22.1. Introduction
In recent years, there has been a surge of interest in the propagation of bustards in captivity, in particular the houbara bustard. Captive breeding and restoration programmes for many other bustard species have been established in the Middle East, Europe, Australia, and the former Soviet Union (Bailey et al. 1998c). Producing large numbers of healthy chicks underpins the success of these breeding programmes and as a consequence morbidity and mortality of chicks are potential limiting factors.

Bustard chicks are precocial, hatching with a full coat of natal feathers, open eyes, and the ability to stand within hours (Schulz and Seldon 1996). The bustard chick is protected by a set of defences which enable it to cope with most conditions that could affect it in the wild. By introducing bustards to intensive captive breeding conditions man has initiated their domestication and has changed the environmental conditions, while the immune system of these birds has remained unchanged. As a consequence bustard chicks are vulnerable to a wide range of disorders. This chapter aims to review the common clinical conditions seen in neonatal bustards. For further information the reader is directed to Bailey et al. (1997b) and Naldo et al. (1998a) who reviewed the diseases of bustard chicks at NARC. In addition Bailey and Anderson (2000a) reviewed commonly encountered hatching and post-hatching problems in bustard chicks.

22.2. Species Differences in Chick Mortality Rate
Differences existed in the mortality rate between species of bustard in the review conducted by Bailey et al. (1997b). Buff-crested bustards had the highest mortality rate, 62%, over the three years of the survey. Reasons for the difference between this and other species have not been fully established. Buff-crested bustards are the smallest species bred at NARC and newly hatched chicks weigh on average 25 g (range 22-28 g) compared with 34 g for white-bellied bustards (range 32-37 g), 41 g for houbara bustards (range 32-49 g) and 98 g (range 84-114 g) for kori bustards (Sleigh unpublished data). The smaller size of young buff-crested bustard chicks hinders the detection of clinical signs, the collection of diagnostic samples and the administration of medication. These reasons could delay prompt diagnosis and treatment, and could indirectly account for the higher mortality rate compared with the other species. The level of inbreeding within the buff-crested bustard collection maintained at NARC is also thought to be high (Anderson 1998c). Anderson (1998c) reviewed the captive management of buff-crested bustards at NARC and found that some pairs consistently produced strong and healthy chicks, while other pairs consistently
produced frailer chicks. In addition, hatchability of eggs from pairs of wild caught buff-crested bustards was higher than for captive bred pairs. Inbreeding is known to cause an increase in neonatal morbidity and mortality in captive-reared mammals (Munson 1993), and could be linked to the comparatively high mortality rates in these birds. Interestingly, other collections in the UAE that were given buff-crested bustard chicks from NARC also found this species difficult to raise (O'Donovan pers. comm.). Cox (2004) recently reported the experience of breeding white-bellied bustards at Jacksonville Zoo in the USA and reported a mortality rate of 45% for the first 160 days in parent-reared chicks.

22.3. Age-related Morbidity and Mortality of Bustard Chicks
Early care of chicks during the first 30 days after hatching is clearly important. Fabian (1980) noted that 76% of great bustard chick mortality occurred in the first two weeks after hatching. Bailey et al. (1997b) reported that forty-five percent of all bustard chick morbidity occurred during the first 30 days after hatching and subsequently varied between 8-15% for each successive month. Fifty percent of all mortality occurred during the first 30 days after hatching and also declined each successive month. Ostrowski et al. (1996a) reported that mortality of houbara bustards of less than a year old was higher than that of other ages and that the highest percentage of mortality occurred during the first month after hatching. Van Heezik and Ostrowski (2001) reported that up to 25% of the houbara bustard neonates died at the National Wildlife Research Center (NWRC, Taif, Saudi Arabia), during the first month of life. However, subsequent experience at NWRC has shown that the mortality rate of houbara bustards during the first month of life can be drastically reduced to about 4-5% by implementing hatching management, rearing and feeding methods, as well as monitoring techniques (Stiévenart 2002), that are better adapted to the species.

22.4. Hatching Disorders

22.4.1. Poor Hatchability
Poor hatchability is a condition of fertile eggs and should not be confused with infertility. It is hard to assess whether egg losses are a problem of fertility or hatchability due to an inability to differentiate between infertility and death in houbara eggs lost during incubation (Heezik 2000). While hatchability is affected by storage and incubation conditions in domestic poultry it is also a genetic characteristic and consequently hatchability could be a selectable trait. Causes of poor hatchability in bustards are poorly understood.

