg α-$_2$-agonist), depending on the drug combination chosen (Box 55-1).[22,33,35]

Potent opioids are the primary drugs used for general anesthesia in rhinos. Etorphine is mostly commonly used, although carfentanil (in some species, this has been suggested to cause excitable inductions) and, more recently, combinations of etorphine and thiafentanil have also been administered to rhinos. Opioids are typically combined with azaperone, α_2-agonists, or acepromazine to decrease complications. Hyaluronidase may be included to increase absorption and decrease induction time. To deepen the anesthesia or for prolonged procedures, supplemental doses of etorphine, ketamine, propofol, or inhalant anesthetics have been used. Midazolam, diazepam, and guaifenisen infusion may provide additional muscle relaxation. Suggested doses for recumbent immobilization in captive rhinos are given in Box 55-2.[5,22,33–35,41]

White rhinos and, to a lesser extent, Indian rhinos appear to be more sensitive to the effects of opioids compared with black rhinos and exhibit muscle tremors, limb paddling, hypoxia, hypercapnia, and hypertension.[33,35] Butorphanol has been administered to antagonize respiratory depressive effects in white rhino (10–20 : 1 mg butorphanol to etorphine ratio); however, it may also lighten

BOX 55-1 Standing Chemical Restraint Doses for Adult Captive Rhinoceros[22,33,35]

Black Rhino
- 0.5–0.85 milligram (mg) etorphine, intramuscularly (IM)
- Doses as low as 0.25 mg used to walk rhino into crate
- 25–50 mg butorphanol, IM or intravenously (IV)
- 20–30 mg butorphanol + 20–50 mg detomidine, IM

White Rhino
- 0.8–1.5 mg etorphine, IM
- 50–70 mg butorphanol + 100 mg azaperone, IM
- 1.1 mg etorphine + 5 mg acepromazine + 15 mg detomidine + 15 mg butorphanol, IM
- 120–150 mg butorphanol + 5–7 mg medetomidine, IM (give 1–2 mg nalorphine, IV, to keep standing)

Indian Rhino
- 100 mg butorphanol + 100 mg azaperone, IM
- 0.5–1.5 mg etorphine, IM

Sumatran Rhino
- 25–40 mg butorphanol, IM
- 120–150 mg butorphanol + 5–7 mg medetomidine, IM (use 1–2 mg nalorphine, IV to keep standing)
- 0.8–1.5 mg etorphine, IM

BOX 55-2 Recumbent Immobilizing Doses for Adult Captive Rhinoceros[5,22,33–35,41]

Black Rhino
- 1.5–2 mg etorphine + 2–3 milligrams (mg) medetomidine, intramuscularly (IM)
- 2.5–3 mg etorphine + 20–60 mg azaperone, IM
- 1.5–2mg etorphine + 2–3 mg medetomidine + 1 gram per liter (g/L) ketamine in 5% guiafenisen drip
- 0.7–1.2 mg carfentanil ±10 mg midazolam
- 70–120 mg butorphanol + 100–160 mg azaperone, IM
- 120–150 mg butorphanol + 5–7 mg medetomidine, IM

White Rhino
- 1.2 mg carfentanil, IM
- 2–3 mg etorphine + 20–40 mg azaperone, IM
- 120–150 mg butorphanol + 5–7 mg medetomidine, IM ± 5% guaifenisen drip
- 70–120 mg butorphanol + 100–160 mg azaperone, IM

Indian Rhino
- 3.5–3.8 mg etorphine + 14 mg detomidine + 400 mg ketamine, IM
- 2.5 mg etorphine + 10 mg acepromazine, IM
- 0.7–1 mg carfentanil, IM
- 120 mg butorphanol + 80 mg detomidine, IM

Sumatran Rhino
- 30–50 mg butorphanol + 50–60 mg azaperone, IM
- 1 mg etorphine + 60 mg azaperone, IM
- 10 mg butorphanol + 10 mg detomidine IM, wait 20 min then 1.2 mg etorphine + 5 mg acepromazine, IM

the plane of anesthesia.[21] It should be used with caution in black rhinos, since they appear to be more sensitive and may suddenly get to their feet.[35] Other partial opioid agonist–antagonists are routinely used in the field and may be adapted for captive rhinos, when available (e.g., nalbuphine). Butorphanol–azaperone and butorphanol–medetomidine or detomidine combinations have successfully induced recumbency in captive Sumatran, Indian, and white rhinos.[34,35,41]

Oxygen supplementation by intratracheal intubation or nasal insufflation (flow rates of 15–30 liters per minute [L/min]) may increase oxygen saturation values.[24] Doxapram has been administered for apnea in rhinos but may only provide short-term relief. Partial or complete reversal should be considered in severe cases of hypoxia.

