CHAPTER

48

RATITES

James Stewart

Ratites are a group of large, ground-dwelling birds that share the common characteristic of flightlessness. The term ratite is derived from the Latin *ratitus*, meaning raft, and refers to the shape of the sternum that lacks a keel. There is no scientific classification "ratite," but the term is used to collectively describe the ostrich (from Africa), rhea (from South America), emu (from Australia), cassowary (from Australia and the New Guinea archipelago) and the kiwi (from New Zealand).[1] Once considered to be a single category of related primitive birds, the ratites are now considered to be derived from unrelated groups of flighted birds that have adapted to a highly specialized terrestrial lifestyle.

In the 1980's, it was estimated that between 90,000 to 120,000 ostriches existed on approximately 400 farms as part of a multi-crop rotation system in South Africa. During the same time period, a fledgling industry arose in the United States and by the end of the decade, it was estimated that there were close to 10,000 ostriches and about 3,000 emus in the US.[55]

This growing interest in breeding ostriches and emus has occurred due to the birds' potential as producers of meat, feathers and skin. Ostrich skin is characterized by prominent feather follicles and is a high status product of the leather industry. Ostriches yield a red meat that has the flavor and texture of beef, yet has the cholesterol and nutritional value of poultry. Ostrich feathers, still used for dusters, are also used in the manufacture of fashion and theatrical attire.

Characteristics

Ostrich

The ostrich is the largest living bird, reaching over 200 kg in weight and 2.7 m in height. The ostrich that was first described (nominate race) was put in the family Struthionidae and the genus *Struthio*. The bird's dome-backed profile, desert living conditions and odd-shaped feet are reminiscent of the camel, thus the species name, *camelus*. Four subspecies are currently recognized; their range is through the northern, eastern and southern savannahs of the African continent. The Syrian ostrich of the Arabian peninsula became extinct in 1941.

Struthio camelus camelus (North African ostrich) ranges from Morocco to Ethiopia and Uganda. A male *S. c. camelus* has a red coloration of the skin of the neck and thighs and a bald crown of the head. The hens of all races are indistinguishable. The eggs can be differentiated among subspecies, and those of the nominate species are characterized by fine stellate pores.

S. c. massaicus (East African or Masai ostrich) inhabits Kenya and Tanzania. The male has a red neck and thigh and the crown of the head is feathered.

S. c. molybdophanes (Somali ostrich) is found in Ethiopia and Somalia. The male has a blue-gray neck and thighs and the crown of the head is bald.

S. c. australis (South African ostrich) ranges throughout southern Africa. The neck and thighs of the male are gray and the head is feathered.[5] The large pores in the shells of this subspecies leave the egg with a pitted appearance.

A hybridized ostrich that is a combination of *S. c. australis, S. c. massaicus* and *S. c. syriacus* is referred to as a "domestic" or "African black" ostrich. The careful breeding of this bird since the 1870's has resulted in a smaller, calmer bird that has higher quality feathers than its free-ranging relatives.[55]

Ostriches can run up to 40 mph for several miles and can kick forward with powerful and accurate blows. Ostriches have large eyes with substantial visual acuity, and large ear canals with a keen sense of hearing. These birds have been known to breed for up to 42 years, and may live over 80 years. The male has black and white plumage at maturity, while the females and juveniles have dull brown plumage.

Rhea

Rheas are frequently referred to as South American ostriches, but are related only in superficial appearance. Weighing 25 kg and standing 1.5 m in height, the rhea is the largest bird of the western hemisphere. Two distinct species are recognized. The Common Rhea ranges throughout the central continent, and as many as five subspecies have been described. Darwin's Rhea, including three subspecies, is a smaller bird residing in the eastern foothills of the Andes. Free-ranging rheas eat grass, leaves and insects.

Cassowary

Cassowaries are stout, heavy-bodied birds reaching 85 kg in weight and 1.5 m in height. They are distinguished by a large bony casque on the forehead that is used to deflect brush as they dart through their dense rain forest habitat. Three species are found on New Guinea; one also ranges in northern Australia. Cassowaries are solitary, the males are highly aggressive and these birds adapt poorly to most captive settings.

Emu

Emus are closely related to cassowaries and share the same type of feather. The shaft and aftershaft are equally well developed, giving each feather the appearance of being doubled. The single existing species is found throughout Australia, while the Tasmanian and Kangaroo Island emus became extinct only in the past century. Adult emus may weigh 55 kg and stand 1.7 m in height. Emus may live 30 years and have strong eyesight and hearing. Like ostriches, emus are good swimmers. Free-ranging emus consume grasses, herbs, fruits and insects.

Other Ratites

Included among the ratites is the kiwi, and frequently the tinamous. Both groups are specialized ground-dwelling birds that bear little resemblance and no relationship to the larger species. This chapter will focus primarily on the ostrich as the model ratite, with only occasional comment on the emu, rhea and cassowary.

Clinical Anatomy and Physiology

Despite their impressive size, ratites are, nonetheless, birds, and their anatomy and physiology are fundamentally similar to a psittacine model. The deviations from this model, including their size, are all adaptations to a terrestrial lifestyle. The similarities and differences are clinically relevant and should be familiar to the ratite veterinarian.[2,11,19]

Integument

The skin of ratites is thick along the legs and body, but relatively thin along the neck where it is subject to tears (Figure 48.1). The ostrich, emu and rhea have sternal callosities. In addition, the ostrich has a callosity distal to the pubic bone and another distal to the hock joint; both anatomic areas contact the ground in a recumbent bird. Apteria are present along the lateral body wall and provide convenient access sites for surgery and diagnostic procedures such as ultrasound. In contrast to other birds, the feathers of the ostrich function to shade the body, rather than insulate it, and an ostrich will erect the feathers when hot and flatten them when cold. Ostriches have no feathers on the thigh, while in other ratites feathers extend to the tarsometatarsus. Ostrich, emu and rhea feathers lack barbiceli, making their feathers less water-resistant than those of other birds (Color 48.14).

Musculoskeletal System

The rhea, emu and cassowary have three toes (digits 2, 3 and 4), each with four phalanges. The ostrich is the most specialized runner and has only two toes (digits 3 and 4); the metatarsal-phalangeal joint is suspended so that the standing weight is born entirely by the digits (Figure 48.2). The pubic bones of the ostrich form a solid ventral symphysis to support the weight of the abdomen (Figure 48.3).

Corresponding with flightlessness, the pectoral musculature is greatly reduced and the sternum lacks a keel. Because there is no need for flight, the thoracic girdle is modified, and the fused scapula, coracoid and clavicle are attached to the cranial sternum (Figure 48.3). The patella is absent in ratites. In the

FIG 48.1 Ratite skin is thin and easily torn by sharp projections on fences and transporting vehicles. The trachea and jugular vein are visible through the tear in the skin (courtesy of James Stewart).

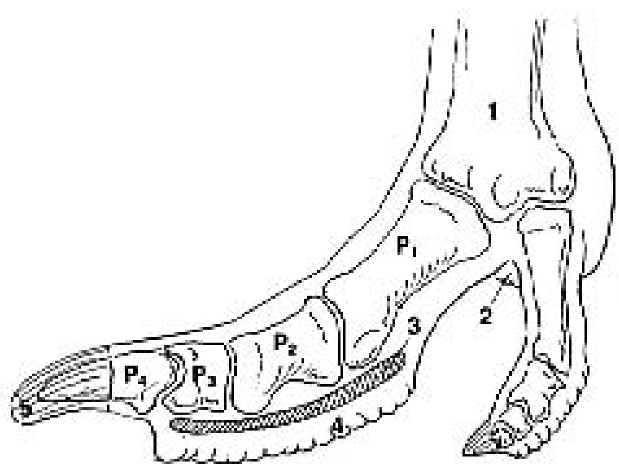


FIG 48.2 The ostrich foot is composed of two toes. 1) tarsometatarsus 2) metatarsal phalangeal pad 3) digital cushion 4) phalangeal pad 5) toenail (modified with permission from Murray Fowler[19]).

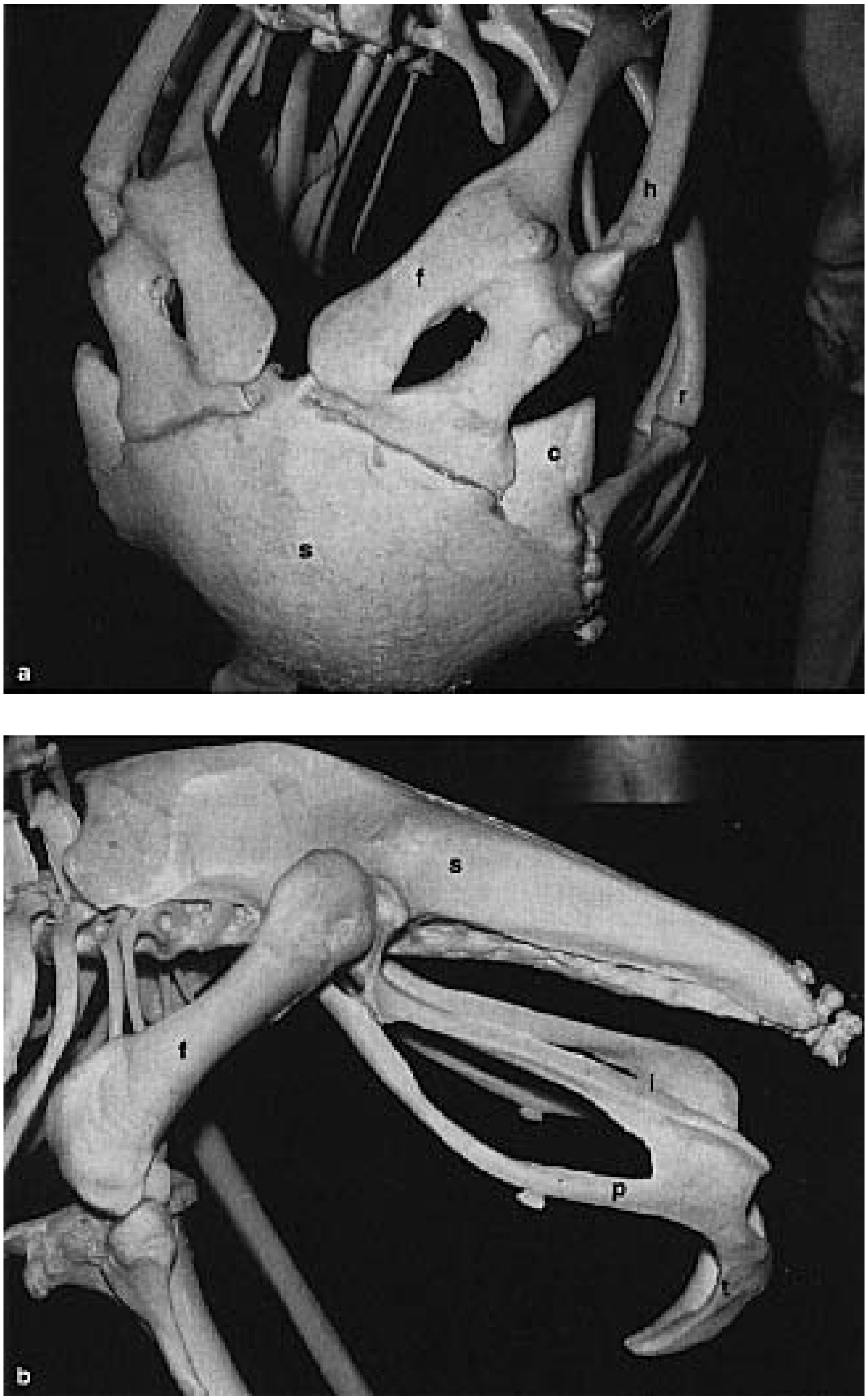


FIG 48.3 **a)** The sternum (s) of ratites lacks a keel, and the coracoid and scapula are fused to form the scapulocoracoid (f). Other structures of interest include the first rib (r), cartilaginous extension of the sternum (c) and the humerus (h). **b)** The pubic bones (p) of the ostrich are fused to form a solid ventral symphysis that supports the weight of the bird. Other structures include the femur (f), ilium (s), ischium (i) and pubic symphysis (t) (courtesy of Murray Fowler,[19] reprinted with permission).

ostrich there may be a small bone in the tendon of insertion of the muscle on the cnemial crest of the tibiotarsus. The crest is projected craniodorsally providing extra leverage for quick, sure forward movement of the leg when the bird is running, swimming or kicking. In the ostrich and emu, one of the tarsal bones remains unfused to the contiguous bones, which should not be misinterpreted as the knee radiographically.[19]

The ventral midline area of the abdominal wall consists only of the aponeuroses of the abdominal muscles. A surgical incision made along the midline penetrates the skin, subcutaneous fat (minimal) and a dense fibrous abdominal tunic. The next layer is retroperitoneal fat, which may be two to eight centimeters thick, especially in the emu. When a laparotomy is performed, the bulk of this adipose tissue should be peeled away prior to closing the body wall (Color 48.26).[19,20]

Respiratory System

Unlike other birds, the sternum is fixed and bears the weight of the resting ratite. Respiration occurs by lateral excursions of the chest wall, which must be considered during anesthesia and recovery. The normal respiratory rate in adult ostriches is 6 to 12 bpm, which may increase to 40 to 60 bpm during periods of stress, exercise or with high temperatures.[19] The syrinx is poorly developed in these relatively avocal birds. The lungs and air sacs are similar to those of other avian species, but the air sac capacity is greatly reduced. The distinct visceral outlines created by the air sacs in the radiographs of psittacines are not present in ratites. The femur is the only pneumatized long bone in ratites.

Emus have a longitudinal cleft in the trachea 10 to 15 cm cranial to the thoracic inlet that opens into a resonation chamber for vocalization (Color 48.16). This area is particularly well developed in the female. In the chick, a thin membrane covers the cleft. Air di-

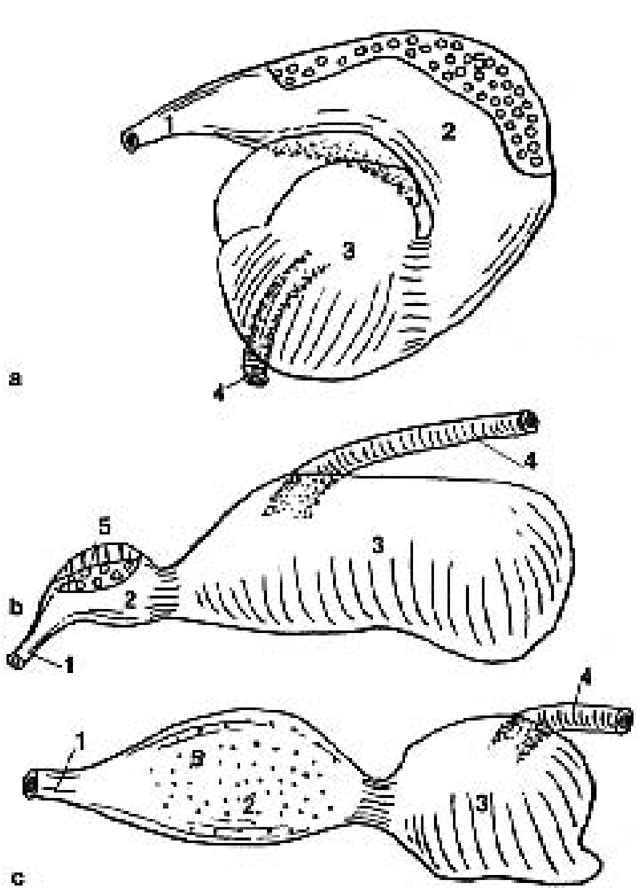


FIG 48.4 Anatomic arrangement of the 2) proventriculus and 3) ventriculus in the **a)** ostrich, **b)** rhea and **c)** emu. 1) esophagus 4) duodenum and 5) glandular area of the proventriculus (modified with permission from Murray Fowler[19]).

rected into the pouch causes a drumming sound in the female and a growling sound in the male. The skin of the neck enlarges laterally when the pouch is inflated.

The presence of this expandable pouch may complicate inhalation anesthesia in mature emus. If positive pressure ventilation is used to inflate the air sacs and ventilate the lungs, air may be directed into and thus inflate the pouch. Inflation of the pouch can be prevented by wrapping the lower neck with a self-adhesive bandage, taking care not to place excessive pressure on the major vessels of the neck. Ostriches may inflate the neck by gulping air into the esophagus.[19]

Digestive System

The digestive system of ratites reflects the ecological niche of these large grazing ungulates. Ratites have no crop, and the large proventriculus serves the feed storage function. Material deposited into the esophagus during tube-feeding is routinely regurgitated, creating a risk for aspiration. Consequently, gavage feeding requires that a tube extend into the proventriculus.

The proventriculus of the ostrich is a large, dilated, thin-walled structure that is easy to access surgically because it extends caudal to the ventriculus (Figure 48.4). In most avian species, the entire inner surface of the proventriculus secretes digestive enzymes. In contrast, the secretory region of the ostrich proventriculus is restricted to an area of glandular tissue on the greater curvature. The distal extremity of the ostrich proventriculus passes dorsal to the ventriculus and empties into this organ through a large opening on its caudal aspect. Ventricular foreign bodies can be easily removed through an incision made into the proventriculus.[19,20]

The ostrich ventriculus is a thick-walled structure similar to that in seed-eating birds. The ventriculus is situated slightly to the left of the midline at the caudal border of the sternum. Though the proventriculus and ventriculus can normally contain small stones, gastric impaction from the consumption of foreign bodies is a common problem in ratites, particularly in juvenile birds (Color 48.30).