Malnutrition of the breeder flock, particularly regarding their vitamin and trace element status is known to depress hatchability in other species (Huchzermeier 1998). In poultry the following nutritional deficiencies can cause embryonic death or poor hatchability (Angel 1993):
- Vitamin A - early mortality.
- Vitamin E - early and late embryonic mortality.
- Vitamin D - late embryonic mortality.
- Riboflavin - mortality in mid-incubation, dwarving, oedema.
- Folic acid - late mortality, reduced hatchability.
- Iodine - prolonged incubation, lowered hatchability.
- Selenium - lowered hatchability.

An increased percentage of dead-in-shell chicks occurred at one houbara bustard breeding project in Dubai following changes to the pellet formulation. The new pellet was found to have significantly lower vitamin E levels (McKinney pers. comm.). It is also worth noting that hypervitaminosis E in laying hens also results in reduced hatchability and living chicks (Sünder et al. 1999).

The altitude of the breeding project needs to be considered too. Hassanzadeh et al. (2004) found significantly lower hatchability in commercial broiler eggs incubated either at sea level or at 2000m above sea level. More chicks hatched earlier, but embryonic mortality was higher in eggs incubated at high altitude. Higher corticosterone levels, as well as chronic hypoxia, were found in incubated eggs at high altitude.

22.4.2. Early Embryonic Death
On candling of eggs, early embryonic deaths are often very hard to differentiate from infertile eggs. In addition to the nutritional causes listed above early embryonic deaths can be caused by poor storage conditions and incubator temperatures that are too high. Saint Jalme et al. (1994) found that artificial insemination in houbara using an inappropriate insemination pattern results in increased embryonic mortality.

22.4.3. Malpositioning
Embryo malpositioning is not an uncommon condition and it is estimated that 1-4% of mature psittacine embryos will be in an abnormal position at full development before hatch (Clipsham 1996). Causes of malpositioning in other species include incorrect turning, abnormal shaped eggs, poor temperature control, careless handling, physical deformities of the embryo, dietary deficiencies and genetic defects in inbred birds (Olsen and Duvall 1994, Brown et al. 1996). The cause of malpositions in bustards is often not established and review of incubation records often reveals that...
are normal and other chicks hatched normally from the same incubator. A kori bustard with a head over the right wing malposition was considered to be caused by errors in the turning of the egg (Bailey and Anderson 2000). Table 14.3. in Chapter 14 summarises the classic malpositions in chick embryos.

22.4.4. Shell abnormalities
This has been discussed in Chapter 19.

22.5. Investigation of Hatching Problems in Bustards
A variety of techniques can be used to assist an avian embryo that is having problems hatching including radiography, endoscopy and ovotomy (Ensley et al. 1994, Bailey and Anderson 2000).

22.5.1. Radiography
Radiographic imaging to evaluate chick position in Californian Condor (Gymnogyps californianus) eggs has been shown to be a valid technique in determining whether eggs that are having problems hatching should be assisted (Ensley et al. 1994). Radiographs of bustard eggs can be taken with standard X-ray equipment using screen films. Average egg weights and the exposure settings used at NARC for different bustard species are listed in Table 22.1. The technique is to take four radiographs of an egg that is positioned along its long axis horizontal (Fig. 22.1). The egg should be rotated through 90 degrees after each radiograph and the eggshell marked 1-4 so that radiographic findings can be correlated with the egg. Using this technique it is usually possible to identify radiopaque structures including limb bones and the characteristic V-shaped mandible. From a series of radiographs it is usually possible to determine in malpositioned embryos the position of the head by identification of the ventro-dorsal profile of the mandible (Fig. 22.2). Radiography is a particularly useful technique for investigating problems with kori bustard eggs which are harder to candle compared with other bustard species because of the thickness of the shell.

22.2

22.1 A bustard egg is prepared for radiography (Photo credit Tom Bailey).

22.2 Radiograph of malpositioned kori bustard with the head in the small end. An arrow identifies the bill. Reproduced by kind permission of the Avicultural Society, Avicultural Magazine, 105: (3); 114-126, 2000. (Photo credit Tom Bailey).