On some occasions other than medical procedures, rhinos may need to be sedated, as for crating and transport. For short-duration tranquilization or sedation, benzodiazepines (2–6 hours; adult doses: midazolam 5–50 mg, IM; diazepam 10–30 mg, IM) and azaperone (2–4 hours; 80–200 mg, IM) are useful choices in rhinos. Long-acting neuroleptics (LANs) are typically administered in free-ranging rhinos after capture for transport and boma acclimation, although they have also been used in captive rhinos for longer-duration tranquilization. Zuclopenthixol acetate (Clopixol-Acuphase) at doses of 60 to 200 mg lasts 3 days, and perphenazine enanthate (Trilafon-LA) does not take effect for 12 to 18 hours but lasts 7 to 10 days (100–200 mg, IM).[5]

SURGERY

Most surgical procedures involve the skin, eyes, digits, and horn, including treatments for corneal ulcers, cataracts, pododermatitis, wounds, and tumors. Because of the thickness and inelasticity of the rhino skin, suturing of wounds often results in dehiscence, so unless necessary, wounds are often left to heal by secondary intention. Use of wire sutures, stents, and mattress patterns may improve closure of the rhino skin. Surgical management of horn and integument problems, including tumors, may be achieved with the use of operant conditioning, standing sedation, or full immobilization.

Surgical treatment of ocular problems, including corneal ulcers and cataracts, is fairly common. Management of pododermatitis by surgical debridement has also become a more routine procedure, especially in Indian rhinos. Treatment of osteomyelitis in a black rhino, involving surgical debridement and vacuum-assisted closure, has been recently described.[16] Surgical repairs of rectal prolapse in black and Indian rhinos and patent urachus in a white rhino calf have been reported. Although most abdominal surgeries are unsuccessful, an adult white rhino has survived an exploratory celiotomy.[40] Laparoscopic techniques for rhinos are still in their developmental stages but have been successfully used for reproductive procedures such as uterine biopsy and oocyte retrieval.[18]

OTHER PHARMACEUTICALS

With the exception of studies on anesthetics and a few vaccines, no pharmacokinetic trials in rhinos have been performed. Most clinicians use the horse as the model to determine drug dosages, especially for antibiotics and antiparasitics. Commonly used antibiotics include oral trimethoprim–sulfadiazine equine formulations, parenteral large-animal cephalosporins, and oral fluoroquinolones. Antiparasitics are not routinely required in captive rhinos, but oral and injectable ivermectin; oral fenbendazole, pyrantel pamoate, and niclosamide; and pour-on acaricides, including flumethrin 0.5%, have been used in rhinos.[22] Nonsteroidal anti-inflammatories are frequently prescribed for analgesia.

PHYSICAL EXAMINATION AND DIAGNOSTICS

Rhinos should be trained to permit sample collection and clinical examination. Resting values for heart rate, respiratory rate, temperature, and other values have been obtained for nonrestrained black and white rhinos. The various species appear to be similar with regard to heart rates (30–40 beats per minute) and respiratory rates (6–12 breaths per minute). Rectal temperatures are typically 34.5°C to 37.5°C, although temperatures may be higher in anesthetized rhinos (37°C–39°C) because of exertion or muscle tremors.[22,35] Limited information on electrocardiography (ECG) values in rhinos is available.[19] Indirect blood pressure has been measured in unsedated black and white rhinos by using a human blood pressure cuff around the base of the tail. Mean values reported for unanesthetized white rhinos are 160 +/–2.9 millimeters of mercury (mm Hg; systolic), 104 +/–2.3 mm Hg (diastolic), and 124 +/–2.2 mm Hg (mean blood pressure).[6] In anesthetized animals, etorphine may cause hypertension, but some authors have observed more variable mean blood pressure values (107–280 mm Hg) depending on the drugs used and when measurements were taken.

Hematologic and biochemical parameters have been published (Tables 55-1 through 55-3).[14,22] Although most values may be interpreted as being similar to other perissodactyls, total protein and globulins tend to be higher and sodium and chloride lower than in domestic horses. Hypophosphatemia (low blood phosphorus) is a recognized problem in captive black rhinos, with levels dropping below 1 milligram per deciliter (mg/dL). Low serum phosphorus has been linked to hemolytic anemia and other blood disorder syndromes.[10,22] Supplemental doses of elemental phosphorus (preferably chelated) (10 to 24 g, orally [PO], once daily [SID]) are used in black rhinos until normal serum levels are reestablished, as reported anecdotally (Valdes E, personal communication). In critical cases, intravenous sodium or potassium phosphate may be administered at 14.5 millimoles per hour (mmol/hr), but serum calcium should be carefully monitored.

Venipuncture may be routinely performed on awake captive rhinos with the use of conditioning of the animal, restraint devices, or both. The most commonly used sites for blood collection are the auricular vein, radial vein (inside the forelimb crossing the carpus), and metacarpal vein (lower inside forelimb). The ventral tail (coccygeal) vein has also been used, particularly in Sumatran rhinos, and the blood collection technique is similar to that used in domestic cattle. Arterial access is available for blood gas sampling from the medial auricular artery. Rhino blood cells resemble those of domestic horses. Nucleated red blood cells (RBCs) and reticulocytes may be observed in anemic animals. Blood smears should be carefully screened for the presence of hemoparasites, especially in recently captured or imported animals.