Rheas have a small proventriculus and an elongated ventriculus. The stomach is slightly to the left of midline, dorsal to the sternal notch and more caudal to the sternum than is found with ostriches. In emus

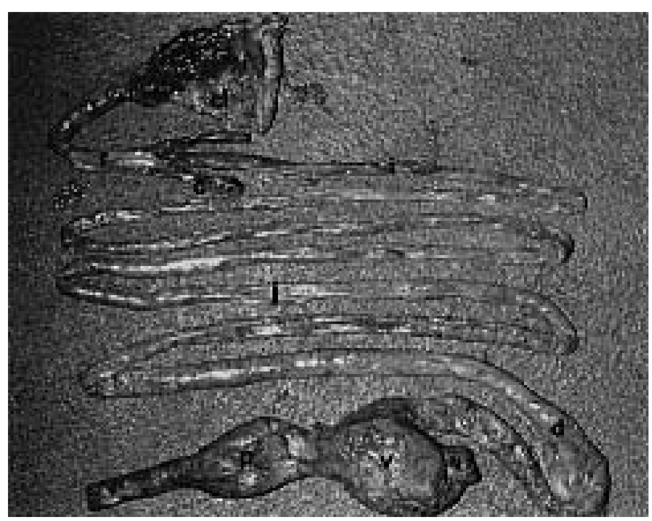
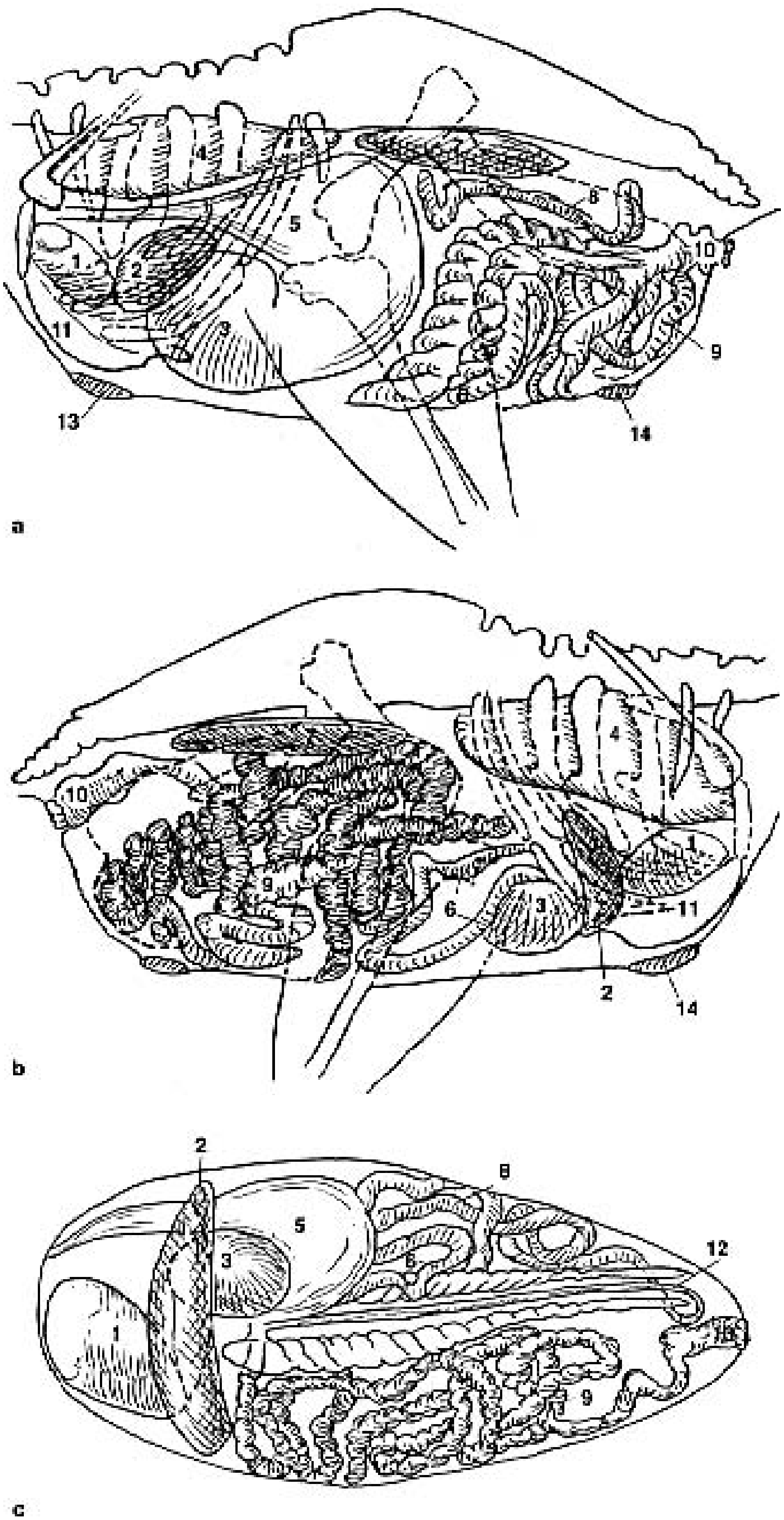


FIG 48.5 Digestive tract of an emu. Proventriculus (p), ventriculus (v), duodenum (d), jejunum (j), ileum (i), ceca (c), rectum (r) and cloaca (cl) (courtesy of Murray Fowler, reprinted with permission[19]).



a

b

c

and cassowaries, the proventriculus is large and spindle-shaped, and the ventriculus is slightly larger and less heavily muscled than the proventriculus (Figure 48.4). Cassowaries have no koilin membrane.

The opening from the ventriculus to the duodenum is on the right side in all ratites.[19,20] Intestinal length and morphology are highly variable. The small intestine is most important in the emu, in which it occupies most of the abdomen caudal to the ventriculus.

The cecae are paired in ratites (Figure 48.5). In the ostrich and rhea, the elongated, well developed ceca, visible immediately after entering the midline abdominal wall, course diagonally from right to left in a caudal direction. The lumen of the ceca appears sacculated as a result of spiral folds that increase the surface area in the organ and facilitate the fermentive digestion of fiber (Color 48.27). The cecae of the emu and cassowary are short.

The large intestine of the ostrich is voluminous and occupies the caudal right abdomen (Figure 48.6). The long, large intestine is considered necessary to digest high-fiber food items. The large intestine of the emu is short (10 to 15 cm). The gastrointestinal transit time is slow in ostrich and rheas (36 hours) and much faster in emus (5 to 6 hours). Interestingly, emus produce a large portion of their energy through fermentation even though they have poorly developed ceca, a short colon and a rapid gastrointestinal transit time.[26] These differences in gastrointestinal morphol-

FIG 48.6 Thoracoabdominal anatomy of an ostrich: **a)** left lateral view **b)** right lateral view and **c)** ventrodorsal view. 1) heart 2) liver 3) ventriculus 4) lung 5) proventriculus 6) duodenum 7) kidney 8) jejunum 9) rectum 10) cloaca 11) sternum 12) ileum 13) sternal callosity and 14) pelvic callosity (modified with permission from Murray Fowler[19]).

ogy and function would suggest that free-ranging ostriches and emus ingest different feeds and may require different diets in captivity; however, these differences are rarely considered when formulating diets for these birds.

In the ostrich, the urodeum and coprodeum are separated by a muscular sphincter, making the ostrich the only bird that can urinate independent of defecation. The coprodeum is a large sac that may be covered by a dark tough membrane similar to koilin. The cloacal bursa begins to involute by 18 months of age in ostriches and rheas and is complete by two to three years in the male rhea, and three to four years in the female rhea (Figure 48.7).[60]

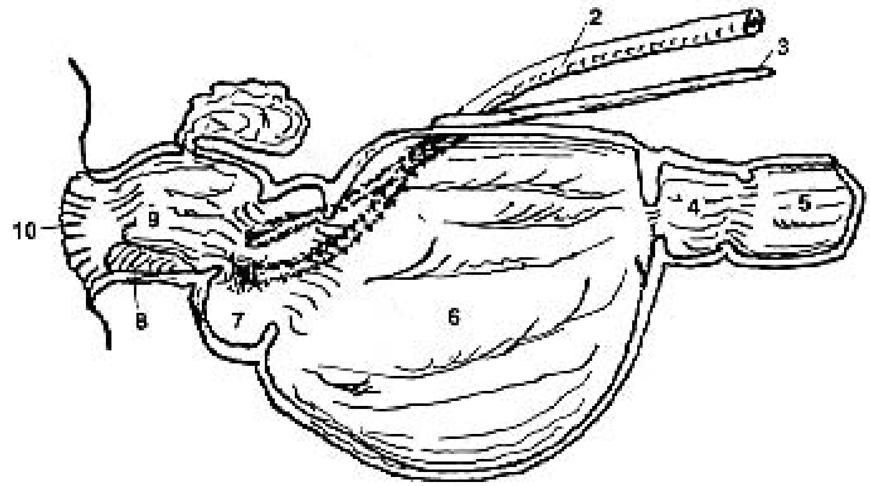


FIG 48.7 Right lateral view of the cloaca of an ostrich. 1) bursa 2) ureter 3) genital tract 4) rectal pouch 5) rectum 6) coprodeum 7) urodeum 8) genital eminence 9) proctodeum and 10) vent (modified with permission from Murray Fowler[19]).

The liver is cranial to the ventriculus and caudal to the transverse membrane (avian equivalent of a diaphragm). There is no gallbladder in the ostrich, but this organ is present in the emu and rhea.[19,20]

Reproductive System

All female ratites have a single left ovary and oviduct similar in form and function to other birds. All of the follicles ("eggs") that the female will have are present at birth. As a hen reaches sexual maturity, the follicles begin to develop, so the ovary has many visible follicles of different sizes at any one time (see Color 29).

FIG 48.8 Testicle size and location in a mature rhea. Cranial division of the left kidney (k), left testicle (t) and left adrenal gland (a) (courtesy of Murray Fowler, reprinted with permission[19]).

The male ratite has two intra-abdominal testicles that are located near the kidney. During the breeding season, the testicles increase 200 to 300 percent in size (Figure 48.8). The cock does not produce sperm during the non-breeding season.[19] Testicles are tan in all ratites except emus, in which they are black. Male ratites have a phallus that serves to transport semen from the ejaculatory ducts in the cloaca of the male to the cloaca of the female. The phallus is shaped differently in ostrich, emu and rhea; however, the function is the same, and the phallus contains a dorsal groove through which the semen travels (Figure 48.9).[19] The avian phallus serves no function within the urinary system and does not contain a urethra.[19]

Ratites of both genders possess a genital prominence that extends from the ventral aspect of the cloaca. This prominence may be visualized or palpated to determine the gender of any aged individual.[25] Gender determination is easiest in chicks between one

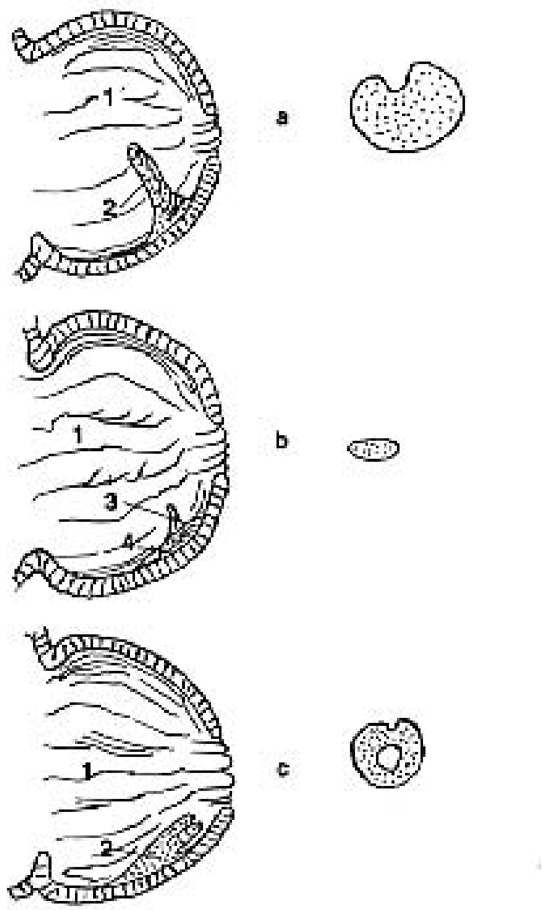


FIG 48.9 Left lateral view of the cloaca of juvenile ratites. The cross sectional view of the reproductive organ is also provided. **a)** male ostrich, **b)** female ostrich and **c)** male rhea or emu. 1) proctodeum 2) phallus 3) clitoris and 4) genital eminence (modified with permission from Murray Fowler[19]).

and three months of age. A lubricated gloved finger is used to expose the cranio-ventral aspect of the cloaca.

The male ostrich chick has a phallus that is conical in cross section, contains a palpable core of fibroelastic tissue and is characterized by the presence of a seminal groove. By comparison, the clitoris in the hen is laterally compressed, soft and lacks the seminal groove. The clitoris of the adult female remains approximately one to two centimeters in length (Figure 48.10). By six months of age the phallus of the male is approximately three to five centimeters in length and is readily detected on the ventral wall of the cloaca by palpation. The phallus of the adult male ostrich is "J"-shaped and when everted curves to the left (Figure 48.11). The phallus of the emu and rhea are considerably smaller, and the males are usually distinguished from females by the spiral conformation of the phallus.

Anatomic Considerations for Sample Collection and Supportive Care

Blood samples can be collected by routine venipuncture techniques used in most other avian species. Venipuncture can be performed using the jugular, brachial and medial metatarsal veins. The right jugular vein is larger than the left as in other avian species and is a convenient site for venipuncture or placement of intravenous catheters (Figure 48.12). The brachial vein is inaccessible in the reduced wings of the cassowary and emu, but is well developed in the large wings of the ostrich and can be easily accessed. The medial metatarsal vein is readily accessible in sedated or immobilized adult ratites and in unsedated chicks. The medial metatarsal vein generally is not used in standing adults due to the potential for being kicked.[4] Reference hematologic and biochemical values for the ostrich, emu and cassowary are listed in the Appendix.[36,37,41,58]

Intravenous catheters can be placed in any of the vessels used for venipuncture. The medial metatarsal vein is a common preference for intravenous catheterization of chicks (Figure 48.13). Catheterization of the brachial vein (18 ga) is preferred in adults. A 14 ga catheter can be placed in the jugular vein of an adult ostrich. Catheters should be secured in place using tissue adhesive followed by a light bandage.[4]

Samples for cytology and for culture and sensitivity can be collected from the oviduct of adult hens (see Chapter 29). Abdominocentesis can be performed on birds with clinical signs suggestive of intestinal torsions, penetrating foreign objects, egg yolk peritonitis or retained eggs. A teat cannula is the safest device for use for abdominocentesis in ratites (see Chapter 10).

Oral medications are relatively simple to administer by orogastric tube to chicks or tractable adults. Occasional feedings can be provided by placing an equine stomach tube directly into the proventriculus; however, to perform gastric lavage or supply sustained enteral nutrition, an equine stomach tube is passed through an esophagostomy incision and is sutured into place. The tube is most easily placed by introducing it orally into the esophagus, making an incision over the cranial end of the tube and retracting it back through the incision. Blended canine maintenance kibble administered TID has been suggested as an effective enteral nutrition product.[22]

Parenteral medications are often administered to ratites. Subcutaneous administration of medications

Ratites

Unless otherwise noted, color photographs in this section are courtesy of Brett Hopkins.

Color 48.1
a) A normal, healthy, well developed ostrich egg shell. Note that the shell is clean, has an even surface and is free of debris. **b)** An extremely thin egg shell from an ostrich shows abnormal deposition of calcium (blebs) and contamination of the shell with sand and debris.

Color 48.2
Two ostrich embryos died at about 40 days of incubation. The chick on the left is severely edematous, which in other avian species can be caused by excessive humidity during incubation. The chick on the right has a closed umbilicus with one-third of the yolk sac remaining externalized.

Color 48.3
Two ten- to fifteen-day-old ostrich chicks. The chick on the left has a distended abdomen caused by the retention of an infected yolk sac. The bird on the right has a small, tucked abdomen secondary to starvation.

Color 48.4
Two ostrich embryos that died late in the incubation period are stunted (40% smaller than normal), have abnormal feathering and curled feet. These findings are characteristic of a riboflavin deficiency in other avian species.

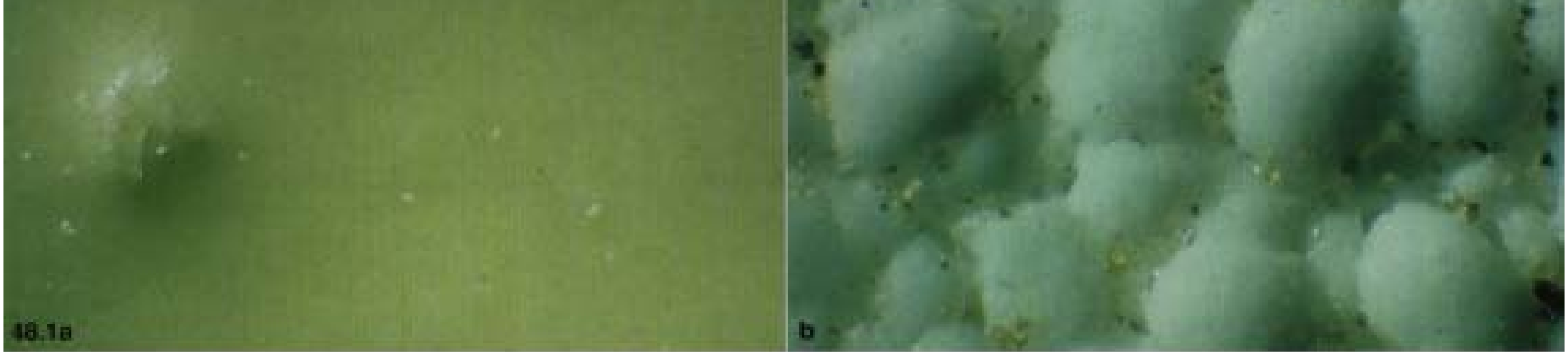
Color 48.5
a) Star-gazing in a one-day-old ostrich chick is suggestive of thiamine deficiency. Note also the edematous limb. **b)** The same chick is shown two days after parenteral administration of thiamine.

Color 48.6
A bulging, inflamed umbilicus is suggestive of an active yolk sac infection. These birds are best managed by surgically removing the infected yolk sac before an irreversible septicemia develops (courtesy of R. Korbel).

Color 48.7
The skin has been removed from the abdomen of a three-day-old emu chick to better visualize the infected yolk sac. *E. coli* was recovered from the yolk.

Color 48.8
a) A three-week-old ostrich chick that died following a five-day period of anorexia and progressive weight loss. Volvulus of the small intestines was caused by rotation of the yolk sac. Note the congestion of the yolk sac and distal portion of the small intestines (arrow). The kidneys (k), proventriculus (p) and ventriculus (v) are pale. **b)** A pendulous yolk sac (arrow) caused volvulus of the small intestines in a three-week-old ostrich chick.