Urinalysis panels in captive rhinos are similar to those for horses, with the large numbers of calcium carbonate crystals creating a normal milky yellow appearance of urine. Calcium oxalate, phosphate, and ammonium crystals may also occur, depending on the diet. Occasionally, dark discoloration of urine, which is associated with the pigmentation of certain browse species, may be mistaken for blood or myoglobin (e.g., ash, mulberry). Normal values for the different rhino species have been published and typically are pH of 8.0 to 8.7 and specific gravities of 1.010 to 1.030.[15]

Because of the rhino's size, cerebrospinal fluid (CSF) collection has not been successful except in a few rhino calves.[22] Therefore, extrapolation of normal values from domestic horses and other perissodactyls should be used for interpretation.

Incorporation of scales into rhino facilities has permitted monitoring of body weights and physical condition. Average ranges for adult body weights for each of the rhino species are as follows: black rhino 800–1350 kg, white rhino 1800–2200 kg, Indian rhino 1800–2200 kg, and Sumatran rhino 600–800 kg.[22]

Radiography of the distal extremities, skull, horn, and, in calves, thorax has become more routine and useful with digital technology. Thermography is also available in many institutions. Localization of inflammation associated with acute lameness, laminitis, abscesses, and changes in skin temperature associated with dermatologic conditions may all be detected by using this tool. Fluoroscopy has also been used for skull and horn imaging to

TABLE 55-1

Mean Hematology Values in Rhinos (±SD)[14]

Parameter	Black Rhino	White Rhino	Indian Rhino	Sumatran Rhino
White blood cell (WBC) × 10^3/microliter (μL)	8.42 (2.48)	9.30 (2.46)	7.20 (1.33)	8.27 (1.55)
Red blood cell (RBC) ×10^6/μL	4.01 (0.88)	5.77 (1.28)	6.43 (0.86)	5.32 (1.09)
Hemoglobin, gram per deciliter (g/dL)	12.0 (2.0)	13.8 (3.8)	13.4 (1.5)	12.4 (1.6)
Hematocrit %	33.4 (5.7)	36.9 (9.3)	37.0 (4.6)	36.9 (4.2)
Mean corpuscular volume, (fL)	85.7 (9.0)	63.8 (7.8)	57.8 (4.9)	71.5 (11.2)
Mean corpuscular hemoglobin, picogram per cell (pg/cell)	30.5 (3.3)	23.5 (1.9)	21.3 (3.0)	23.9 (3.8)
Mean corpuscular hemoglobin concentration (g/dL)	35.7 (2.7)	37.9 (7.3)	36.3 (3.2)	33.5 (2.1)
Platelets ×10^3/μL	284 (83)	378 (103)	178 (53)	198 (135)
Nucleated RBC/100 WBC	0	1 (1)	0	—
Reticulocytes %	1.6 (2.9)	—	—	—
Neutrophils ×10^3/milliliter (mL)	5.24 (2.18)	5.42 (2.05)	5.13 (1.24)	4.86 (1.16)
Lymphocytes ×10^3/mL	2.48 (1.1)	2.35 (1.15)	1.74 (0.67)	2.52 (0.90)
Monocytes ×10^3/mL	0.43 (0.32)	0.65 (0.55)	0.22 (0.15)	0.36 (0.22)
Eosinophils ×10^3/mL	0.25 (0.22)	0.54 (0.59)	0.32 (0.31)	0.37 (0.21)
Basophils ×10^3/mL	0.17 (0.09)	0.10 (0.05)	0.13 (0.05)	0.08 (0.01)
Neutrophilic bands ×10^3/mL	0.27 (0.35)	0.71 (1.18)	0.22 (0.20)	0.31 (0.24)

TABLE 55-2

Mean Blood Chemistry Values in Rhinos (±SD)[14]

Parameter	Black Rhino	White Rhino	Indian Rhino	Sumatran Rhino
BUN (mg/dL)	13 (3)	16 (3)	3 (2)	6 (2)
Creatinine (mg/dL)	1.1 (0.2)	1.8 (0.4)	1.3 (0.2)	0.9 (0.1)
Uric acid (mg/dL)	0.5 (0.2)	0.9 (0.8)	0.3 (0.2)	—
Bilirubin (mg/dL)	0.3 (0.1)	0.3 (0.3)	0.4 (0.3)	0.2 (0.1)
Glucose (mg/dL)	69 (21)	97 (39)	82 (25)	76 (13)
Cholesterol (mg/dL)	102 (37)	93 (26)	53 (21)	48 (21)
CPK (IU/L)	255 (248)	409 (722)	260 (203)	617 (398)
LDH (IU/L)	595 (427)	537 (320)	267 (149)	231 (38)
Alkaline Phosphatase (IU/L)	80 (55)	92 (51)	80 (41)	17 (6)
ALT (IU/L)	16 (7)	16 (9)	7 (7)	6 (3)
AST (IU/L)	85 (27)	71 (25)	61 (27)	39 (9)
GGT (IU/L)	27 (18)	19 (14)	18 (16)	6 (2)
Total protein (g/dL)	7.6 (0.9)	8.5 (1.0)	7.5 (0.9)	7.5 (0.4)
Globulin (g/dL) (electrophoresis)	4.9 (0.9)	5.3 (0.8)	4.5 (0.7)	3.8 (0.7)
Albumin (g/dL) (electrophoresis)	2.6 (0.4)	3.2 (0.5)	2.9 (0.5)	3.6 (0.6)
Fibrinogen (mg/dL)	104 (195)	101 (241)	350 (84)	324 (85)

ALT, Alanine aminotransferase; *AST*, aspartate aminotransferase; *BUN*, blood urea nitrogen; *CPK*, creatine phosphokinase; *GGT*, gamma-glutamyltransferase; *g/dL*, gram per deciliter; *IU/L*, international unit per liter; *LDH*, lactate dehydrogenase; *mg/dL*, milligram per deciliter.

detect dental, jaw, and horn problems, including fractures and neoplasia.

DISEASES

Infectious Diseases

Mycobacterium bovis and *M. tuberculosis* have caused infections in captive rhinos.[22] Although not currently reported in free-ranging rhinos, *M. bovis* infection was recently reported in a black rhino brought into captivity in South Africa.[12] Initial infection may be asymptomatic or result in progressive weight loss and emaciation, with coughing and dyspnea occurring in the terminal stages. Nasal discharge may be present but is inconsistent. Most infections are pulmonary. Antemortem testing includes intradermal tuberculin test, tracheal lavage, gastric lavage, or both for mycobacterial culture, and serologic tests. Retrospective analyses of sera from *M. tuberculosis*-infected black rhinos, with the use of ElephantTB Stat-Pak (Chembio Diagnostic Systems Inc., Medford, NY), have shown positive results.[11] Treatment with isoniazid, rifampin, ethambutol, and pyrazidamide has been attempted. However, assessment of a successful response is limited.

Mycobacterium avium subspecies *paratuberculosis* has recently been isolated from a wild-caught black rhino with diarrhea and

TABLE 55-3

Mean Serum Mineral Values and Blood Gases in Rhinos (±SD)[14]

Parameter	Black Rhino	White Rhino	Indian Rhino	Sumatran Rhino
Calcium (mg/dL)	12.7 (1.0)	11.8 (0.9)	11.4 (0.8)	13.3 (1.1)
Phosphorus (mg/dL)	4.8 (1.1)	4.0 (0.9)	4.0 (0.9)	3.7 (0.7)
Sodium (mEq/L)	133 (3)	134 (5)	132 (3)	133 (4)
Potassium (mEq/L)	4.7 (0.6)	4.7 (0.8)	4.1 (0.4)	4.6 (0.6)
Chloride (mEq/L)	96 (0.3)	95 (4)	91 (3)	100 (3)
Bicarbonate mEq/L	23.3 (4.2)	18 (0)	27.0 (0)	—
Carbon dioxide (mEq/L)	25.4 (9.9)	25.3 (8.8)	27.3 (3.7)	22.8 (2.4)
Iron (µg/dL)	227 (66)	176 (67)	152 (70)	—
Magnesium (mg/dL)	3.34 (3.45)	118.2 (232.5)	7.95 (8.56)	—

mEq/L, Milliequivalent per liter; *mg/dL*, milligram per deciliter; *µg/dL*, microgram per deciliter.

weight loss. After a course of antimycobacterial drugs, clinical signs resolved, and fecal cultures were negative.[4]

Salmonella infection may cause enteritis and fatal septicemia in captive and newly captured wild rhinos. In a retrospective survey of captive black, white, and Indian rhinos in the United States, 11% demonstrated positive cultures, usually associated with clinical signs. Research has shown that asymptomatic black rhinos may carry and intermittently shed *Salmonella* in their feces.[23] Clinical infection may occur secondary to transport, changes in diet, immobilization, concurrent disease, or exposure to a large number of organisms. Lethargy, anorexia, signs of colic, diarrhea, and death may be observed. Successful treatment using trimethoprim–sulfamethoxazole and supportive care is possible if initiated early. However, treatment of asymptomatic animals is not recommended.

Leptospirosis usually presents with depression and anorexia. Other signs may include hemolytic anemia (not present in all cases), hemoglobinuria, colic, and development of skin ulcers. Abortion has also been linked to infection with leptospirosis in an Indian rhino.[22] Fatality rates are high in clinically affected black rhinos, although successful treatment with trimethoprim–sulfamethaxozole and ceftiofur has been reported.[29] Diagnosis is based on high antibody titers (microagglutination test [MAT]) and confirmed by detection of leptospiral organisms in urine or tissues (through immunofluorescent antibody [IFA] test or, more recently, polymerase chain reaction [PCR] test). Low levels of antibodies have been observed in free-ranging and nonvaccinated black rhinos without evidence of disease. Preventive measures include annual vaccination of black and possibly Indian rhinos with a multivalent large animal product, rodent and wildlife control programs, and good husbandry to minimize contamination of feed and water.