Color 48.9
Infection of the pipping muscle may occur following the insertion of a non-sterile microchip.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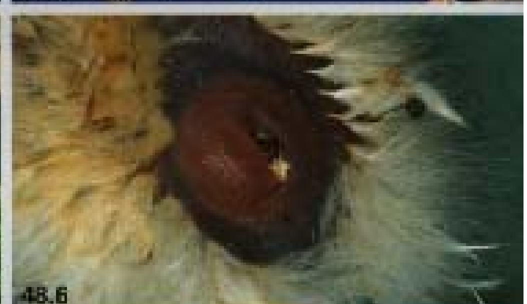
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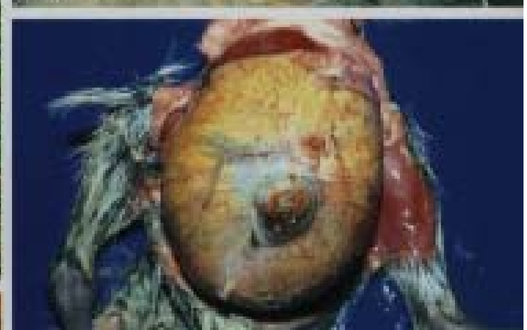
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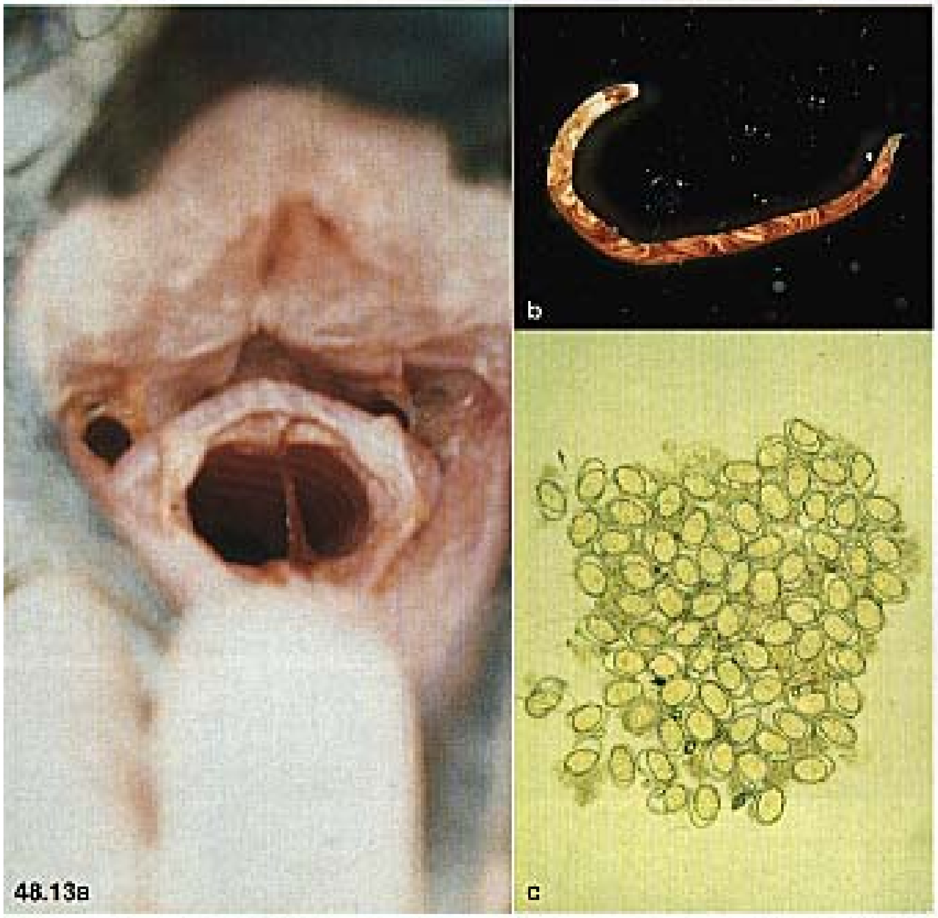
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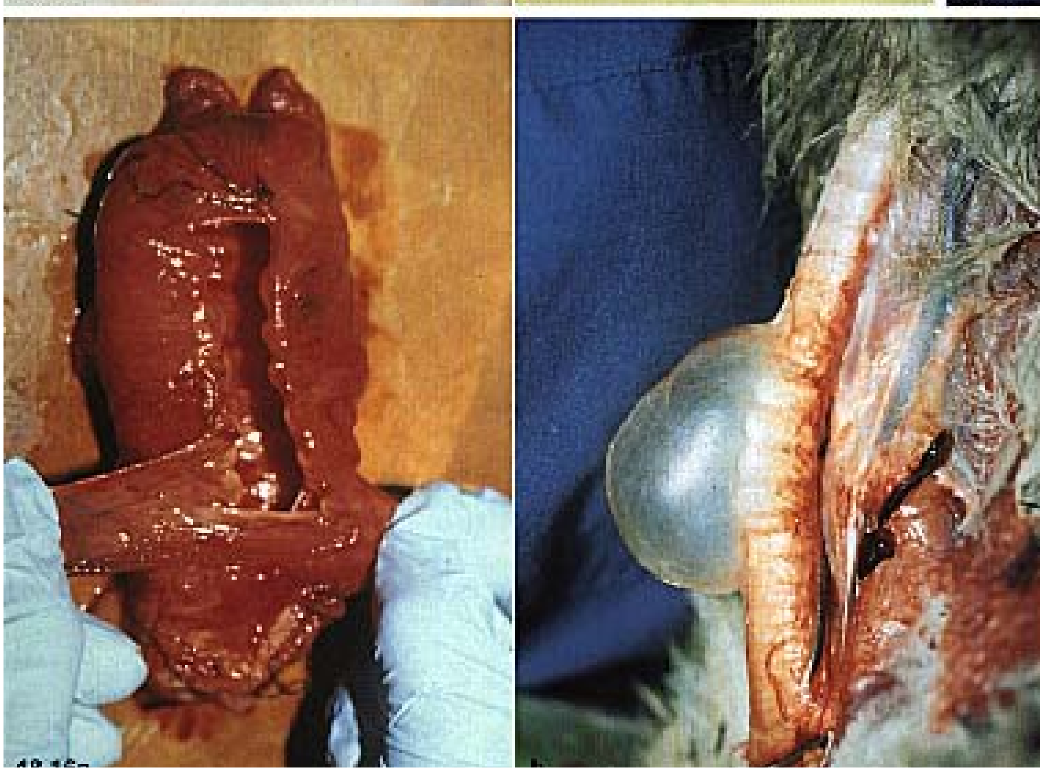
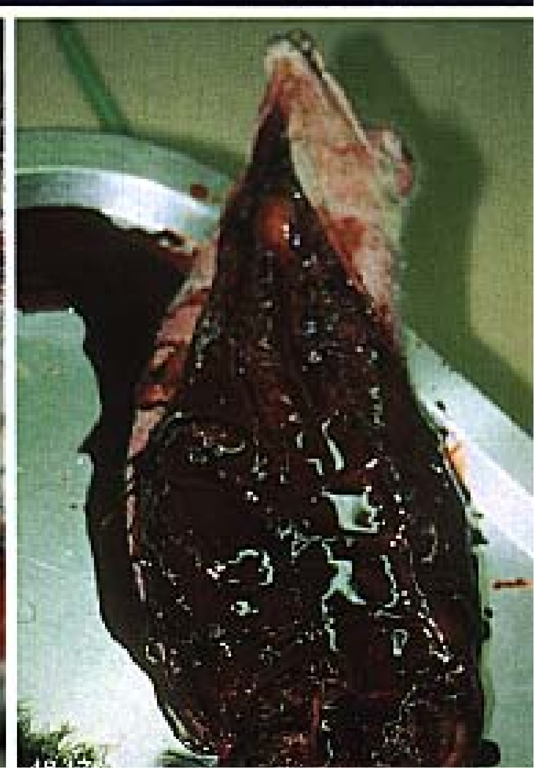




Ratites

Color 48.10
Cataract in a yearling ostrich (courtesy of James Stewart).

Color 48.11
Severe *Pseudomonas* sp. keratitis in a two-month-old ostrich. A fibrino-necrotic plaque is visible over the cornea.

Color 48.12
Uric acid deposits in the eyelid of a rhea that died from renal failure. Accumulations of uric acid were also noted on the tongue, thoracic and cervical vertebrae, heart, ventriculus, liver, intestines and kidneys.

Color 48.13
a) Rostrocaudal view of the oral cavity of an emu demonstrating hemorrhagic mucus in the trachea. This bird was infected with the tracheal worm *Syngamus trachea*. **b)** Female tracheal worm removed from an emu. **c)** Tracheal worm infections can be diagnosed by demonstrating the eggs in mucus collected from the trachea or pharyngeal area.

Color 48.14
Difference in quality between the feather of an African black ostrich (above) and a wild-type ostrich is shown (courtesy of James Stewart).

Color 48.15
Aortic rupture of unknown etiology in a six-month-old female ostrich that died acutely. The site where the aneurysm formed between the intima and media of the base of the heart is clearly visible. A copper deficiency has been discussed as a possible cause of this lesion.

Color 48.16
a) Tracheal diverticulum in a six-month-old emu. **b)** The diverticulum has been inflated. This structure is particularly well developed in adult female emus.

Color 48.17
A three-year-old male ostrich bled to death when the client ruptured the carotid artery while using a shepherd's hook to control the bird.

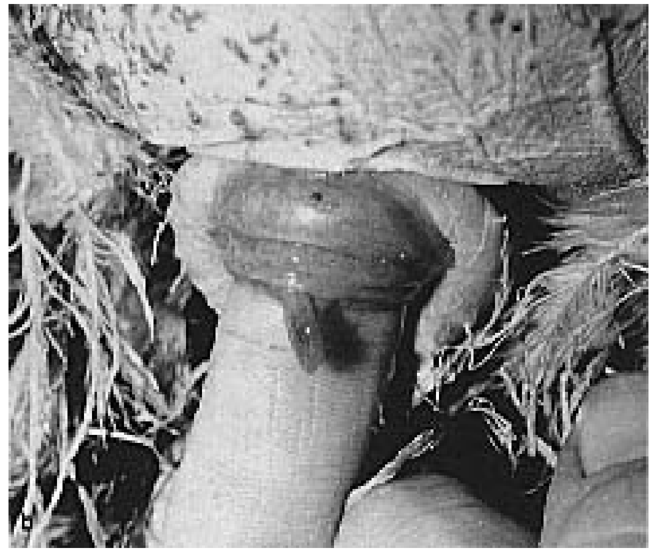


FIG 48.10 **a)** Erect phallus of a three-month-old ostrich male. The phallus is firm, conical in shape, curves to the left and has a prominent seminal groove. **b)** The erect clitoris of a three-month-old ostrich hen. The clitoris is soft, small and laterally compressed (courtesy of James Stewart).

is difficult because the skin adheres to the underlying tissues. Subcutaneous medications can be given in the knee web cranial to the thigh.[4]

The pectoral musculature of ratites is greatly reduced, and the large thigh muscles are frequently selected as a site for intramuscular injections. It has been suggested that this site may be inappropriate for the administration of nephrotoxic or renally excreted drugs because of the renal portal system. However, in one study involving the clearance of aminoglycoside, there was no difference in plasma levels when the drug was given in the posterior or anterior portions of the body.[30] This finding suggests that the renal portal system may not be of importance when considering drug administration in ratites. The epaxial musculature along either side of the spine serves as an alternative site to the thigh muscles for IM injections.

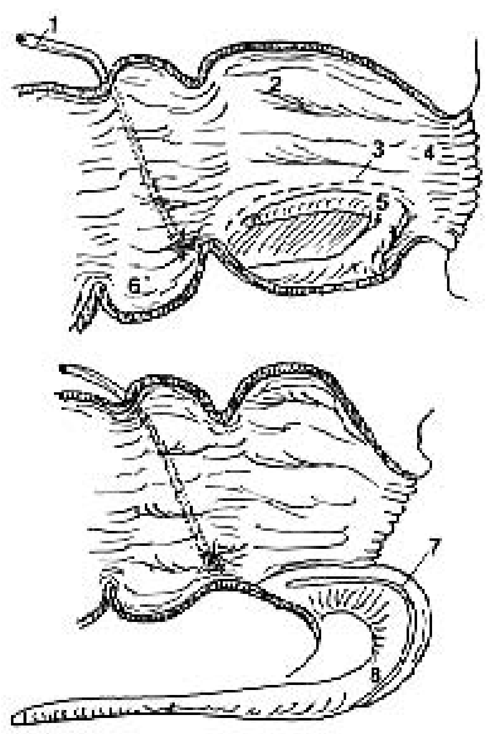


FIG 48.11 Left lateral view of the cloaca and phallus of a mature male ostrich: 1) vas deferens 2) proctodeum 3) crypt on the floor of the proctodeum 4) vent 5) retracted phallus 6) urodeum 7) dorsal sulcus and 8) erect phallus (modified with permission from Murray Fowler[19]).

FIG 48.12 The right jugular vein is easily accessible for venipuncture (courtesy of James Stewart).

FIG 48.13 The medial metatarsal vein is the preferred site for catheterization in ratite chicks (courtesy of Louise Bauck).

Adult Bird Management

Identification

It is suggested that all ratites be permanently identified. The importance of properly identifying birds is dramatically illustrated by a case in which an ostrich was identified on a health certificate as an "African black" and was insured for twenty thousand dollars. The bird died, and at necropsy the veterinarian identified the bird as a "blue-necked" ostrich, and the animal was cremated. The insurance company declared that the dead bird was not insured.

The implantation of a microchip provides one fail-safe method of identification. Sterilized microchips can be implanted immediately after hatching in the left side of the pipping muscle approximately two to three centimeters below the ear (Color 48.9). These chips help in the recovery of stolen birds, satisfy most insurance company requirements for identification and provide unmistakable identification for record keeping purposes.

Nutrition

Numerous pelleted rations are commercially available for ratites. Most of these are locally produced and are highly variable in content and quality. Information on the specific nutritional requirements of ratites is lacking. In South Africa, range-raised ostriches are fed on alfalfa pastures supplemented with maize. Captive ratites seem to maintain a good state of health and reproduction when fed a diet that contains 16 to 20% protein, 10% fat and 10% fiber. Diets with 18% protein produced the best weight gains in one small nutritional study that compared only the effects of varying levels of protein.[23] The calcium to phosphorus ratio should be about two to one.

Several commercial ratite foods are available with 18 to 23% protein and greater than 10% fiber. The feeding of higher protein diets (27%) to young birds would be expected to induce an accelerated rate of growth and may predispose them to leg deformities. Nutritional deficiencies are unlikely in birds that are fed a pelleted ration supplemented with good quality grazing areas.[31]

The adequacy of the breeder diet is reflected in the eggs produced. Generally speaking, hens deficient in carbohydrates, proteins and fats produce fewer, smaller eggs. Vitamin and trace mineral deficiencies can result in nutrient-deficient eggs. The effects may be graded with the level of the deficiency. Characteristic lesions may be noted at various stages of incubation or several days post-hatch. Gastrointestinal or other systemic disease may affect nutritional uptake and metabolism, resulting in nutrient-deficient eggs.

Hypovitaminosis A has been described in rhea chicks with clinical signs of epiphora, oral abscesses and decreased growth. A goose-stepping gait was thought to have been corrected with supplemental vitamin B_6.[16]

Thiamine deficiencies are thought to cause star-gazing (Color 48.5), and riboflavin deficiencies may be a cause of curled toe deformities in ratite embryos (Color 48.4) (Hopkins B, unpublished). Pantothenic acid and biotin have been associated with curling of the feathers and hyperkeratosis of the skin, particularly around the mouth, beak, feet and neck (Color 48.25).[44]

In a group of ostrich chicks fed crushed corn, hypovitaminosis E was suspected to have been the cause of muscle degeneration, which was characterized by paresis, poor weight gains and high AST (up to 1600 U/l) and CPK (up to 69,600 U/l) activities.[4] Vitamin E and selenium deficiencies also may occur in birds fed locally produced foods from regions with low levels of selenium in the soil.

Manganese deficiencies have been associated with slipped tendons in gallinaceous birds, but a deficiency of this mineral has not been associated with porosis in ratite chicks. Angular limb deformities are probably multifactorial, with decreased exercise, genetics and diets of high fat and protein all being involved.[44]

Restraint and Transportation

Ratites are large fractious birds that can easily injure themselves or attendants. Clients and veterinarians should be aware of the dangers associated with handling ratites and should be well versed in restraint techniques. Male ostriches are particularly aggressive during the breeding season and must be handled with caution. Any handling procedure is best performed in an area with solid walls in which the lights can be dimmed. Many basic procedures can be performed in these confined areas without the need for excessive physical restraint.

The natural defense of ratites is the kick, enhanced by the well developed toenails. Ostriches kick straight forward at chest level to the bird, followed by a downward sweep of the foot. Emus and cassowaries may kick either forward or backward and may incorporate wide lateral swings into the range of motion. All ratites jump with great agility, and when restrained, cassowaries roll onto their backs with their legs flailing.

When physical restraint is necessary, it is best to make slow, methodical movements. Working with untrained birds is an exercise in patience. It is important when handling chicks to use gentle restraint because rough handling can cause fractures, tendon damage and severe bruising. Whenever possible, larger chicks are usually herded rather than carried, but they may also be guided by placing one hand across the sternum and the other below the pelvis.

Most adult ostriches will become tractable when the head is covered with a dark, tight-fitting cloth hood, such as a sweatshirt sleeve (Figure 48.14). The sleeve is placed over the arm, the bird is grasped by the beak and the sleeve is then inverted over the head of the ostrich. The natural curiosity of a captive ostrich is usually sufficient to tempt the bird close enough to the handler to grasp the beak. A long smooth shepherd's hook can also be used to grasp the ostrich around the neck and lower the head, but the handler must be prudent of potential injuries to himself or the bird (Color 48.17).

Ostriches may also be restrained without a hood if one person holds the head and neck horizontal to the ground while a second person provides upward and forward pressure to the pelvis. Grasping ostriches by the wings is a common cause of fractures and paralysis. Emus can be crowded into a corner and restrained by standing straddled over the bird's hips while holding the bird across the sternum with the hands (Figure 48.15).

FIG 48.14 A sweatshirt sleeve or similar cloth tube can be placed over the head of a ratite to keep it quiet and to facilitate examination or recovery from anesthesia (courtesy of James Stewart).

Mechanical Restraint

Enclosures designed with catch pens, alley systems and stanchions facilitate the restraint of ratites. Facilities designed for cattle and horses usually include fencing inappropriate for use with ostriches. One side of the standard horse trailer is suitable to accomplish most procedures on an adult ostrich. A stanchion is used in the commercial feather industry to restrain ostriches for the clipping and plucking of feathers and is excellent for veterinary procedures. The stanchion consists of two thigh-high side-bars in the formation of a "V," with a strap to be placed over the shoulders and a bar to be positioned behind the legs and below the pelvis, thus restricting the bird's motion in all directions.

Chemical Restraint

Anesthetic protocols for the different ratite species are basically similar when adjusted to body size.[31,54] The author's preference for smaller birds (under 20 kg) is face mask induction with four per cent isoflurane, followed by intubation and maintenance at two to three percent levels. The success of any anesthetic episode can be improved by performing the procedure in a small, quiet, dark room. Injectable agents are adequate for short procedures such as wound repair or casting in large birds. The author's agent of choice is tiletamine-zolazepam administered intravenously at 2-8 mg/kg depending upon the desired duration of anesthesia. Induction time is less than 15 seconds, and cardiac and respiratory functions are well maintained. The duration of anesthesia for a single dose is approximately 20 to 40 minutes, and supplemental doses may be administered as needed.

FIG 48.15 Emus can be restrained by straddling the hips of the bird and wrapping the hands around the sternum (courtesy of James Stewart).

Alternatively, ketamine hydrochloride may be administered IV at 2-5 mg/kg when used in conjunction with either xylazine at 0.2-0.3 mg/kg or diazepam at 0.2-0.3 mg/kg. Ketamine alone gives unacceptable results. A smoother recovery from injectable agents may be facilitated by the administration of aza-

FIG 48.16 Isoflurane is the anesthetic of choice for use in ratites. Anesthetized birds can be intubated with standard, cuffed endotracheal tubes that are available for small animals (courtesy of Tom Tully).

paerone IM at 1 to 2 mg/kg following induction, or the administration of diazepam IV at 0.2 to 0.3 mg/kg during recovery. In one study of ostriches, induction with tiletamine/zolazepam at a dose of 4.4 mg/kg IM was found to provide the best induction and recovery.[12]

For surgical anesthesia, large ratites are generally induced with low doses of tiletamine-zolazepam by intravenous administration and maintained on either 2 to 4% halothane or 2 to 4% isoflurane. Mature ostriches can be intubated using 14 to 18 mm cuffed endotracheal tubes (Figure 48.16). Intermittent positive pressure ventilation can be performed with a peak pressure of 15 to 20 cm of H_2O. The tidal volume of ratites is considered to be 10 to 15 ml/kg. Birds may become apneic immediately after or commonly at 15 to 20 minutes into anesthesia. These birds should be provided IPPV at 6 to 30 breaths per minute until $paCO_2$ levels have stabilized.

Bradycardia, apnea, hypercapnia, hypocapnia and movement are complications of anesthesia in ratites. Glycopyrrolate (0.011 mg/kg) was effective in reversing bradycardia (<30 bpm) in one bird. Heart rates in young ostriches at rest are normally 100 to 150 bpm and in the adults, 80 bpm.[24] The respiratory rate in a group of anesthetized ostriches was 25 to 40 bpm and the heart rate was 65 to 70 bpm. Mean blood pressure varied from 165 to 220 mm HG. In another group of anesthetized ostriches, the mean blood pressure was 60 to 137 mm Hg.[39]

Ratites can easily injure themselves during the ataxic phase of anesthetic recovery, particularly following injectable agents. Extubation should occur in recovery when the bird is swallowing. Large, shaded areas, padded with mats or straw and clear of objects or walls within reach of the flailing legs can be used for recovery. Alternatively, a bird may be packed in a crate that is heavily padded with straw to restrict extension and flailing of the legs. Because ratites respire with lateral excursions of the chest, the sternal position for recovery is preferred. Small ratites can be recovered by wrapping them in a towel and using manual restraint. Adults should remain hooded with minimal disturbances. When the bird sits sternally with the head held upright, the hood should then be removed.

Transportation

When considering the transportation of ratites, it should be remembered that these birds are bipedal and have difficulty balancing when provided unstable footing. Excessively large trailers with slippery surfaces have been a leading cause of injury and mortality. Chicks and juveniles may be transported by land or air most safely if confined to small individual crates. Specifications for air shipment of ratites are outlined in the International Air Transportation Association live animal regulations and container requirements. The individual compartment of a standard horse trailer is well suited for the routine transport of one adult ostrich. The compartment should be modified to have solid smooth walls to the floor, and adequate traction can be provided by covering the floor with wet wood shavings or sand. Hauling birds at night tends to keep them calm and reduces the possibility of overheating. Food and water should be offered two to three times per day, but should be removed from the compartment while traveling.

Ostrich Management

Housing

Adult ostriches are maintained in outdoor paddocks.[53] They are tolerant of extremes in weather conditions, faring well even in snow. Birds in southern climates can be maintained outdoors in the win-

FIG 48.17 Wire (smooth or barbed) should never be used for fencing ratites. This breeding bird became entangled in the wire and died (courtesy of James Stewart).

ter with adequate protection from the wind, but birds in northern areas may require completely enclosed areas to survive long periods of sub-zero temperatures. Ostriches are gregarious in nature, with one male breeding several hens; however, they do have preferences, and incompatible pairs are common when birds are not allowed to select their mates. The breeding group in intensive operations consists of one male and one to three females. Semi-intensive farms utilize large paddocks containing many birds with an excess of hens. Visually separating breeding groups and allowing a hen to select a male may improve reproductive success. Housing trios adjacent to each other may cause males to spend time soliciting attention from females in adjacent enclosures, while neglecting the hens in his pen.

Breeding paddocks in intensive systems are typically one-quarter to one-half acre in size. A paddock with 5,000 square feet would be considered minimum for a pair of adult ostriches. Fencing should be approximately two meters tall, clearly visible to a running bird and designed so that the feet or neck cannot become entangled within the fence. The bottom of the fence can be raised 40 cm from the ground to prevent the bird's legs and feet from becoming entangled.

Stranded wire fence (barbed or smooth) should never be used for ratites (Figure 48.17). Wood corrals, 2 x 4" field fencing, chain-link fence or pipe fencing are all effective. Electric fencing may be necessary to prevent terrestrial predators from entering the compound.

Birds should have a supply of fresh water and dry food at all times. Placing the food and water station in the fence line with a flap to allow access without entering the paddock is the easiest way to maintain ostriches. Ratites readily adapt to the use of automatic water supplies and bin-type feeders. These devices reduce labor and minimize disturbances in the breeding group.

Breeding Behavior

In general, ostriches reach puberty around two years of age, but are not at full reproductive maturity until four years of age.

Ostriches are long-day breeders, are photoperiod dependent and primarily breed in the summer (Table 48.1). Free-ranging ostrich cocks do not produce sperm in the non-breeding season. With increasing day length, testosterone production increases and secondary sexual characteristics such as reddening of the beak and legs, vocalization and territorial displays (kanteling) begin (Figure 48.18). Sperm production, which is controlled by follicle stimulating hormone, starts at the same time.

CLINICAL APPLICATIONS

Specific factors that might affect the onset of maturity in ostriches include:

- The "breed" - The smaller African black ostrich matures earlier than the larger North African subspecies ("red-necked").
- The season of hatch - Birds that hatch during a period of increasing day length mature faster than those that hatch during a period of decreasing day length. Throughout its lifetime, a bird hatched early in the year will produce better than a bird hatched late in the year.
- The plane of nutrition
- The environment - The specific effect of temperature on reproductive activity is unknown; however, extremes in temperature may stop production or reduce egg fertility.[27]

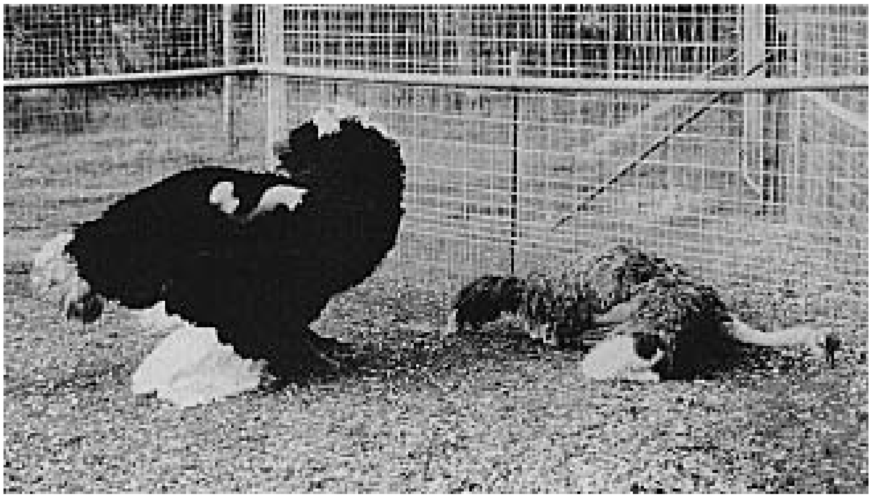


FIG 48.18 Courtship behavior in ostriches. The male (left) drops to his hocks, fans his wings and slaps his head on his back (kanteling). The female (right) is on the ground with the neck extended in a receptive position (courtesy of James Stewart).

The cock displays to the hen during breeding season by dropping to his hocks, fanning his wings and striking the back of his head on either side of his back. In captivity, males frequently display and make a booming noise to any visitor that approaches the enclosure. The hen flutters her wings, drops her head and makes a snapping motion with her beak. She will drop to the ground with her head extended. The male mounts from the left placing his right foot on the hen's back (Figure 48.18). He drops to his hocks and intromission occurs. During copulation, the male will strike his back with his head. When ejaculation occurs, the male makes a guttural sound.[27]

In the United States, the breeding season varies from north to south. Birds in the northern US have a laying season from May to September, while birds in the southern US may produce all year. The nest consists of a shallow depression in the ground that is formed and protected by the male. Ostriches and rheas usually lay eggs in the afternoon or evening while emus generally lay at night. If left to parental care, the eggs are incubated by the male at night and the female during the day. In free-ranging groups, the dominant hen will incubate the eggs during the day. The dominant hen recognizes her eggs, and if the nest is overcrowded (20 to 25 eggs), the hen will remove the eggs laid by rival females. Nondominant hens may lay in several nests and be bred by several males. Both adults brood the chicks.[27]

Ostrich hens are indeterminate layers and if the eggs are removed, a hen may lay an egg every other day throughout the breeding season. Free-ranging hens may have a normal clutch size of 15 to 25 eggs. Forty is the average for hens in captivity; however, some birds may produce 70 to 100 eggs per year.

Both sexes may have periods of reproductive quiescence within the breeding season, each lasting three or four weeks. During this time the female stops laying and the male "goes out of color" (ie, the bright red coloration of the face and tarsal scutes fades).

Breeding males may become territorial and aggressive, and stressful social interactions within and between breeding groups can decrease reproductive behavior and egg fertility. These problems can best be prevented by having visual barriers between breeding groups. Fertility in the male may decrease with prolonged breeding periods, and separating the breeding adults for several weeks may improve egg fertility.[31]

Emu

Breeding Behavior

Emus are managed in a manner similar to ostriches, but with proportionately smaller facilities. During

TABLE 48.1 Factors Affecting Ostrich Reproduction[27]

Behavior:	Most of the breeding birds available in the US are from free-ranging stock and have not been genetically selected to adapt to captive conditions. Some individual birds adapt well to enclosed spaces and varying terrain while others do not.
Genetics:	Highly productive hens are more likely to produce daughters that also are extremely productive.
Environment:	Severe weather fluctuations, looming predators and objects overhead have been shown to decrease productivity.
Age:	Ostriches sexually mature two to three years after hatch, and production improves as the bird ages. The most productive age for ostriches has not been established.
Nutrition:	Obesity is one of the biggest causes of decreased production in birds. Although nutritional deficiencies can decrease productivity, nutritional excesses are just as detrimental (Color 48.26).
Season:	Production is best in the early and middle portion of the breeding season.

breeding, the female sits on the ground with her cloaca everted. The male drops to his hocks behind her and intromission occurs. The male pecks the hen's back and makes guttural noises during ejaculation. Breeding occurs most often in the morning and evening hours.[27] Adults are typically maintained as pairs, but colony breeding is effective. Eggs are laid in grass or straw and are partially hidden. The male conducts the incubation and chick-raising. The male may not leave the nest during the entire incubation period, leading to a substantial degree of weight loss.

Emus are short-day breeders, with a breeding season that lasts from October to March in the United States. Free-ranging emus are gregarious during the non-breeding season, but these birds tend to form pairs or trios during the breeding season. Free-ranging birds may begin egg laying at two to three years of age. Captive females may require one to two extra years to reach sexual maturity.

Rhea

Breeding Behavior

The male rhea performs the courtship displays, builds the nest, incubates the eggs and rears the young. The male attracts the apparently disinterested females with wing-spreading and head-swaying displays. Several females will lay eggs near a nest established by the male. The male collects these eggs for about one week and then initiates incubation of all the eggs at one time so that the hatch is synchronized.[27]

TABLE 48.2 Infectious Diseases Reported in Ratites

	Ostrich	Rhea	Emu	Cassowary
Viral				
Coronavirus	x			
Alphavirus			x	
Avipoxvirus	x	x	x	x
Influenzavirus type A	x			
Paramyxovirus type 1	x	x	x	x
Bacterial				
Bacillus anthracis	x			
Bordetella avium	x	x		
Clostridium botulinum	x			
Campylobacter jejuni	x	x		
E. coli	x	x	x	x
Edwardsiella tarda	x			
Pasteurella multocida	x			
Haemophilus paragallinarum	x	x		
Clostridium perfringens type C	x	x	x	
Clostridium colinum	x			
Salmonella spp.	x	x	x	x
Treponema sp.		x		
Mycobacterial				
Mycobacterium avium	x	x	x	
Mycoplasmal				
Mycoplasma sp.	x			
Mycoplasma synoviae	x			
Mycoplasma gallisepticum	x			
Mycoplasma meleagridis	x			
Chlamydial				
Chlamydia psittaci	x		x	
Mycotic				
Aspergillus fumigatus	x	x	x	
Aspergillus flavus	x		x	
Aspergillus niger	x			
Candida albicans	x	x	x	
Rhizopus oryzae	x			

Medical Disorders and Therapies

Among the ratites, ostrich diseases are best documented because of the development of a breeder industry (Table 48.2). When viewed from the perspective of disease, ostriches are little more than giant chickens. Most medical disorders of ratites have models within the commercial poultry industry. The important differences lie with the susceptibilities and relative prevalence of these diseases. Many of the infectious diseases are also shared by psittacines, waterfowl and other common companion and aviary birds. Sound management dictates that ratites should not be reared in close proximity to other types of birds.

Due to the lack of research targeted at accurate identification of organisms that affect ratites, the list provided in Table 48.2 will certainly prove to be incomplete. There are many disease syndromes that epidemiologically suggest an infectious etiology for which a specific pathogen has not been described. Waste management, sanitation and human movement patterns within the flock are essential in preventing the transmission of infectious agents from paddock to paddock or from farm to farm. Ratite clinicians must be acutely aware of the role they can play in the transmission of disease through improper hygienic practices. New birds should be quarantined in an area separated from the remainder of the group for at least one month. During this period, the birds should receive a thorough physical examination and should be treated for parasites.

Reproductive Abnormalities

On the average, 50% of the ratite eggs produced annually in the United States are infertile. This represents a considerable economic loss given current market values for fertile eggs. Fertilization of the egg must occur during the first 15 minutes after ovulation while the egg is in the infundibulum.[27] Ratite hens are subject to all the reproductive disorders seen in other birds including oviduct infections, egg retention, uterine prolapse, internal ovulation and egg-related peritonitis. The anatomy, physiology and pathogenesis of disease are comparable to the psittacine model (see Chapter 29). In contrast to the smaller avian species, ratites may be afflicted with severe reproductive disorders for months or even years, but remain otherwise healthy and exhibit no outward signs of disease. Excessive ventrodorsal movement of the cloaca when a hen is jogging may be an early sign of egg-related problems.

A diagnosis of reproductive tract disease is based upon the reproductive history, physical examination (including cloacal palpation and eversion of the phallus), and diagnostic tests including hematology and serum biochemistry, oviduct cultures, abdominocentesis, radiology and ultrasonography.

Prolapse of the phallus has been described in male ostriches. A partial prolapse may occur in reproductively active males with no adverse effects. The precise etiology is unknown, but debilitation toward the end of the breeding season and extreme weather fluctuations have been suggested as causes. Frostbite or necrotizing dermatitis are frequent sequela to a prolapse. Full prolapse requires replacement of the phallus into the cloaca, with or without a purse-string suture, and administration of nonsteroidal anti-inflammatory agents. If the phallus is traumatized, daily washes with a disinfectant solution and administration of systemic antibiotics may be indicated. The prognosis is good if the damage is not too extreme.

Intersex appears to be common in the ostrich. The black pigment of the male's feathers is due to a lack of estrogen. A mature black bird that sexes cloacally as a hen will not reproduce and may have inactive ovaries, testes or both. Many young hens may be very dark brown or even have a few black feathers, but become gray with maturity.

Prolapse of the vagina can occur without egg laying and may be seen in hens less than one year of age. These prolapses are thought to be caused by unseasonably cold temperatures. Replacement and application of a retention suture are usually corrective.[27]

Peritoneal hernias occur in the caudal abdominal cavity, allowing the intestines and uterus to prolapse into the pericloacal region. Affected hens appear to have a large pericloacal swelling. Ultrasound is diagnostic. Surgical repair is required.

E. coli, *Pseudomonas* spp., *Acinetobacter* spp. and other gram-negative bacteria are common causes of oviduct infections in ostriches. Affected hens generally present with a history of erratic egg production, cessation of egg production or malformed or odoriferous eggs. On physical examination, the temperature and respiration are variable. The hen may have a discharge below the cloaca and may have a peculiar odor. Affected hens often have white blood counts ranging from 20,000 to 100,000 (pronounced heterophilia in acute cases or lymphocytosis in chronic cases); however, the severity of the infection varies with the etiologic agent. In mild cases, only the uterus or shell gland (metritis) may be affected, and in these hens clinical signs range from the formation of abnormal shells to the cessation of breeding.

Salpingitis or peritonitis may also occur with chronic infections or those that occur secondary to septicemia.[27] Therapy for metritis should include appropriate antimicrobial therapy, multiple vitamin and calcium injections. Surgical (laparotomy) or nonsurgical (vaginal) flushing of the oviduct can be used to remove accumulated debris.

Mycoplasma spp. and paramyxovirus have been isolated from the reproductive tracts of ratites but their clinical importance is unknown.[27] Papillomas have been described in the reproductive tract of both cocks and hens.

Egg binding may occur in ratite hens and is thought to be caused by genetic factors, malnutrition, cold weather or lack of exercise. Many affected hens are asymptomatic, while others may present with a history of tenesmus or with a vaginal prolapse. An impacted egg may be palpable in the caudal abdomen. Radiology or ultrasound may be required for diagnosis. Medical treatment consists of increasing the bird's ambient temperature along with the injection of multivitamins, calcium and oxytocin (prostaglandin may be superior, see Chapter 29). Ovocentesis procedures that have been described for correcting egg binding in other avian species are dangerous in the ostrich because of the likelihood of fractured egg shell damaging the oviduct. Impacted eggs should be removed surgically.

None of the methods traditionally used to artificially collect semen from birds is effective in ostriches because of their physical size, demeanor and lack of sexual imprinting response. Ostrich semen has been collected by means of forced massage and voluntary response; however, the semen collected is usually contaminated with urine, making assessment of concentration, volume and pH unreliable.[27]

Emu semen can be easily collected by voluntary ejaculation because the birds do sexually imprint on humans. Emu ejaculation volume averages 1.2 milliliters with 4.4 billion sperm per milliliter. Average pH is 7.32. Beltsville chicken semen extender in a 1:1 dilution has been found to be an appropriate diluent in some birds.[27]

Gastrointestinal Abnormalities

Ingestion of foreign bodies is a common problem in ratites. These birds are likely to swallow anything that fits into their mouths, and their keen eyesight and curiosity all but ensure that they will find many unusual items in their pen. Stones, sand, hardware and long-stemmed grasses are common offenders (Figure 48.19). The consumption of materials that induce impactions may be caused by primary enteric disease, inadequate feed availability, nutritional inadequacies and movement to a new environment with a different substrate. Ingestion of foreign bodies can be reduced by making certain that pastures and paddocks are covered with grass and do not contain abundant or clearly visible rocks or sand (Color 48.30). Decreasing stress by slowly introducing birds to a new area also may reduce the consumption of foreign bodies.