Fatal gastroenteritis caused by *Clostridium* infection has occurred in adult white rhinos. Neosporosis has also been documented in a white rhino calf.[42] Colitis of unknown etiology and secondary endotoxic shock led to the death of an adult black rhino. *Escherichia coli*, *Campylobacter coli*, and *Pseudomonas* sp. have caused enteritis in hand-reared rhinos.

Encephalomyocarditis virus (EMCV) infection usually results in acute death from myocarditis. Diagnosis is usually based on isolation of the virus from heart, spleen, or other tissues at necropsy. Prevention should target rodent control in endemic areas.

Fungal pneumonia is usually caused by *Aspergillus* sp. and is primarily observed in black rhinos that have had concurrent disease and broad-spectrum antibiotic or corticosteroid therapy. Clinical signs may include weakness, weight loss, epistaxis, or other signs consistent with pneumonia. Diagnosis is challenging, although serology and bronchoscopy, with cytology and fungal culture, may be useful. Long-term treatment with antifungal drugs (e.g., itraconazole) is expensive and has unknown efficacy.

Death caused by anthrax has been observed in wild rhinos and has been implicated in a die-off of several Javan rhinos in the Ujon Kulan National Park. Most cases result in sudden death. Foamy discharge from the mouth and nostrils may be seen and can appear similar to EMCV infection. Diagnosis is based on identification of anthrax bacilli in blood or tissue smears. Vaccination of ranched rhinos has been used in some endemic areas of Africa, since rhinos have been infrequently affected during outbreaks. Black rhino in the Etosha National Park, Namibia, have been vaccinated for more than 30 years.[39]

Isolated cases of rabies and tetanus have been documented in rhinos.[22] Antibodies to West Nile virus (WNV) have been detected in black, white, and Indian rhinos in endemic areas of the United States. Black rhinos and Indian rhinos have been vaccinated with a commercial equine product without any apparent adverse effects. In the author's limited experience, black rhinos appear to develop some humoral response to vaccination, although in one study, Indian rhinos did not develop significant titers. Infections with eastern equine encephalitis virus (EEEV), western equine encephalitis virus (WEEV), or Venezuelan equine encephalitis virus (VEEV) have not been reported in any of the rhino species.

Antibodies to African horse sickness, Akabane, bluetongue, epizootic hemorrhagic disease of deer (EHD), Wesselbron, and Rift Valley fever viruses have been detected during serosurveys of free-ranging African rhinos, although no association with disease has been established.[20,22]

Parasitic Diseases of Rhinoceroses

Internal Parasites

A variety of parasites, including nematodes (*Chabertia*, *Necator*, *Bunostomum* spp.), trematodes (*Paramphistomum* sp.), cestodes (*Anoplocephala* sp., hydatid cyst), and protozoa (*Balantidium coli*), have been found in wild-caught Indian rhinos. *Balantidium* has also been reported in white rhino.[31] Larvae of *Gyrostigma* sp., the rhinoceros bot fly, are commonly found in the stomachs of free-ranging black rhinos. Over 40 species of helminths have been identified in African rhinos, the majority being nematodes. Although most are asymptomatic, some such as *Diceronema versterae* may cause tumorlike lesions in the stomach. The most abundant species is a small pinworm, *Probstmayria*, found in the cecum and colon of black rhinos.[31] Trematodes and cestodes (*Anoplocephala* sp.) are also found in African rhinos. Tapeworms, coccidia, and, occasionally, strongyles are found in captive rhinos, although they are rarely a cause of disease.

Tsetse flies transmit trypanosomes that may lead to serious consequences, such as fatal infection or abortion, in naïve black and white rhinos that are translocated from a tsetse-free area.[31] Hemoparasites (*Babesia bicornis*, *Theileria bicornis*, *Theileria equi*) have been observed in free-ranging African rhinos. *T. equi* is relatively common in white rhinos, but neither *Theileria* sp. appear to cause disease. However, *B. bicornis* and *T. bicornis* have been associated with increased mortality in black rhinos stressed by capture and transport.[30]

External Parasites

Skin ulcers in free-ranging black rhinos have been associated with a filarial parasite, *Stephanofilaria dinniki*, presumed to be transmitted

by a blood-sucking arthropod. Recently, an outbreak of filariasis in both white and black rhinos occurred in the Meru National Park, Kenya, and lesions resolved after treatment with ivermectin and long-acting amoxicillin.[26]

In free-ranging African rhinos, tick infestation is common and includes *Rhipicephalus*, *Dermacentor*, *Ambylomma*, and *Hyalomma* species. Since these ticks are vectors for a number of diseases, treatment of recently captured or newly imported rhinos with acaricides is usually required for movement.

Noninfectious Diseases

Injuries such as skin lacerations, punctures, abrasions, and other wounds are common in both captive and free-ranging rhinos. Common sense application of wound treatment principles apply, although the thick skin of the rhino does not lend itself to primary closure, and abscesses have a tendency to undermine the integument along fascial planes.

Horn avulsion, cracks, or other trauma may be self-induced as a result of acute or chronic rubbing, damage from the enclosure, or intraspecific fighting. Laminitis and neoplasia may also affect the horn. Treatment may involve debridement, antibiotics, wound treatment, and fly control. Dermatitis, especially in Indian and Sumatran rhinos, may be caused by inadequate access to wallows and pools. Exfoliative dermatitis has been reported in a captive white rhino. *Malassezia pachydermatis* and *Candida parapsilosis* were identified in black rhino with dermatitis and successfully treated using natamycin solution.

Neoplasia is relatively uncommon in rhinos. Cases of squamous cell carcinoma (various locations) in white, black, and Indian rhinos, cutaneous melanoma in black and Indian rhinos, and isolated cases of thyroid carcinoma, hepatocellular carcinoma, and acute lymphoblastic leukemia in black rhinos have been reported.

Although all species of rhino are susceptible to developing pododermatitis caused by inappropriate substrate, long-term indoor housing in northern climes, limited access to a pool, or other husbandry conditions in captivity, the Indian rhino appears to be more susceptible to chronic foot problems.[1] Management of the condition includes improvements in husbandry and in medical and surgical interventions. Medical treatment may be in the form of oral antimicrobial medication and topical use of copper sulfate and oxytetracycline. Regular hoof trimming and surgical debridement of necrotic lesions, along with use of collagen products for granulation tissue stimulation, may lead to improvement in appearance and comfort of the animal. In addition to the factors mentioned above, nutritional imbalances (e.g., zinc) have also been investigated. Black rhinos develop laminitis that may or may not be related to idiopathic hemorrhagic vasculopathy syndrome (IHVS). Frequent foot trimmings, analgesics, and antibiotics are needed to manage this condition, as with laminitis in domestic horses.

Corneal trauma and secondary infection may result in corneal ulceration and perforation. Corneal ulcers may become severe in Indian rhinos. Surgical management of a melting corneal ulcer with the use of a conjunctival graft has been described in one case. Exposure keratitis is a relatively common problem for captive Sumatran rhinos. Medical and surgical treatments are similar to those for equine corneal conditions.

Noninfectious causes of GI problems in rhinos may be a result of dental problems, dietary changes, dehydration, ingestion of foreign material (i.e., sand), changes in GI motility because of inflammation, neoplasia, or inadequate dietary fiber. Gastric ulcers have been observed on gastroscopy and at necropsy in rhinos that have received long-term nonsteroidal anti-inflammatory therapy, have concurrent disease, or are undergoing a stressful condition. Gastroprotectants such as omeprazole, histamine 2 (H_2)-blockers, or sucralfate have been used in rhinos both prophylactically and therapeutically, although treatment depends on a specific diagnosis.

Since 2001, chronic glomerulonephritis, renal failure, or both have been recognized as a contributing cause of death in at least seven black rhinos. Significant changes in blood urea nitrogen (BUN) and creatinine values or in urinalysis were not always evident in these cases, making diagnosis difficult until necropsy. In contrast, two white rhinos (aged >40 years) developed progressive chronic renal failure characterized by uremia, isosthenuria, and hypercalcemia. Nutritional management with the use of a high-energy, low-protein feed (Equine Senior, Purina Mills, St. Louis, MO) appeared to stabilize both animals (Ferrell S, Radcliffe R, personal communication).

Toxicities

Seven fatalities occurred among a group of 20 black rhinos captured and housed in bomas constructed with creosote-treated wood in Zimbabwe. The presumptive cause of death was liver dysfunction caused by creosote toxicosis. Exposure of all rhino species to creosote-treated housing materials should be avoided. Free-ranging white rhinos have succumbed to blue-green algae toxicity (*Microcystis* spp.). Periodic algal blooms occur in water bodies with high organic matter and low water levels following dry summers and abnormally warm falls in the Kruger National Park.

Three black rhinos died from suspected vitamin D toxicosis caused by an accidental incorporation of high levels of vitamin D in the commercial black rhino pellet.[13] Caution should be used when treating with high dosages of cocciodiostats such as salinomycin, which have been reportedly toxic to rhinos.[22] An apparent adverse drug reaction to firocoxib in a white rhino resulted in a generalized vesiculobullous dermatitis.[37] Food items to be avoided in rhino diets include kale, onions, red maple (*Acer rubrum*), and members of *Brassica* plants because of their predisposition to cause hemolysis.[22]

Diseases of Unknown Etiology in Black Rhinos

Skin disease may be the most common health problem in black rhinos, with over 50% having at least one episode during their lifetime. Several distinct syndromes have been reported in captive rhinos.

Superficial necrolytic dermatopathy has also been called *ulcerative skin disease*, *vesicular* or *ulcerative dermatopathy*, and *mucosal* or *cutaneous ulcerative syndrome*.[25] Initial signs are epidermal plaques or vesicles that progress to ulcers, often over the pressure points, ear margins, coronary bands, and tail tip. Oral or nasal ulcers may develop concurrently. The affected rhinos may also be anorexic, depressed, and lame, have oral or nasal bleeding, and lose weight. These animals may have decreased albumin and hematocrits. In most cases, the skin lesions are associated with other concurrent health issues, including GI and respiratory diseases. Management includes symptomatic treatment. If the lesions become extensive, the condition may be fatal. Treatment with cryotherapy and steroids has been successful. Lesions may resolve spontaneously.