Impactions may be acute or chronic, primary or secondary. The most common clinical presentation includes lethargy accompanied by small, firm, fecal balls and a distended abdomen. Occasionally, affected birds may appear lame or be unwilling to rise due to weakness or pain. A cloacal prolapse may occur in chicks with proventricular impaction. Eighty-five percent of impactions occur in birds under six or

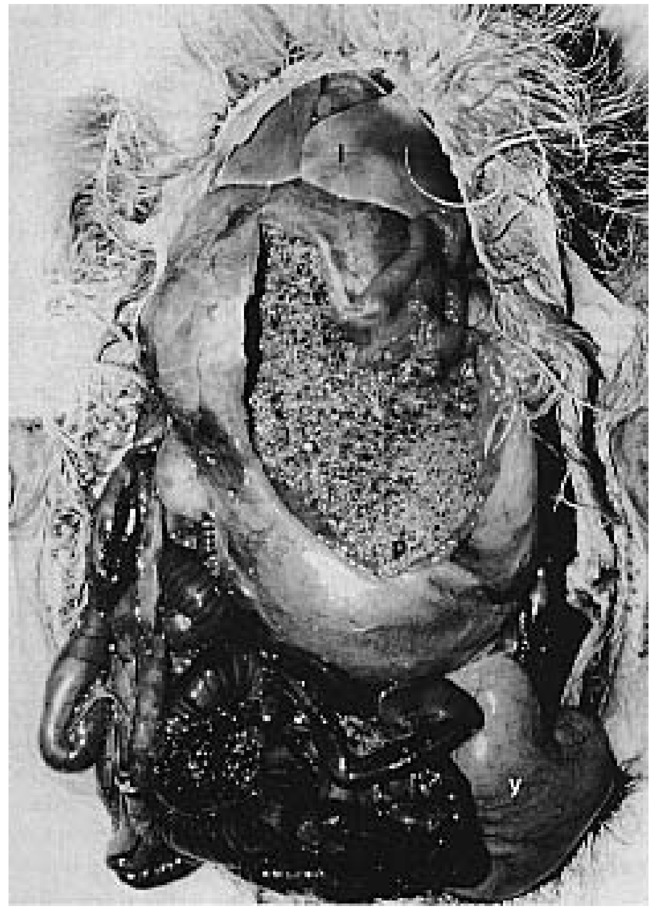


FIG 48.19 Sand impaction of the proventriculus (p) and ventriculus (v) in an ostrich chick. Note that the proventriculus in ostriches extends caudal to the ventriculus. Also visible are the liver (l) and yolk sac (y) (courtesy of James Stewart).

seven months of age, with 10 to 12% occurring in birds six to twelve months of age and 3 to 5% occurring in adults.[61]

Ingested foreign objects initially settle in the well developed proventriculus and obstruct the flow of ingesta or act as valves by blocking outflow into the ventriculus. In chronic cases, the blockage may also involve the ventriculus. Ventricular ulcers often develop from the trauma of constant grinding against an immobile mass.[22] Chronic and severe distension of the proventriculus may cause a permanent loss of muscle tone. These irreparable changes can be prevented through the early diagnosis and surgical correction of gastric impactions.

An impacted proventriculus can frequently be palpated on the left side of the abdomen by identifying the caudal and dorsal extremities of this organ. Impactions with rocks or sand can best be detected by palpating caudal to the sternum. Impactions caused by grasses and leaves may be more difficult to palpate.[22] Radiography can be used to document the presence of foreign bodies and an enlarged stomach. Ultrasound and gastroscopy may be other effective diagnostic techniques.

Psyllium administered by stomach tube may be effective in resolving mild cases of gastric distension, but true impactions can be resolved only by surgical removal of the foreign material. The proventriculus of the ostrich lies caudal and to the left of the ventriculus, and the surgical procedure is a slight variation of that used in psittacines (see Chapter 41). The proventriculus may be approached via either a midline or left paramedian incision that extends caudally from the caudal margin of the ventriculus.[28,52] A 15 cm skin incision is made caudal to the end of the sternum just to the left of midline, and the peritoneum is incised to expose the proventriculus. Allis tissue forceps or stay sutures can be used to manipulate the proventriculus. The proventriculus is temporarily sutured to the abdominal wall to minimize contamination of the coelomic cavity with ingesta. The proventriculus is then incised and the contents are removed. Closure is in two layers with a simple continuous primary closure that is oversown with a continuous inverting suture pattern.

Recovery from proventriculotomy is usually uneventful when the impaction is detected early. Chronic impactions may be accompanied by gastric atony, ulcers and candidiasis that require additional therapy. Birds can be offered alfalfa pellets and corn as soon as they are fully recovered from anesthesia.[28]

Many ingested objects will penetrate the wall of the ventriculus or proventriculus causing peritonitis (Color 48.29). The diagnostic procedures are the same as those used for gastric impaction. A proventriculotomy to remove the foreign body and surgical removal of necrotic peritoneum is indicated. Other common digestive disorders include cloacal prolapse and intussusception. Cloacal prolapse may be associated with diarrhea, or more frequently, with tenesmus due to constipation. Treatment of the prolapse is similar to methods used in other birds, but the initiating cause must be addressed to prevent recurrence (see Chapter 41). Intussusception is caused by hypermotility and gastrointestinal tract irritation and is often the result of an abrupt dietary change, especially when the new diet is higher in fiber.

Any number of ingested toxins can cause enteritis. Cantharidin, for example, is a toxin produced by the Three-striped Beetle, which swarms to feed on Mesquite trees and alfalfa hay. Mortality levels of 25% were reported in a group of emus that consumed these beetles that were attracted to a barn light.[61]

Torsion of the large bowel occurs sporadically. The etiology is unknown, but it is speculated that an abrupt change in feed may be an inciting factor. Clinically, these birds are dehydrated, depressed and produce a scant diarrhea. Diagnosis is confirmed through an exploratory laparotomy. Intestinal anastomosis may be effective in resolving mild cases that do not involve an extensive amount of the gastrointestinal tract.[61]

Porphyrins, a breakdown product of chlorophyll, are sometimes seen in the urates, giving them a red or orange coloration. Clients often mistake this substance for blood. This porphyruria is routinely noted following the ingestion of fresh green vegetation, and becomes more prominent during colder weather when water intake reduction makes the urine more concentrated.

Fractures

Leg fractures are common in ratites, and successful repair is difficult in these large, fractious, easily stressed, bipedal birds that must be able to ambulate postoperatively.[18,35] Femoral and proximal tibiotarsal fractures in ostriches frequently result in internal exsanguination. Tarsometatarsal fractures are usually open and infected. Prolonged recumbency results in muscle necrosis and tendon contraction.

The long-term use of slings is unsuccessful with ostriches, but emus tolerate them well. Tibiotarsal fractures in small birds under 15 kg are best repaired with a through-and-through, six-pin, modified external fixation device (see Chapter 42). Plates may be used in larger birds if bone quality is normal (Figure 48.20). Fractures of the phalanges, fractures of the distal metaphysis of the tarsometatarsus and luxations of the metatarsal-phalangeal or interphalangeal joints can be stabilized with fiberglass casts, to which most ratites readily adapt.

Wing fractures in ostriches frequently occur secondary to improper restraint. These may be resolved by placing the wing in a normal anatomic position and taping it to the body for six weeks; however, intramedullary pinning usually produces more satisfactory

FIG 48.20 A bone plate was used to stabilize a dome osteotomy site in an emu with a valgus deformity of the tibiotarsal bone. Fractures of the leg are relatively easy to repair surgically, but are rarely successful long-term because of problems in managing these large, fractious, bipedal patients (courtesy of Louise Bauck).

results. The author prefers a closed reduction and fixation technique. A small diameter pin may enter the distal caudal surface of the humeral shaft and be advanced through the fracture site into the proximal fragment. Alternatively, the pin may enter the proximal fragment at the fracture site, be advanced proximally out the deltoid crest and then retrograded into the distal fragment. The wing is then taped to the body for a period of six weeks to provide rotational stability to the fracture site.

Ruptured Aorta

Ostriches are prone to spontaneous rupture of the aorta (Color 48.15). These are most often located at the aortic arch, but ruptures in the caudal aorta have also been described. Surprisingly, this condition is seen with some frequency but the cause is unknown. Copper deficiencies have been implicated in other species. Overweight yearlings that are subjected to physical stress are most commonly affected.

Degenerative Myopathy

A large percentage of the young ostriches, rheas and emus submitted for necropsy have evidence of degenerative myopathy, with the majority of affected birds being less than six months of age.[47] It should be noted that myocyte degeneration appears the same histologically, regardless of the cause. In birds, several etiologies for degenerative myopathy have been reported, including capture myopathy, selenium or vitamin E deficiency, furazolidone and ionophore toxicity.[33] Clinical signs of degenerative myopathy that have been described in ratites include depression, reluctance to rise or move, and a rapid progression to death (two to five days). White foci and streaks in the myocardium and muscles of the hind limbs and myocyte degeneration with infiltration of macrophages and early calcification may be noted in birds that die several days after transport.[47]

Furazolidone is a nitrofuran antibiotic commonly used in the poultry industry. This compound is premixed in some chick starter feeds. Young turkey poults appear particularly susceptible to intoxication. Ionophore coccidiostats, such as monensin, lasalocid and salinomycin, are frequently added to chick starter feeds. These compounds may be contributing factors in the development of degenerative myopathy, and ratite producers should avoid the use of turkey or chick starter feeds that contain furazolidone and ionophores.

In the southern United States, pastures may contain the shrubs or trees of coffee senna (*Cassia occidentalis* or *Cassia obtusifolia*). In ruminants, ingestion of these beans may cause diarrhea, weakness, gait abnormalities, recumbency and muscular lesions consistent with degenerative myopathy. *Cassia* spp. intoxication has not been reported in ratites, but exposure to this toxic plant should be considered in cases of acute myocyte degeneration.[47]

Some authors believe that capture myopathy, which has been described in ratites with some frequency, is simply the acute manifestation of a chronic subclinical deficiency of selenium or vitamin E.[13] Degenerative myopathy appears to be primarily a disease of young ratites and higher levels of vitamin E may be required for growth. Two rheas with limb abnormalities had a mean plasma vitamin E level of 1.34 mcg/ml, compared with a level of 11.5 mcg/ml in apparently healthy rheas.[13] The mean serum levels of vitamin E in 23 ostriches was 2.1 mcg/ml, and in 23 emus was found to be 2.39 mcg/ml.[32] Circulating vitamin E levels in five rheas with no suspected deficiency ranged from 9.0 to 14.5 mcg/ml (mean of 11.60 mcg/ml).[13] Additional work is required to determine whether there is an age-related difference in serum vitamin E levels.

Normal liver selenium levels are not available for ratites but levels below 0.25 ppm are considered deficient in poultry, and levels below 0.35 ppm are considered marginally deficient. Adequate liver selenium levels in poultry range from 0.35 to 1.00 ppm wet weight.[46] Liver selenium levels ranged from 0.176 ppm to 0.986 ppm and liver vitamin E levels ranged from 0.69 mcg/gm to 9.10 mcg/gm in a group of ratite neonates with clinical or histologic lesions suggestive of degenerative myopathy.[47]

Additional investigations to determine the normal serum and organ levels of selenium and vitamin E and their correlation with degenerative myopathy, diet and health are necessary. Studies in horses with degenerative myopathy suggest that skeletal muscle and adipose tissue may be a better sample for determining vitamin E levels than liver.[47]

Therapy and Prevention

Treatment with vitamin E followed by immediate correction of the diet is the recommended therapy and is generally effective in early cases.[62] One author has suggested the parenteral administration of 3.0 mg of vitamin E and 0.06 mg of selenium/kg of body weight at two days of age, then weekly thereafter for a total of three injections.[43]

It is probably safer to supplement a bird with vitamin E rather than selenium. The latter has a low therapeutic index and can readily reach a toxic level. A chick with clinical signs suggestive of degenerative myopathy that was treated with this regime died despite therapy and had histologic lesions suggestive of the syndrome. Liver selenium levels in this bird were 3.738 ppm. The toxic liver selenium level in poultry is considered 4.00 ppm wet weight.[46]

A safer treatment regimen may be to use injectable vitamin E at a dose of 5.0 mg/kg IM every other day until clinical signs resolve. Adding oral formulations of vitamin E to the drinking water or the feed (100 IU/kg of feed) can be used for maintenance therapy. This treatment regime was successful in stopping morbidity and mortality in a flock of emu chicks experiencing degenerative myopathy.[47]

Neoplasms

Neoplasias have been reported in all ratites with none of particular prevalence. Lymphoid tumors have been described in ratites, and their similarity to tumors caused by leukosis virus in poultry warrants further investigation.

Lymphoma was diagnosed in a three-year-old female red-necked ostrich with a history of weight loss, mild bilateral conjunctivitis and a bright green mucoid diarrhea. On palpation, the liver was enlarged with rounded edges, and a bilobed mass was detected in the thoracic inlet. Abdominocentesis revealed a small amount of straw-colored, cloudy fluid with changes suggestive of chronic active hepatitis. Ultrasonography of the abdomen revealed an enlarged oviduct and multiple nodules within the liver. Abnormal clinical pathology results included a marked leukocytosis (160,000), lymphocytosis (95%), hyperproteinemia (6.9 g/dl), anemia (26%), hypoglycemia (102 g/dl), hypergammaglobulinemia and elevated AST (450 U/l) and CPK (6286 U/l) activities.[59]

Viral Disease

There has been little definitive work completed on viral diseases in ratites; consequently, specific diagnostic tests as well as vaccines are currently unavailable. The transmission of viruses from paddock to paddock or from farm to farm should be prevented through good sanitation practices and by not allowing visitors on the farm. Optimum nutrition, environmental conditions and reduced stress will ensure that a bird has an adequate immune system to resist disease.

Newcastle disease was the only disease of viral etiology reported in the ostrich prior to 1987. Recent international interest in ostrich production, particularly in the United States and Israel, has prompted further viral investigations. Numerous viruses have been detected in ratites by virus isolation or electron microscopy, but the clinical relevance of most of these findings is uncertain. Newcastle disease virus,[49] coronavirus, reovirus, influenzavirus and togavirus have been associated with specific diseases.[21]

Coronavial particles were reported in the small intestines of an 18-day-old ostrich chick that died following a one-week history of anorexia, lethargy, weakness and diarrhea. Lesions included dilation of the proventriculus, nutritional osteodystrophy and degenerative myopathy.[21]

Avian influenza was associated with high levels of mortality among ostriches in South Africa. Clinical signs in affected birds included respiratory signs, conjunctivitis, green discoloration of the urine and death. Although ostriches up to 14 months of age have been shown to be affected, morbidity is greatest in chicks under six months of age. Mortality may reach 80% in hatchlings and is complicated by secondary bacterial and nutritional problems. Characteristic postmortem findings include fibrinous air sacculitis, mucoid sinusitis, multifocal hepatic necrosis, splenomegaly and nephritis.

Fowlpox infections are well documented in ostriches.[45] This disease presents primarily as the dry form, although diphtheritic lesions may also occur. Vesicles that turn to encrustations form along the eyelids, ear openings, beak, neck and legs. Morbidity may be high but mortality is low. A commercial fowlpox vaccine administered at 10 to 14 days of age appears to provide some protection.

Eastern equine encephalomyelitis virus has been associated with high mortality (14 of 23 birds in one outbreak) among flocks of emus in the southeastern United States.[57] Clinical signs include profuse hemorrhagic diarrhea, depression, ataxia and death. Terminally, affected birds may become recumbent and develop hemorrhagic hyperemesis. Paired serum samples can be used to document an increase in antibodies indicating an active infection. Some infected birds will respond to supportive care.[57]

An inactivated equine vaccine is apparently effective in preventing the disease in emus. The initial vaccination is given at three months of age followed by boosters at six-month intervals. Written consent from the client and clearance from the insurance carrier should be obtained before the extra-label use of this vaccine is initiated.[57]

Bacterial Disease

Bacterial infections in ratites are similar to those described in other birds and may be associated with conjunctivitis, sinusitis, pneumonia and air sacculitis, gastroenteritis, omphalitis and septicemia. Young chicks are most susceptible, and the diagnostic techniques and treatments are comparable to those of other avian species. Although the normal intestinal flora for ratites has not been established, the birds are terrestrial; thus, ample gram-negative bacteria would be expected in the healthiest of individuals.

Common pathogens include *Pseudomonas, Klebsiella, Proteus, Salmonella* and *Campylobacter* spp. (see Color 24). *E. coli* can be a pathogen, but it is also a normal component of the ratite intestinal flora, and is frequently misrepresented as the cause of mortality in ostrich chicks (Color 48.21). Clostridial enteritis is a common disorder in ratites of all ages and is often associated with the excessive consumption of wet soil (Colors 48.18, 48.19). Botulism, clinically characterized by paralysis and death, has historically been a significant industry problem in adult ostriches in South Africa.

Tuberculosis is a common finding in adult ostriches. Affected birds develop a chronic wasting syndrome with visceral tubercles that can be detected by exploratory laparotomy. Salmonella outbreaks have been described in three- to six-week-old birds presented with acute weight loss, lethargy and bilaterally symmetrical distal limb edema. The total serum protein in affected chicks was less than 1 g/dl. Diarrhea was present in chronic cases but did not occur in peracute infections that resulted in rapid death.[61] *Staphylococcus* sp. is frequently associated with omphalitis and septic arthritis.

Ostriches are the only birds susceptible to anthrax, and the symptoms and diagnostic methods are identical to those for mammalian hoof-stock. Commercial anthrax vaccines are safe and effective in ostriches, following standard recommendations for hoof-stock. The client and insurance company should provide written consent before the extra-label use of this vaccine.

Mycotic Disease

In South Africa, aspergillosis causes the condemnation of up to ten per cent of inspected ostrich carcasses. Granulomatous nodules are most frequently distributed through the parenchyma of the lung, and only occasionally in the air sacs (see Chapter 22). Infections in older birds are enhanced by the inhalation of dust from dry feeds and soil. Outbreaks in chicks are associated with prolonged antibiotic therapy or inadequate hatcher and brooder hygiene.

If they occur, clinical signs include dyspnea, outstretched wings, exercise intolerance, anorexia and weight loss. Heterophilia is a common clinico-

Ratites

Color 48.18
Glandular portion of the proventriculus in a twenty-three-month-old ostrich with a *Clostridium perfringens* gastroenteritis. Note the congestion and hemorrhage in the mucosa of the proventriculus.

Color 48.19
a) Normal (left) and diseased leg from a six-month-old emu with *Clostridium shovaei* (black leg). **b)** Note the cavitation, hemorrhage and necrosis of the muscle.

Color 48.20
Stifle and tibia of a 20- to 22-month-old ostrich with severe hypoproteinemia. The white discoloration of the skin is caused by subcutaneous edema.

Color 48.21
E. coli infection in the stifle joint of a three-week-old ostrich chick. Note the white, coagulated exudate in the synovium of the joint. The infection can be seen extending into the muscle planes.

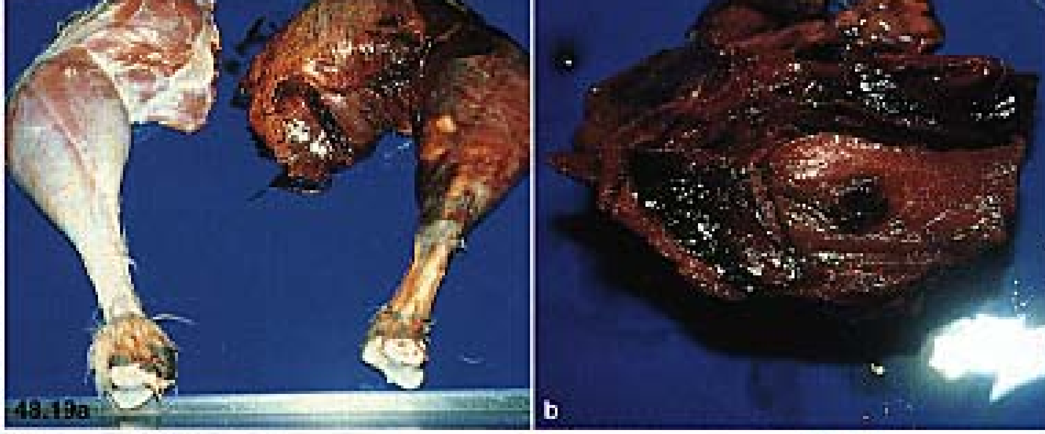
Color 48.22
Exuberant granulation tissue on the proximal metatarsus of an ostrich that was chronically lame and spent most of its time in sternal recumbency.