Eosinophilic granuloma syndrome usually presents with oral and nasal nonhealing ulcers and granulomas, which may lead to epistaxis or oral bleeding.[32] Cytology shows a predominance of eosinophils associated with lysis of collagen and mineralization. Although the lesions may resolve spontaneously, usually over 1 to 7 months, they may recur. Treatment has included the administration of corticosteroids, local cryotherapy, or laser therapy. Eosinophilic granulomas in wild rhinos are typically associated with *Stephanofilaria dinniki*.

A rare single case of primary vitiligo has been reported in a black rhino. The condition began around the nares at 2 years of age and progressed to include multiple areas without any evidence of other syndromes.[38]

All rhino species appear to accumulate dental tartar in captivity, especially if they are not given access to hard or coarse food items. However, black rhinos appear to develop severe proliferative gingivitis, not always directly associated with the degree of accumulation of calculus.[2,39] Like horses, rhinos also develop dental points that may eventually create clinical problems with prehension as they age and require periodic dental floats.

Cases of hemolytic anemia in the captive black rhino population appear to be decreasing since the peak occurrence in the 1990s. Of

the 47 known occurrences, a high mortality rate (75%) was observed among the 39 animals that had been affected.[6] Possible etiologies include a hereditary deficiency of glucose-6-phosphate dehydrogenase (G-6-PD) leading to decreased RBC adenosine triphosphate (ATP) levels, hypophosphatemia, and hypovitaminosis E.[22] Leptospirosis is a known cause of hemolytic anemia in this species but was not associated with all cases. Management of this syndrome includes intravenous or oral supplementation of phosphorus, supplementation of vitamin E, prophylactic antibiotics for secondary infections, and whole blood transfusion in severe cases.

IHVS in black rhinos is characterized by severe limb, facial, and neck swelling associated with a nonhemolytic anemia. Additional signs include lethargy, respiratory stridor, laminitis and nail sloughing, aural hematomas (swelling of the ears), and oral or skin ulcers. The syndrome presents acutely without any known cause, and recurrent episodes are likely. The fatality rate is high, but a number of animals have recovered with antibiotic and nonsteroidal anti-inflammatory therapy with fatty acid and phosphorus supplementation, and topical treatment of lesions. Between 1995 and 2007, 13 black rhinos were documented to have been affected, often with recurrent episodes. The majority of cases occurred during the cooler months (October–March) in animals that lived in Texas or the southern United States. It has been proposed that the syndrome is an immune-mediated vasculitis, similar to equine purpura hemorrhagica. A possible association with *Streptococcus* sp. infection has been observed. Treatment is supportive.

Iron storage disorder (ISD) results in significantly higher tissue iron and serum ferritin levels in captive black and Sumatran rhinos than those measured in wild or recently captured animals. Levels appear to increase with time in captivity. In contrast, values for ferritin and tissue iron do not appear to be elevated in captive white or Indian rhinos.[8,22] Although hemolytic anemia, vitamin E deficiency, and hereditary disorders have all been proposed as potential causes, it is now believed to be related to dietary factors in the captive browser species. Hemosiderosis (tissue iron accumulation) is a common finding in multiple organs in black rhinos, although inflammation and lesions associated with these changes are infrequently observed (hemochromatosis). A recent fatality in a captive Sumatran rhino was associated with multi-organ hemochromatosis. Recommendations to minimize accumulation and reduce iron load include low iron diets, provision of browse, therapeutic phlebotomy (regular large-volume blood collection), and treatment with iron-chelating agents in those individuals with suspected clinical disease.

Leukoencephalomalacia, a severe neurologic disease caused by necrosis of the cerebrum, was diagnosed in four female black rhino calves; three became comatose within 1 to 7 days of signs and eventually died. Some investigators hypothesize that the cause may be related to dam age (mean 17.3 years) or excessive maternal iron.[22] Research has focused on possible congenital or hereditary causes of encephalomalacia. No known treatment exists.

REPRODUCTION

Female Anatomy and Reproduction

Specific reproductive parameters are provided in Table 55-4.[18,22] The female reproductive anatomy is similar in all species, with a bicornuate uterus, which has a short body and long tubular horns. Ovaries are found caudal to the kidneys but typically cranial to the last rib. The cervix is a thick, fibrous structure containing a complex system of folds. Wrinkling of the vagina may make visualization of the cervical os difficult. A hymen may be present in nonbred females. Two mammary glands are positioned in the inguinal region.

Estrus cycles are variable among the species, with the longest occurring in the Indian rhino at 43 to 48 days, although the white rhino has been reported to have cycle lengths of 31 to 35 or 66 to 70, which may be caused by fetal resorption.[18] Ovarian activity and pregnancy may be monitored with transrectal ultrasonography and fecal, urinary, or serum hormone assays. Because of the risk of fetal resorption, early pregnancy should be confirmed starting at 2 weeks with ultrasonography. Embryonic vesicles may be visualized as early as 15 days following ovulation. Late pregnancy may be monitored with transabdominal ultrasonography of the flank region. Early embryonic loss has been documented in wild and captive black, white, and Sumatran rhinos.[18] Black and Sumatran rhinos have been successfully treated for early fetal resorption with oral progestin.