Color 48.23
Rotational deformity of the left tibiotarsal bone in a two-week-old emu. The distal tibiotarsus has rotated 180 degrees causing the plantar surface of the left foot to be oriented cranially. The rotation occurred over a six-day period.

Color 48.24
An ostrich with exuberant granulation tissue on the caudal aspect of the tarsometatarsal area following a skin injury. Lesions near the joint can progress to cause synovitis or arthritis, particularly if the wounds are infected with bacteria.

Color 48.25
Hyperkeratic lesions of the skin that are suggestive of pantothenic acid and biotin deficiencies are common in two- to six-month-old ostriches. **a)** Hyperkeratosis of the eyelids and cracking of the lower beak. **b)** Bilateral cracking of the skin at the commissures of the beak. **c)** Severe hyperkeratosis and cracking of the plantar foot surfaces. **d)** Hyperkeratosis of the skin on the distal third of the neck.


48.18


48.19a
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48.21


48.22


48.23


a
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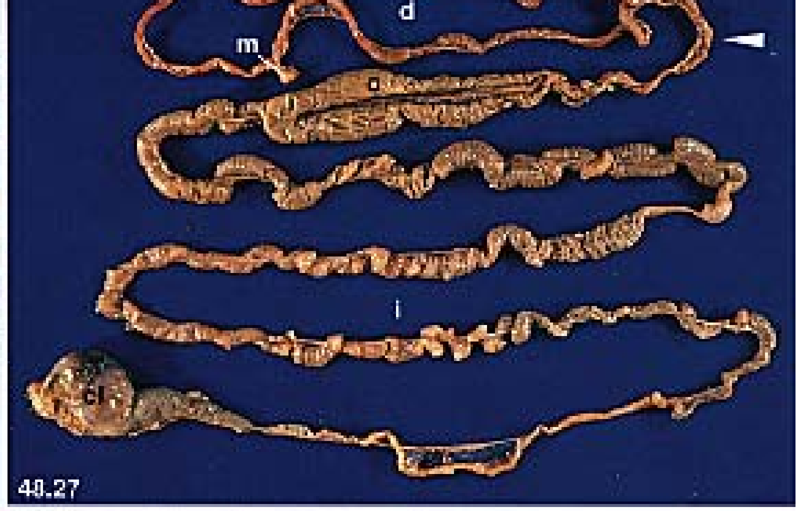

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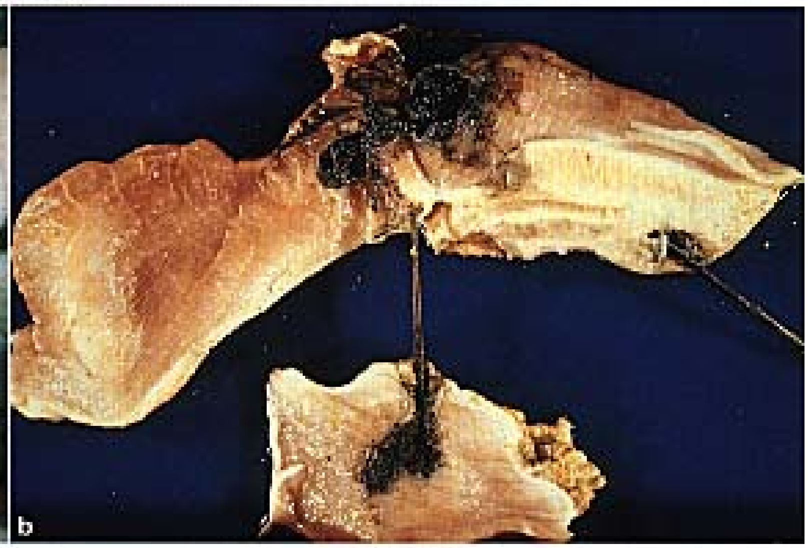

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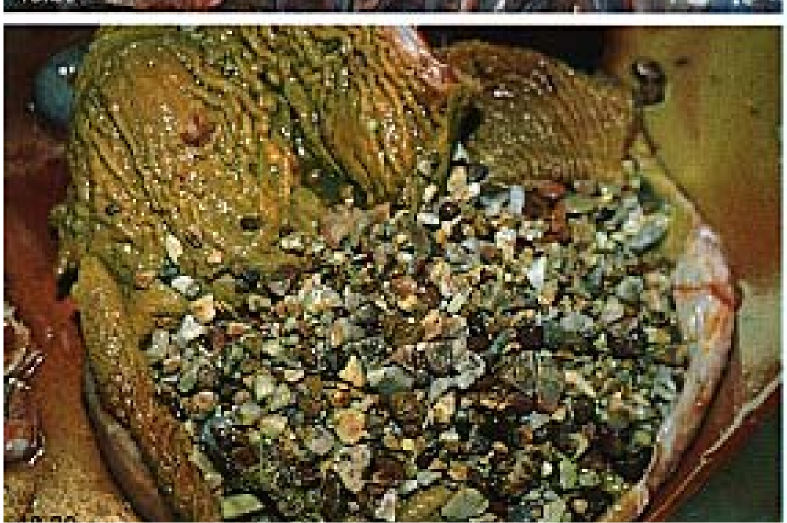
48.29



48.30

48.31

Ratites

Color 48.26
Massive fat accumulation is shown in the coelomic cavity of an adult ostrich that was being fed an excess quantity of grain. The ostrich died from heart and liver failure secondary to obesity.

Color 48.27
In this intestinal tract of a four-week-old ostrich, note that the large intestine is much longer than the small intestine and is divided into a thin-walled, smooth section caudally. The cloaca is distended with urine. duodenum (d), pancreas (arrow), Merckel's diverticulum (m), cecum (c), large intestines (i), cloaca (cl).

Color 48.28
a) A nail penetrated through the ventricular wall of a rhea. **b)** Hardware disease in a six-month-old ostrich that ingested two nails. One nail has penetrated the proventriculus, mesentery and lodged near the acetabulum. This bird died of inanition.

Color 48.29
Large consolidated hematoma (arrow) was caused by the passage of a nail through the proventriculus. The mesentery has effectively contained the nail within the lumen of the hematoma. This was an incidental finding at necropsy. ventriculus (v) and large intestines (i).

Color 48.30
Rock impaction in the ventriculus of an eight-month-old ostrich that had been recently moved to a new paddock.

Color 48.31
Necropsy exposure of an ostrich. The incision demonstrates an approach to the proventriculus (p). Note the fascial membrane that separates the proventiculus from the intestines.

pathologic finding. Clinical changes usually indicate an advanced disease and recovery is unlikely. Endoscopy can be used to document air sac lesions and to perform biopsies or obtain cultures from diseased tissues. Serologic tests for *Aspergillus* sp. may prove to be useful in ratites but have not been adequately investigated.[61]

Flock management should be carefully evaluated to prevent other cases. Appropriate antibiotics to treat secondary bacterial pathogens and aerosolized and oral antifungals are suggested therapies, but are rarely effective. Aspergillosis is best prevented by reducing a bird's access to organic debris, reducing stress, minimizing the use of antibiotics and providing adequate ventilation.

Candidiasis of the proventriculus, esophagus and mouth may occur in ostrich chicks. Infections are most common in birds maintained in damp environments or secondary to proventricular impaction or the long-term use of antibiotics. Chlorhexidine, ketoconazole or nystatin have been discussed as effective therapies. Candidiasis may be prevented with good hygiene and a dry environment.

Mycoplasma and Chlamydia

Mycoplasma spp. infections in ostriches are an enigma. Serologic tests designed for poultry occasionally yield positive results, but the interpretation of these results is speculative. Mycoplasma have been identified on culture as well, but there is no firm evidence to implicate these microbes as the cause of clinical disease in ratites. Ostriches in the United States are gradually falling under stricter guidelines for interstate transport including screening for mycoplasma, and the need for an accurate diagnostic test is imminent.

A pigeon-like isolate of chlamydia has been diagnosed in rheas and ostriches.[25a] Birds raised on an open range may be at risk. Treatment of chlamydial infections in ratites with chlortetracycline (CTC) at the rate of 400 g per ton of feed for 45 days may be expected to be effective (see Chapter 34).

Parasites

Ratites are susceptible to a number of parasitic infections. The most important parasites are listed in Table 48.3.

Protozoa

Intestinal protozoa including *Cryptosporidium, Toxoplasma, Histomonas, Giardia* and *Trichomonas* spp. have been discussed as causes of severe and transient diarrhea in ratites (see Chapter 36). It is unclear the extent to which these organisms cause disease in ratites, but they should be treated when identified. Coccidiosis is a common finding in emu chicks, but is not confirmed as a clinically important problem in ostriches. Asymptomatic leukocytozoon infections are common in ostriches in Africa.

Cestodes

The tapeworm *Houttuynia struthionis* is abundant on South African ostrich farms and has been seen sporadically in the United States. Chicks are particularly susceptible, becoming unthrifty with high mortality rates. Adults are unaffected. A diagnosis is made by identification of parasite segments passed in the feces. The intermediate host is unknown, but infestations can be controlled with regular use of fenbendazole at 15 mg/kg orally.[17]

Nematodes

The wireworm *Libyostrongylus douglassi* is an economically important parasite of ostriches. The adult worms and third and fourth stage larvae reside in the glandular crypts of the proventriculus. The resultant inflammation obstructs gastric secretions and inhibits digestion. Food decays within the stomach, and the disorder is referred to as "vrotmaag" or rotten stomach. Eggs may survive in dried feces for over a year, and the infective third stage larvae develop to maturity in 29 days. Diagnosis is made by identification of the trichostrongyloid-type egg in the feces. The eggs can be confused with those of the harmless cecal worm, *Codiostomum struthionis*. Levamisole hydrochloride dosed at 30 mg/kg is routinely administered monthly to chicks and four times per year to adults. Fenbendazole (15 mg/kg)[17] and ivermectin (0.2 mg/kg) are also considered to be effective.

Tracheal worms *Syngamus trachea* have been associated with hemorrhagic tracheitis in emus (Color 48.13). The ostrich guinea worm, *Dicheilonema spicularum,* is a large filarial worm found in the subperitoneal connective tissue. Females may exceed 2.1 m in length and 2.5 cm in diameter. They are frequently found in free-ranging ostriches where they induce no clinically detectable problems.

Filariid nematodes *Chandlerella quiscali* were removed from the spinal cord and lateral ventricles of the brain of emus with clinical signs that included

TABLE 48.3 Protozoa, Helminths and Arthropods Reported in Ratites

	Ostrich	Rhea	Emu	Cassowary
Protozoa	*Aegyptianella pullorum, Blastocystis* sp., *Cryptosporidium* sp., *Giardia* sp., *Isospora struthionis, Histomonas meleagridis, Leukocytozoon struthionis, Plasmodium struthionis, Toxoplasma gondii, Trichomonas* sp.	*Histomonas meleagridis, Toxoplasma gondii*	*Eimeria* sp., *Giardia* sp., *Trichomonas* sp.	*Toxoplasma gondii*
Trematodes	*Philophthalmus gralli*			
Cestodes	*Houttuynia struthionis*	*Chapmania tauricollis, Houttuynia struthionis*	*Davainea australis*	*Davainea casuarii, Davainea infrequens*
Nematodes	*Baylisascaris procyonis, Codiostomum struthionis, Dicheilonema spicularum, Libyostrongylus douglassi, Paronchocerca struthionis, Struthiofilaria megalocephala*	*Ascaridia orthocerca, Deletrocephalus dimidiatus, D. cesarpintoi, Dicheilonema rheae, Habronema incerta, Odontospirura zschokkei, Paradeletrocephalus minor*	*Baylisascaris* sp., *Chandlerella quiscali, Dromaestrongylus bicuspis, Syngamus trachea*	
Gnats	*Simulium* spp.			
Fleas	*Ctenocephalides felis*			
Louse flies	*Struthiobosca struthionis*			
Lice	*Struthiolipeurus struthionis*	*Struthiolipeurus andinus, S. nandu, S. renschi, S. stresemanni*		
Ticks	*Amblyomma gemma, A. hebraeum, A. lepidum, A. variegatum, Argus persicus, Haemaphysalis punctata, Hyalomma albiparmatum, H. dromedarii, H. impeltatum, H. luscitanicum, H. marginatum, H. truncatum, Otobius megnini, Rhipicephalus deltoides, R. guilhoni, R. sanguineus, R. turanicus*	*Amblyomma parvitarsum, Ixodes brunneus*		*Amblyomma papuanum*
Mites	*Gabucinia bicaudata, G. sculpturata, Paralges pachynemis*	*Gabucinia bicaudata, Paralges pachynemis*		

torticollis, ataxia and abnormal gait followed by recumbency and death.[3] Only two- to five-month-old emus were affected, with adult and yearling emus apparently resistant. The cause of apparent resistance in these older birds is unclear. It is possible that older birds were immune to the parasite or that the neural tissues were less severely damaged. Emu chicks that were affected by the parasite were repeatedly tested for circulating microfilaria, but all birds were negative during a three-month period of sampling. Several birds with mild neurologic signs were followed for a six-month period and never developed a microfilaremia.

Grackles are the normal host for *C. quiscali*, which is transmitted by *Culicoides* sp. Following a migration of unknown duration, the larvae enter the brain and spinal cord and migrate toward the lateral ventricles of the brain where the adults mature and produce microfilaria. When the microfilaria are ingested by gnats, the life cycle of the nematode is completed. Infected Great-tailed Grackles and *Culicoides* sp. were collected from farms with affected emus.

Prevention of this cerebrospinal nematode may be possible by control of the vector, elimination of the environmental conditions conducive to transmission of the parasite and prevention of larval migration. Treatment would be expected to be ineffective once the larvae enter the CNS because anthelmintics normally do not pass the blood-brain barrier. Several emu farms use anthelmintics in an attempt to arrest larval migration, but it is unclear if this preventative is effective. Although this parasite has not been reported in the ostrich, it should be considered a threat until proven otherwise.[3] *Baylisascaris* sp. has also been shown to cause neurologic signs in ratites (see Chapter 36).

Arthropods

Ostriches are subject to infestation by a variety of external parasites, both host-specific and indiscriminate. The ostrich louse, *Struthiolipeurus struthionis,* is commonly found on ostriches worldwide. These

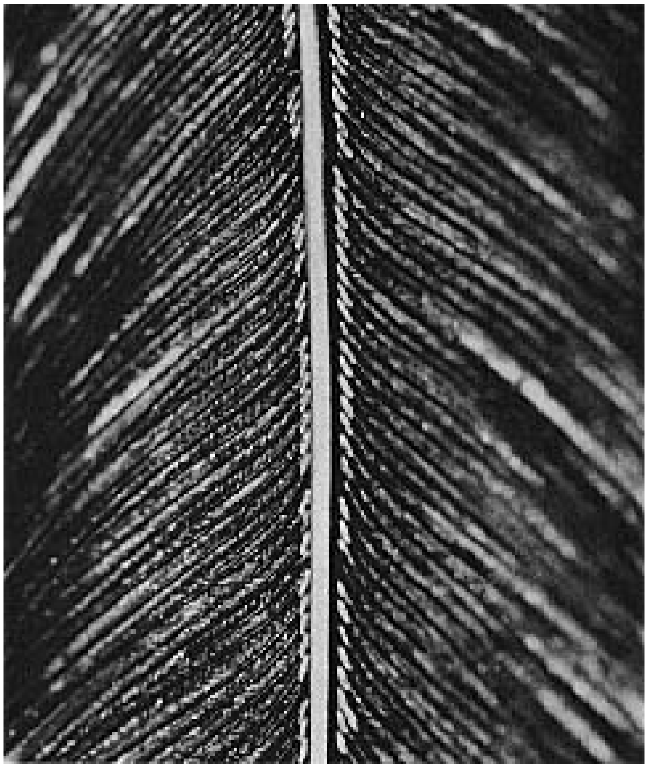


FIG 48.21 Nits of the ostrich feather louse (*Struthiolipeums struthionis*) can be seen along both sides of the rachis (courtesy of James Stewart).

chewing lice feed on skin scale and feathers and by removing the barbules, make the feather coat appear thin and tattered. Infestation is easily diagnosed by identification of the nits glued to the barbs along the shaft of feathers, particularly under the wing (Figure 48.21). The ostrich louse fly, *Struthiobosca struthionis*, is a minor annoyance to African birds but may also bite the human caretakers. Quill mites may be found within the feather shaft or along the external groove of the shaft.

Ticks from a variety of mammalian, avian and reptile hosts have been reported to infest ostriches. They may cause blemishes on the skin that reduce its commercial value. More importantly, many of these tick species are potential vectors of livestock diseases. The discovery of ticks on imported ostriches released from private quarantine in 1989 caused the United States Department of Agriculture to impose an immediate ban on the importation of ratites.[40] Following considerable study and public debate, the importation of ratites was reapproved in 1992 with modified quarantine restrictions and guidelines.

External parasites may be treated with monthly applications of five percent carbaryl or two to four percent malathion in either powder or liquid form. Benzene hexachloride is highly toxic to ostriches and should be avoided. Quill mites are responsive to monthly treatments with ivermectin at standard doses.[29]

Respiratory Problems

Upper respiratory tract infections may be caused by bacteria, mycoplasma, fungi (*Aspergillus* spp.) or possibly viruses (see Color 22). Clinical signs are characterized by ocular or nasal discharge, and birds may be dyspneic and appear unkempt. Treatment is based on the etiology. Affected birds should be isolated immediately and flockmates should be carefully observed. Adequate ventilation and reduced airborne debris are necessary to prevent the spread of infections in the flock. Air sacculitis and pneumonia are most commonly caused by bacteria or *Aspergillus* spp.[61] A sunken sinus syndrome has been described in emus secondary to sinusitis (see Figure 26.8).

Ocular Problems[53,54]

Cataracts are frequently seen in ostriches. They are more common in older birds, but may occur also in chicks only a few months of age (Color 48.10). Unilateral cataracts are of little clinical importance. Bilateral cataracts must be removed, and surgery has been successful.

Lacerations, abrasions or ulcerations to the eye may cause epiphora or blepharospasms. Foreign bodies may or may not be found. The discharge in these cases is usually clear and often is present in only one eye. Diagnosis is made via physical examination and fluorescein-staining of the eye. Standard ocular therapies used in other birds are effective in ratites (see Chapter 26). Subpalpebral flukes may cause chronic epiphora in birds raised in moist areas.

Eye infections generally cause the production of a purulent discharge and will usually be bilateral (Color 48.11). Cytology and cultures are necessary to determine the etiologic agent. Ocular discharges are common with upper respiratory infections. A blocked nasolacrimal duct is recognized clinically by epiphora and swelling within the lower lid. The lacrimal duct can be cannulated and flushed to determine if a blockage has occurred and to correct the problem.

FIG 48.22 Feather picking in ratites may be caused by malnutrition or overcrowded, small or barren enclosures (courtesy of James Stewart).

Feather Loss (With or Without Skin Involvement)

Bacterial folliculitis has been described in ratites and is most commonly caused by *Staphylococcus* spp. The ingestion of parsley was associated with a photosensitization reaction in an ostrich (see Chapter 37). Poxvirus may cause crusty proliferative lesions on the head, periocular area and legs. Lice or mites can damage the skin and feathers of ratites. Adult lice and their eggs can be visualized along the shaft of the feather (Figure 48.21). Five percent Sevin dust is an effective treatment.