Although dystocia is relatively uncommon in rhinos, it does occur in all captive species.[18] If dystocia occurs, oxytocin (100 IU) may be effective if no signs of labor are observed for 4 to 6 hours after rupture of fetal membranes. Otherwise a fetotomy is indicated and has been successfully performed. Cesarean section is not an option in rhinos.

Signs of impending birth include increase in teat size and mild vaginal prolapse up to 30 days prior to calving. Dramatic changes in mammary size, restlessness, change in appetite, and relaxation of

TABLE 55-4

Reproductive Parameters in Rhinoceros[18,22]

	Black Rhino	White Rhino	Indian Rhino	Sumatran Rhino
Age at Puberty*	F: 4–7 years M: 7–10 years	F: 6–7 years M: 10–12 years	F: 5–7 years M: 10 years M	F&M: 7–8 years
Estrus cycle	21–27 days	31–35 or 66–70 days	43–48 days	21–25 days
Gestation length	465–475 days	485–518 days	462–489 days	475–510 days
Intercalving interval	2.5–4 years	2–3 years	3 years	3–4 years
Estrus determination†	Ultrasonography – ovulatory follicle (5.0 cm)	Ultrasonography – ovulatory follicle (3.2–3.5 cm)	Ultrasonography – ovulatory follicle (10–12 cm)	Ultrasonography – ovulatory follicle (2–2.5 cm)
Pregnancy diagnosis	Urine: PdG, second half of gestation, pregnanetriols Fecal: 20 α-protagestagen and 20 keto-protagestagen after 60 days; PdG last half of gestation	Urine: PdG Fecal: pregnanetriols, progesterone	Urine: PdG after 3 months	Fecal: progesterone

*In captivity, onset of puberty may be earlier; 3.5–5.5 years in females and 5.5–9 years in males.
†Specific urinary, fecal, and serum hormone assays have been developed to detect pregnancy in each rhino species.
PdG, Pregnanediol-glucuronide.

pelvic ligaments may be observed within 48 hours of birth. Calving should occur rapidly (1–3 hours) with the placenta passed within 6 to 7 hours.[18]

Reproductive system problems are relatively common in captive rhinos. Disorders occur with greater frequency in nulliparous females and increase with age.[18] Ovarian and paraovarian cysts have been diagnosed with ultrasonography in white rhinos. Black and white rhinos typically have cystic hyperplasia of the uterus, and Asian species have neoplastic changes. Leiomyomas are the most common tumor found in these species, although adenoma and adenocarcinoma have also been reported in white rhinos.

Assisted reproduction has advanced rapidly in rhinos. The ability to induce ovulation, time artificial insemination (AI), and use both fresh and cryopreserved semen has resulted in the conception of seven rhinos by AI, with five live births.[18] With the use of in vitro fertilization technology, the first rhino embryo has been produced.[17]

Male Anatomy and Reproduction

Testes are located along the dorsal preputial fold either adjacent to the inguinal rings or with the caudal aspect protruding between the rear legs and positioned horizontally in the body. The scrotum is more obvious in the Asian species. Accessory sex glands in male rhinos are similar in all four species and include vesicular glands, bulbourethral glands, and a prostate.[18] The penis is curved backward unless erect which allows urination for territorial marking. Lateral projections are found on the glans penis. Semen collection attempts in rhinos have been historically inconsistent, although development of a rhino-specific probe has resulted in more reliable results by electroejaculation.[36] Rhino semen characteristics for all four species have been published.[18] Techniques for cryopreservation of semen from white, Sumatran, and Indian rhinos have been successfully developed and used for AI.

The penis may be traumatized by injuries from mating or masturbation. Testicular fibrosis is a common finding in older males and may be detected by ultrasonography as hyperechoic spots, although it typically does not cause changes in semen quality. Testicular neoplasia, typically seminoma, has been observed in black and white rhinos and may be diagnosed with ultrasonography and biopsy. Hemicastration may be curative. Epididymal cysts (Sumatran and white rhinos), hematoma, or seroma may also be differentiated with ultrasonography and fine-needle aspiration. These conditions may have variable effects on sperm production.

PREVENTIVE MEDICINE

Annual vaccination for leptospirosis is recommended in black rhinos and possibly Indian rhinos. The animal should be observed for 30 minutes following vaccination because isolated cases of anaphylatic -like reactions have been reported. Vaccination for rabies, tetanus, arboviruses (EEEV/WEEV/WNV) may be considered if the area is considered endemic or increased risk factors are involved. Prior to vaccination, serologic screening for leptospirosis (multiple serovars), and WNV is recommended. Other vaccination regimens depend on regional requirements and exposure risks (consider multivalent vaccination for clostridial diseases).

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