Feather picking can be caused by overcrowding, excessive exposure to light at night and a lack of available food. Feather picking is common in adult ostriches maintained in small, barren paddocks and may be a reflection of malnutrition or environmental stresses (Figure 48.22). Large deplumed areas of skin are subject to sunburn. These birds will usually respond to an enriched environment, especially green pasture. In one case, a large, reflective, chrome plate (mirror) was used to distract a male and prevent him from feather picking a hen.

Neural Diseases

Toxins that can cause neurologic lesions in ratites may include plants, oil, grease and insecticides. Endotoxins produced by bacteria can lead to severe ataxia. Infectious causes of neurologic problems include viruses, bacteria, fungi or parasites. Paramyxovirus, EEE virus and Newcastle disease virus have all been associated with neurologic signs in ratites. Overheating may cause ataxia or seizures. The normal body temperature of ratites is approximately 103°F. Birds, particularly chicks, that are panting with extended wings are overheated. Treatment should include cold water baths. Hypoglycemia can cause ataxia and tremors in anorectic neonates. Oral or IV dextrose as well as tube feeding with high-carbohydrate diets three to four times daily is usually curative.[62]

Hatchery Management

The Egg

The general principles of egg management for ratites are no different than those for other avian species.[52,56] Hatchability exceeding 90% of fertile eggs should be expected with well managed commercial ostrich operations. The maintenance of accurate records, including the analysis of unhatched eggs, is an absolute necessity for the elucidation of incubation and hatchery problems.

There is an optimum size for all eggs. Unusually small eggs lose excessive moisture during incubation and produce small dehydrated chicks that rarely survive. Conversely, large eggs produce weak, edematous chicks that frequently fail to hatch (Color 48.2).

The shell of an egg balances two opposing functions: the egg shell must allow the free exchange of oxygen, carbon dioxide and water vapor, yet inhibit the penetration of infectious agents. Thick shells and shells with low porosity inhibit gas and water vapor exchange, while thin, highly porous or defective shells may lose excessive water vapor and readily allow bacterial penetration (Color 48.1). Shell quality is influenced by nutrition, disease and genetics, as well as by the conditions of the nest site and the egg handling methods.[27] Porosity of the shell is a heritable trait, and each hen will have unique eggs.[27] Strict sanitation is essential to maintain hatchability.

The physical health of the hen can also affect egg quality. Pathogens colonizing the reproductive tract may result in a thin or absent shell, or may be a source of infection for eggs that appear otherwise normal. Eggs with rough-textured surfaces, ridges, a lack of a mucin coat or soft shells may indicate metritis. Soft shells may also occur as a result of dietary deficiencies. Yolkless eggs may be caused by metritis, deposition of yolk into the peritoneal cavity or abnormalities of the ovary. Double yolks are postulated to occur with abnormal egg passage through the oviduct.

The physical characteristics of an egg have a strong genetic basis. Commercial poultry have been selected to produce a nearly uniform egg that results in uniform incubation and hatching characteristics. Ostrich breeding stock from free-ranging linage will retain their full natural variability in egg quality and specific incubation requirements.

Ratites lay their eggs on the ground, creating a potential for egg contamination by infectious agents. Management practices require that a clean nest such as sand or straw be available for egg laying and that eggs to be artificially incubated are collected and disinfected promptly. Ostriches typically lay in the evening hours, while emus frequently lay shortly after dark. Eggs should be gently collected from the nest and transported by hand to the preparation area. Excessive jarring of the egg contents or damage to the shell can be fatal to the developing embryo.

Cold storage of eggs allows the use of efficient batch incubation and brooding systems and is a routine practice on large farms. Disinfected eggs are maintained up to seven days between 12.8°C (55.0°F) and 18.3°C (65.0°F) and near 75% relative humidity. Egg-turning is not necessary for storage periods of less than one week. Ideally, eggs are stored directly in the incubation trays and the entire tray may then be loaded into the incubator racks.

Egg washing is a controversial issue in ratite production. It is better to provide the breeding pair with a clean, dry area in which to lay eggs rather than attempting to clean or disinfect dirty moist eggs. Wet washing of eggs involves the use of warm dilute solutions of commercial quaternary ammonia or phenolic disinfectants, chlorhexidine or sodium hypochlorite. Dry washing is performed by using a soft bristle brush to remove gross organic debris and misting the egg with a disinfectant. The dried mucinous cuticle of ratite eggs is particularly well developed and serves as a significant barrier to bacterial penetration. It has been suggested that the wet washing of clean eggs removes this cuticle and increases the incidence of infection over that of a dry cleaning method.

Incubation

The incubation temperatures and humidities required for ratite eggs are lower than those used for other avian species (Table 48.4).[52,56] The incubators currently available for ratite production vary in quality. Many of them are inadequate in several aspects, and faulty incubators are a common cause of hatchery problems. Incubators should generate a uniform temperature throughout the cabinet and maintain the temperature within narrow limits, preferably one- or two-tenths of a degree. Late-term embryos are particularly sensitive to decreasing temperatures, and it is imperative that backup electrical power be available for incubation equipment so that eggs are not chilled if a power outage occurs.

Ventilation is the amount of fresh air brought into the incubator. Circulation is the amount of air movement within the incubator. The minimum ventilation requirement for the incubation of ostrich eggs is calculated at 50 cubic feet of fresh air per hour per 100 eggs. Inadequate ventilation causes an accumulation of carbon dioxide and severely reduces hatchability. Ventilation also controls the humidity in most incubators. Circulation functions to maintain uniformity of temperature, humidity and gas levels throughout the incubator cabinet. Rapid air circulation is critical with the large ratite eggs to effectively dissipate the high temperature and humidity that develops at the egg surface.

Ratite eggs should be incubated in the vertical position with the air cell end upward. Embryonic malposition 2 (head at opposite end from air cell, see

TABLE 48.4 Breeding and Hatchery Parameters for Ratites

Species	Eggs/year	Egg wt. (g)	Temperature	Humidity (%)	Period (days)
Ostrich	40-60	1300-1700	36.0-36.4°C (96.8-97.5°F)	20-40	41-43
Rhea	40-60	400-700	36.0-37.2°C (96.8-99.0°F)	55-70	36-41
Emu	20-40	500-700	36.0-36.7°C (96.8-98.0°F)	25-40	50-57
Cassowary	3-10	500-700	36.0-36.7°C (96.8-98.0°F)	55-70	47-53

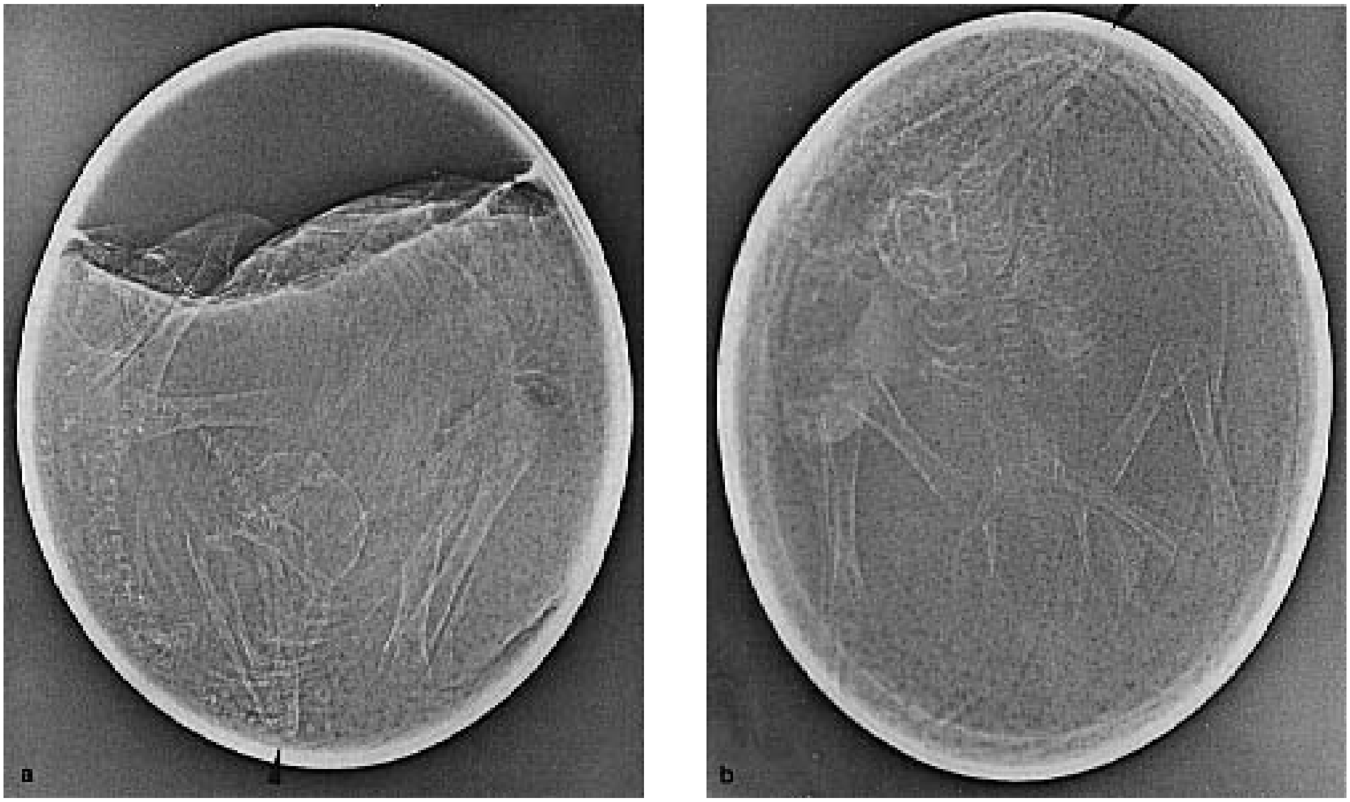


FIG 48.23 Xeroradiographs of ostrich eggs. **a)** Notice the dead embryo skeleton positioned with its head and the tip of its beak (arrow) oriented opposite the air cell end of the egg. The embryo died late in development. The air cell at the top of the egg has not been penetrated. **b)** Notice the dead embryo skeleton in a normal position after penetration of the air cell with the tip of the beak (arrow). The embryo did not pip the shell, and the air cell has been displaced by the embryo (courtesy of David Ley, reprinted with permission[38]).

Chapter 29) is especially common in ostrich eggs incubated in the horizontal position and results in poor hatchability. The incidence of malposition 2 increases from 3% among eggs incubated vertically to 16 to 20% among eggs incubated horizontally (Figure 48.23).

Eggs are rotated during incubation in order to stir the liquid layers of nutrients and waste products around the developing embryo. Inadequate turning increases both embryonic and post-hatch mortality. Following poultry protocol, the egg rotation angle should be 45° from vertical, shifting a minimum of six times per day.

Incubator humidity settings vary with the type of incubator as well as the size and shell characteristics of the eggs. Humidity is generally set at 20 to 40% to achieve the desired evaporative water loss during the course of incubation (13 to 14% for ostrich eggs and 10 to 11% for emu eggs). Egg monitoring during the course of incubation should include determination of weight loss and candling for embryonic development. The weight loss of eggs is fairly linear throughout incubation, and weighing an egg weekly can be used to monitor an embryo's development and guide adjustments in humidity.

It is important to determine when mortality occurred in a dead embryo. Losing ten per cent of fertile eggs during incubation is considered normal, with peaks of loss at 3 to 4 days (organogenesis) and 40 days (respiration change) of incubation. Embryonic death at other times may be caused by incorrect incubation parameters, nutritional deficiencies in the egg, infectious agents, genetic abnormalities, improper egg storage or toxins. If candling indicates that an egg is not developing after ten days of incubation, the egg should be opened to determine if the egg was infertile if early embryonic death occurred (see Chapter 29). The embryonic disc floats up and can be examined for any development. This is best done early in the incubation cycle rather than at day 43, because embryos that die early in incubation decompose rapidly.[27]

Fluctuating incubation temperatures result in unthrifty chicks. High temperatures result in an early hatch, small dry chicks, increased embryonic mortality and malformations (Color 48.2). Low temperatures result in soft, large, weak chicks with a delayed hatch. Excessive humidity (inadequate moisture loss from the egg) may cause a delayed hatch, small air cells, wet edematous chicks and mild degeneration of the leg muscles (Color 48.4). Humidity that is too low (excess moisture loss from the egg) results in large air cells, increased malpositions due to sticky chicks (dry albumen), albumen plugs in the non-air cell end of the egg and weak, dehydrated chicks with poor survivability.

Hatching

Ratite eggs are transferred to a hatcher with the same temperature and humidity settings as the incubator three to five days prior to the anticipated date of hatch. Hatching should be a gradual process that gives the chick time to switch from chorioallantoic to pulmonary respiration. The yolk sac with the blood from the chorioallantois is absorbed, and the navel closes during the last 24 hours of incubation.

The social facilitation of pipping and hatching is strongly developed in ratite chicks, and light, sound and motion help stimulate a hatch. Daily candling of ostrich and rhea eggs allows for careful monitoring of the chicks' progress, and assisted hatches are a common management procedure. The pigmented shells of cassowary and emu eggs prohibit effective candling, and various techniques of percussion and auscultation have been used to evaluate embryo development in these species. Many clients are anxious to help a chick out of the shell and begin to assist with the hatching process prematurely. This procedure results in chicks with a high incidence of yolk sac infections (see Chapter 29).

Chick Management

Ratite chicks are precocial, hatching with a full coat of natal feathers, open eyes and the ability to stand within hours.[53] They should be removed from the hatcher at one to two days of age and placed in brooder pens with other neonates that do not vary more than three weeks in age. Eating and drinking are behaviors that must be learned from older birds, and dehydration or starvation are common in chicks that are housed alone. Bin feeders and automatic water units designed for poultry and gamebirds can be effectively used in recently hatched young. Numerous commercial starter and grower rations are available for ratites. Chicks should be expected to lose weight for three to five days following hatch and then to begin a steady increase in weight gain.

Young chicks should have access to supplemental heat that can be provided by infrared lamps, heated floors or space heaters. Chicks should be maintained at decreasing temperatures with age (90°F one to two weeks, 85°F to 12 weeks).[31] Indoor flooring should be inedible, provide good traction and be easily cleaned. Brushed concrete, with or without perforated plastic or rubber matting, is commonly used. In order to prevent gastric impactions, hay or wood chips should not be used. Adequate ventilation is essential in the brooding area. The general consideration for air circulation is 0.012 cubic feet per minute/per pound of bird for each degree F. For example, if the temperature is 70° and ten birds weighing 50 lbs each are in the space (500 lbs total), then the air exchange requirement is 0.012 x 500 x 70 or 420 cubic feet per minute.[61]

As soon after hatching as ambient temperatures permit, chicks should be moved to outdoor grazing areas with fencing that is low to the ground and with holes no larger than 2.5 cm. Chicks should be considered cold-intolerant and provided supplemental heat in cold weather until they are six months of age. Chicks should be housed with birds of similar age to prevent injuries. Young chicks require constant attention to keep them tame and to quickly detect any developmental problems.

To reduce the chances of foreign body ingestion, chicks should be carefully monitored during their initial introduction to a pasture. Using a grassy area that has been well groomed (cut to three inches) and placing chicks with slightly older birds to serve as feeding models are the best techniques to introduce chicks to pasture. Initial introduction periods should be 10 to 15 minutes in length with daily doubling of the time in the paddocks.

Chick Problems

"Wet" is the term applied to chicks that have not lost sufficient weight during incubation and are consequently edematous at hatching (Color 48.2). These birds may or may not be capable of an unassisted hatch. Diuretics have been suggested to reduce the edema but they are rarely necessary. Most birds will lose the excess water several days after hatch.[61] This condition can be caused by several factors including: 1) Large ostrich eggs (greater than 1700 to 1800 g) that do not have sufficient surface area to allow adequate water loss; 2) Poor egg shell quality, such as low pore density or excessive thickness; 3) High incubation humidity. Relative humidity in the incubator should be 20 to 25% as a starting point. Adjustments must be made to achieve a 13 to 15% egg weight loss throughout incubation.

"Sticky" Chicks

This condition occurs when the inner shell membrane is excessively dry, causing the chick to stick to the membrane. Assisted hatching is mandatory in these chicks or they will not survive. To correct this problem, the humidity in the hatcher must be increased (perhaps as high as 80 to 90% relative humidity) to a point that allows the membrane and its underlying chorioallantois to remain pliable and easy for the chick to tear. This problem can occur if the humidity in the incubator is too low.[61]

External Yolk Sac

If the umbilicus does not close properly, the yolk will protrude from the abdomen to varying degrees (Color 48.2). High incubation temperatures (early hatch), poor gas exchange (may occur with high altitude or high humidity) and egg infections are thought to be the principal causes of improperly absorbed yolk sacs. The cause can best be determined by carefully evaluating the incubation and hatching parameters and culturing affected embryos. In mild cases, the yolk can be placed in the abdomen, the umbilicus covered with antibiotic ointment and gauze, and the abdomen wrapped with self-adherent bandage. Systemic antibiotic therapy should be initiated immediately. If a large quantity of the yolk sac is externalized or the umbilicus has sealed (Color 48.2), the yolk sac should be surgically removed. The prognosis for chicks with externalized yolk sac is poor.[61]

Retained Yolk Sac

Chicks that fail to absorb the yolk sac are generally weak, depressed and may peck erratically at the air with or without their eyes closed. A distended abdomen in a depressed chick less than two weeks old is a characteristic finding (Figure 48.24). Additionally, they may display a characteristic "S"-curve in the neck and feign eating without ingesting any food. These birds will continue to lose weight beyond the normal weight loss seen the first four to five days after hatch.

Retained yolk sacs are thought to be caused by improper incubation parameters, malnourished hens or infected eggs (Color 48.7). Improper incubation temperature as well as inappropriate gas exchange (poor circulation or ventilation) between the embryo and the air surrounding the shell can cause retention of the yolk sac. Ventilation defects are thought to have their most profound effects late in the incubation process and may be most common when an incubator is filled to near capacity, which causes an accumulation of CO_2 if the ventilation is inadequate. Incubation problems are suspected if infectious agents cannot be cultured from the yolk sac of affected chicks.

Inflammation of the umbilicus shortly after hatching can be an early warning sign of an impending yolk sac infection (Color 48.6). Affected chicks have subnormal weight gains over a two- to three-week period and intermittently appear depressed or chilled. These chicks may absorb a portion of their yolk sac and may not have a severely distended abdomen until late in the disease process. Applying an antibiotic ointment to the umbilicus of recently hatched chicks may help prevent infections.

Retained or infected yolk sacs may represent 15 to 40% of a chick's total body weight and should be surgically removed in conjunction with the administration of broad-spectrum antibiotics. *E. coli* is frequently cultured from retained yolk sacs (Color 48.7). To remove the yolk sac, the chick is placed in dorsal recumbency and the abdomen is prepared for surgery. The skin is incised circumferentially around the umbilicus and the incision is extended transversely at the three and nine o'clock positions to the lateral distance required to allow easy removal of the intact yolk sac. The body wall is then incised in a corresponding pattern being careful to not damage the underlying yolk sac. The yolk sac is exteriorized by placing gentle traction on the umbilical stump. The

CLINICAL APPLICATIONS

Management Tips for Chicks[61]

- Feed pellets with 19 to 21% protein content supplemented with fresh grazing as soon as possible.
- Weigh chicks daily for the first two to three weeks after hatching. Weight gain is a good indicator of a healthy chick, and weight loss often precedes clinical signs of disease.
- The single most important requirement for growing ratites is ample exercise.
- Exposure of chicks to infectious agents can be reduced by restricting access of all visitors, utilizing sound hygiene procedures and caring for neonates in a youngest to oldest pattern.

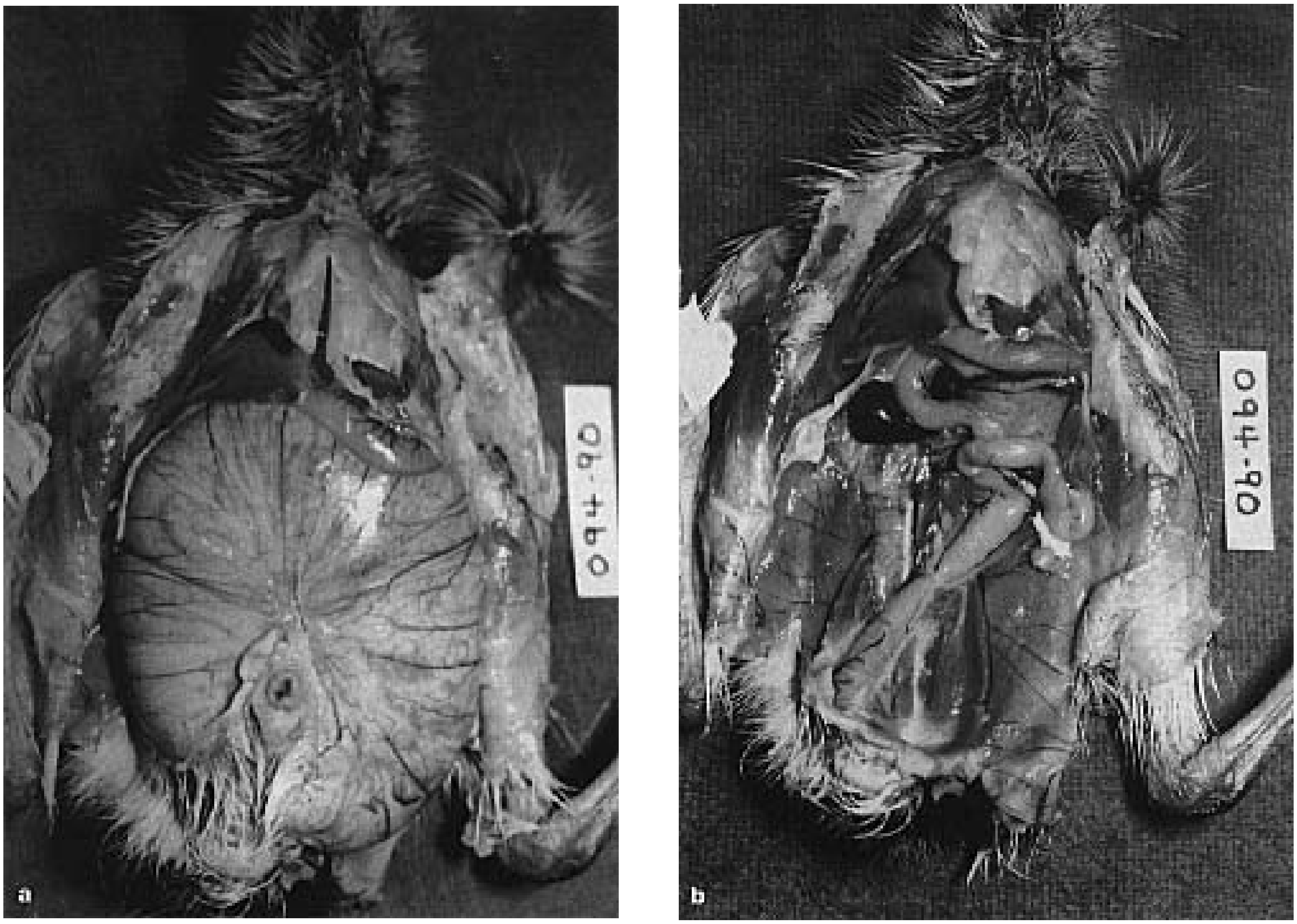


FIG 48.24 **a)** Retained egg yolk in a six-day-old cassowary chick that developed dyspnea and an inability to walk. **b)** The yolk sac has been removed showing cranial displacement of the abdominal viscera (courtesy of Richard Cambre, reprinted with permission J Zoo Wildl Med 23:1992).

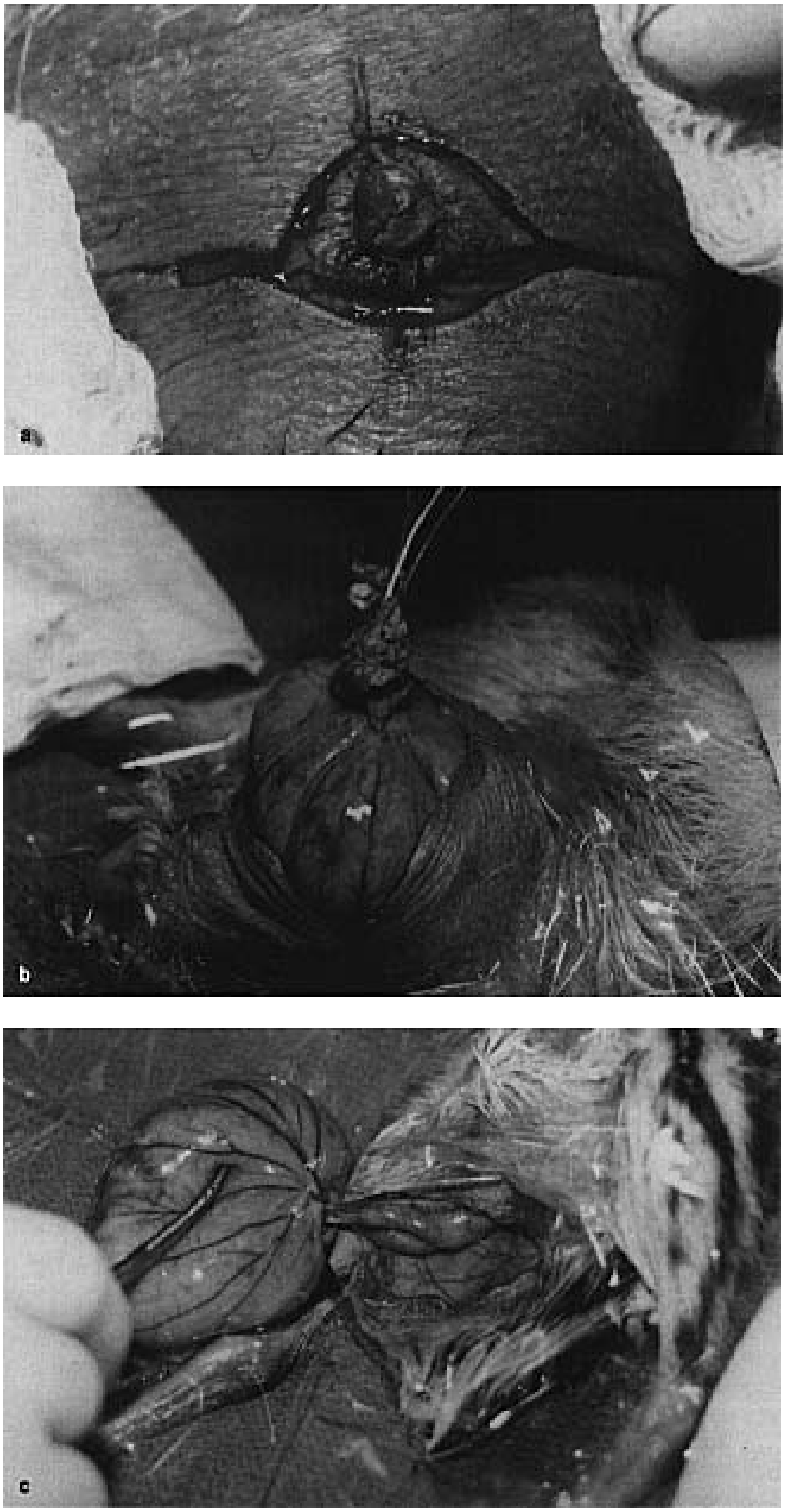
yolk stalk is clamped, clipped or ligated just distal to the intestine to allow the stalk to be transected and the yolk sac to be removed. The body wall is closed with a monofilament, absorbable material in a simple continuous pattern. Broad-spectrum antibiotic therapy is indicated pending culture results. Trimethoprim-based antibiotics can be administered until the results of yolk sac culture and sensitivities are available. Some chicks begin to eat and gain weight within a day or two of surgery while others require nutritional support by tube-feeding for several days before they resume normal growth.[10]

Congenital Disorders

Congenital anomalies described in ostrich chicks include albinism, leucism, melanism, pied, prognathism, crossed beak, choanal atresia, microphthalmia, blindness, meningocele, crooked neck, hernia, schistomus reflexus, polydactyly, third leg, micromelia and hermaphroditism. Congenital defects in chicks may be attributable to genetics, the effects of nutritional deficiencies or teratogens in the laying hen, or may be caused by inadequate egg handling and incubation procedures. The incidence of these problems is usually sporadic and the specific etiology obscure.

Cloacal Prolapse

Prolapse of the cloaca may occur in neonates. This condition is most common in birds less than four weeks of age and has been associated with distension of the abdomen (ie, excessive water-drinking on hot days, proventricular impactions, retention of the yolk sac) and tenesmus. Mild cases can be resolved by simply replacing the cloaca through the vent. More severe cases require retention sutures for one to three days (see Chapter 41).



Stress

Stress is an extremely important consideration in ostrich management and is generally under-rated or ignored. It is the most frequent primary cause of chick mortality. Ostriches are highly social animals that do not readily adapt to change. Young chicks require a stable social group that might include a parental figure in the form of a natural parent, older counselor chick or a human substitute. Complete social isolation of young chicks is tantamount to death.

Management systems in which chicks are transferred through a series of pens are disturbing to the birds. Relocating or mixing chicks from different groups may alter the social structure causing some chicks to be harassed or rejected by dominant birds. These abandoned chicks may exhibit neurotic pacing, inadequate or inappropriate ingestion behavior, intermittent diarrhea and stunting. Chicks shielded from constant human activity become flighty, adjust poorly to captivity and may mature to become substandard producers. Environmental and social stability, combined with the taming of young chicks through continuous human presence, are among the most important components of a successful ostrich management program. Gradually introducing chicks to a new area or situation will help maintain the group stability and reduce stress.

FIG 48.25 **a)** To remove a retained yolk sac, a circumferential incision is made around the umbilicus extending laterally at the three and nine o'clock positions. **b)** The yolk is exteriorized by placing gentle traction on the umbilical stump. **c)** The exteriorized yolk sac showing the vascular and yolk sac connection to the small intestines. These structures are ligated close to the small intestines (courtesy of Richard Cambre, reprinted with permission J Zoo Wildl Med 23:1992).

Musculoskeletal Disorders of Chicks

Rolled toe in ostrich chicks is a problem frequently seen in backyard operations the first few day after hatching (Color 48.22). The distal portion of the main toe is rotated off of the centerline (Figure 48.25).

In poultry, riboflavin deficiencies cause damage of the peripheral nerve trunks and paralysis resulting in curling of the toes and leg weakness; however, in ostriches, rolled toe syndrome seems to be caused by genetic abnormalities, incubation problems or inappropriate substrates during the first week of life while the phalanges are mineralizing. Brooder pens with soft surfaces or wire mesh allow the toe to flex and roll medially. A firm flat surface such as packed dirt or concrete induces proper toe formation.

Rolled toe deformities may be corrected with a variety of simple splints, but become more difficult to resolve as the chick matures. A splint can be applied by wrapping tape around the toe in a direction opposite to the deflection (Figure 48.26). A tongue depressor can be incorporated into the plantar surface of the toe on the final wrap to provide extra stability. Correction of this problem in older chicks requires surgical intervention.

Rotational and angular deformities of the legs are a common problem in the rearing of all ratite chicks and should be viewed primarily as a management problem (Figure 48.27). This condition occurs when one or both legs rotate laterally at the distal tibiotarsus causing the toe to point laterally (Color 48.23). Chicks from a particular breeding pair may have a high incidence of deformities under conditions in which other chicks are raised satisfactorily, implicating genetics as a contributing factor. Chicks raised on slick and slippery surfaces have severe leg deformities. Classical rickets is often seen, with elongated metaphyses of the long bones and ricketic rosaries along the ribs. These chicks respond favorably to adjusted dietary levels of calcium, phosphorus and vitamin D_3. Leg problems associated with inadequate or imbalanced levels of these compounds are exacerbated by high levels of dietary protein.

Leg deformities are more common in birds that are pushed to grow too fast (high-protein, high-fat diets) combined with reduced exercise, and are maintained in areas with poor footing (sand, straw, Astroturf). An injury of the growth plate (nutritional, traumatic) will result in a rapid bending or twisting of the bone due to the rapid growth rates. In long-legged altricial species, bone growth in the tarsometatarsus may be six millimeters/day. In precocial young, the growth rate may be two millimeters/day.[34]

An excessive growth rate induced by high-energy diets may cause an unacceptable level of stress on the cartilage in the developing bones, causing deviations in the growth patterns. Weight gains should be linear, and several days of excess weight gain may induce bone deformities.[6] Chicks are best raised on moderate protein diets (20%) in large pastures with plenty of room for exercise. Empirical observation suggests that the incidence of leg deformities is reduced with increased exercise. Bone strength responds to usage, and it is hypothesized that exercise increases circulation to the developing bone and enhances the mineralization process.

Leg deformities in poultry are associated with deficiencies in manganese, zinc, choline, biotin, folic acid, niacin and pyridoxine, and the involvement of these nutrients in certain leg deformities of ratites is likely. Infectious disease, gastric impaction and dehydration often inhibit the appropriate absorption and utilization of feeds and induce secondary skeletal deformities.

Treatment of afflicted individuals can be attempted with a variety of external splints and slings or by derotational osteotomy and fracture repair, but the prognosis is exceptionally poor. Re-rotation following surgery commonly occurs. Proper positioning with external bracing coupled with an immediate decrease in protein content of the feed may be helpful in arresting the process. It is best to apply bracing at night during roost time and to release the chick with the flockmates during the day. It is stressful for chicks to be alone and, with a rotational problem especially, they need extensive exercise. In commercial ratite production, such techniques are economically unfeasible and individuals with marked deformities should be condemned.

Spraddle Leg

Spraddle leg is caused by a deformity in the coxofemoral joint that prevents the legs from being adducted. This condition, usually associated with edematous chicks, is manifested by the legs being directed laterally resulting in the inability to stand. Hobbling the legs with a self-adherent bandage or placing the chick in a restrictive box that forces the legs together is usually effective if initiated immediately after the problem is noted. The problem can be prevented by ensuring proper weight loss of the egg during incubation.[61]

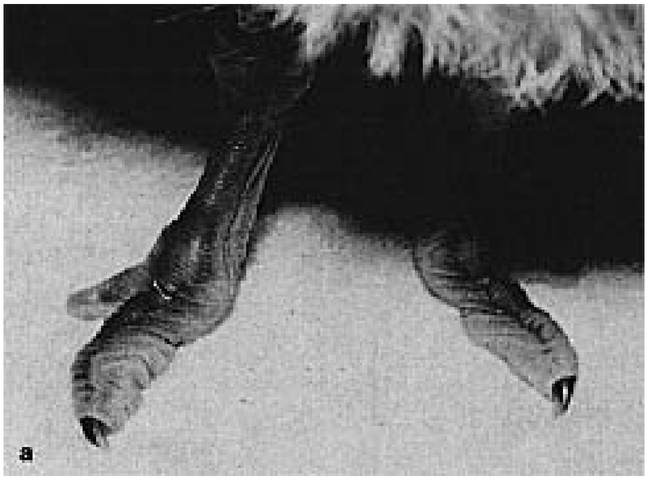


FIG 48.26 Rolled toe deformity **a)** before and **b)** after applying a splint (courtesy of James Stewart).

Ruptured or Slipped Achilles Tendon

Rupture or slippage of the Achilles tendon may occur secondary to valgus or varus deformities of the leg. Manganese deficiency has been suggested as a possible predisposing factor. Management practices must be scrutinized if multiple cases occur in a flock. Feed analysis, feeding frequency, exercise programs, concurrent rotations or angular deformities are areas to evaluate. Surgical repair can be attempted (see Chapter 46).

Septic arthritis is frequently seen in ratites. Numerous gram-negative bacteria, *Mycoplasma* spp., *Staphylococcus* spp. and some fungi have been recovered from affected joints (Color 48.21). These infections may originate from traumatic injuries or can be secondary to septicemia.

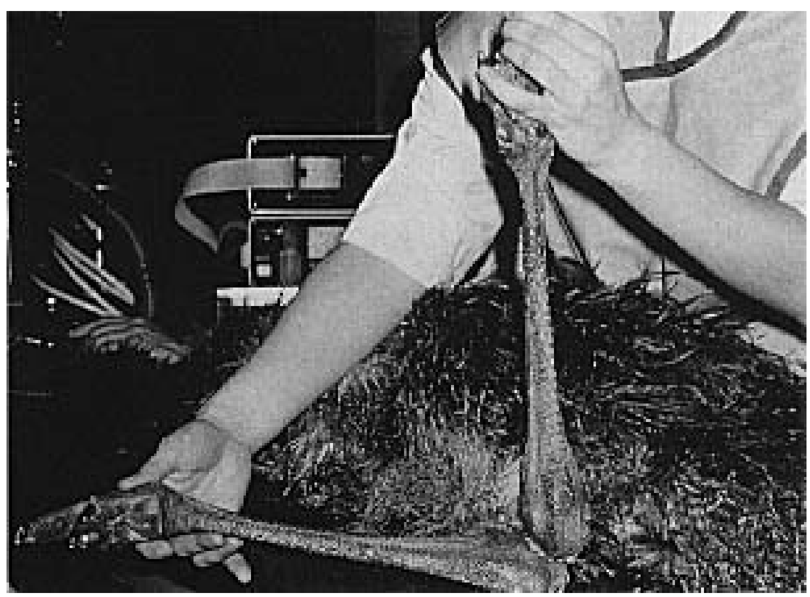


FIG 48.27 Left-sided valgus deformity of the tibiotarsal bone. All angular limb deformities should be considered an indication of management-related problems that need correcting (courtesy of Louise Bauck).

Acknowledgements

Portions of this chapter have been taken in part, with permission, from Fowler ME: Clinical anatomy of ratites. Proc Assoc Avian Vet, 1992, pp 307-309; Blue-McLendon AR: Clinical examination of ratites. Proc Assoc Avian Vet, 1992, pp 313-315; Hicks KD: Ratite production. Proc Assoc Avian Vet, 1992, pp 318-325; Wade JR: Ratite pediatric medicine and surgery. Proc Assoc Avian Vet, 1992, pp 340-353.

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