Table 22.1. Radiographic exposure factors and average egg weights of the bustard species described in the case reports (source of egg weights Anderson 1998a, 1998b, 1998c).

<table>
<thead>
<tr>
<th>Bustard species</th>
<th>Egg weight (g)</th>
<th>KV</th>
<th>mA</th>
<th>Time (seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Houbara</td>
<td>52</td>
<td>60</td>
<td>20</td>
<td>0.12</td>
</tr>
<tr>
<td>Kori</td>
<td>146</td>
<td>60</td>
<td>20</td>
<td>0.2</td>
</tr>
<tr>
<td>Buff-crested</td>
<td>37</td>
<td>60</td>
<td>20</td>
<td>0.12</td>
</tr>
<tr>
<td>White-helied</td>
<td>50</td>
<td>60</td>
<td>20</td>
<td>0.12</td>
</tr>
</tbody>
</table>

From our experience with bustards we have found that it is possible to critically evaluate normal versus abnormal embryo position using radiography when there is doubt following conventional candling techniques. Post-mortem examination of a number of chicks provided further information to correlate with radiographic findings. The effects of serial radiography on bustards and other avian species has been discussed in Chapter 8 (Bailey et al. 2001d).

There is need for more detailed information on the hatching process in normal and abnormal chicks. Only with a larger database of information on the hatching sequence and time intervals for bustards can the aetiology of malpositions be determined and the timing for assistance optimised. The use of fluoroscopy to investigate hatching problems also warrants attention.

22.5.2. Endoscopy
In the larger eggs of kori bustards it is possible to insert a rigid endoscope (2.7 or 4 mm, Richard Wolf, Surrey) through a hole made in the blunt end of the egg. The membrane should be moistened with sterile saline and can be examined to determine embryo viability by monitoring respiratory movements. Use of the endoscope means that the hole in the eggshell does not have to be made larger than necessary. If the chick is
correctly positioned and/or no further immediate intervention is planned the hole in the egg can be sealed using micropore tape (3M Medical-Surgical Division, USA).

22.5.3. Ovotomy

From examination of radiographs the position of the head and bill can be established to determine the entry point for ovotomy (Figure 22.3). The shell over the entry point should be cleaned using dilute iodine (Pevidine antiseptic solution, B. K. Veterinary Products, UK) and cotton wool moistened with surgical spirit. A small hole is made in the eggshell using the point of a No. 10 scalpel blade and small pieces of eggshell are gently removed using forceps. In malpositioned embryos, because the entry site is usually away from the air cell, care should be taken to examine the membrane for regression of the blood vessels. This is determined by moistening the membrane with sterile saline, which then becomes translucent. Regressing blood vessels take on a ghost-like appearance and are only partially or not at all filled with blood. Pieces of eggshell and membrane are then progressively removed until the head and/or bill is found. Once the chick is provided with a breathing hole it should be left and checked every 2-4 hours when progressively more shell and membrane can be removed until the chick is hatched. As more shell is removed the yolk sac is visually examined and chicks are left in the shell until it has fully retracted. Chicks should be re-evaluated every 2-6 hours. If the yolk sac is still visible dilute iodine can be dropped onto it with a moistened cotton bud.

22.5.4. Post-hatching Care to Assisted Hatching Chicks

Assisted hatch chicks are susceptible to post-hatching conditions due to the combination of exhaustion and an incompletely absorbed yolk sac (Figure 22.4). Despite intensive and early antibacterial therapy many of these chicks succumb to yolk sac infections. Some malpositioned bustard chicks appear to be “physically exhausted” by the time intervention has provided a breathing hole or freed them. These chicks have a tendency to fade and die over the first 24-72 hours after hatching. An oxia or long-hatching syndrome has been seen at NWRC-Taif. Newly hatched chicks failed to thrive after a long hatching period and chicks are often lethargic and anorectic. The incidence of this condition at NWRC-Taif was reduced by the addition of oxygen to the atmosphere of the incubators (Ostrowski et al. 1996).

Once a diagnosis of malposition is made in a chick that has failed to internally pip immediate, but careful ovotomy over the site of the bill is recommended to provide a breathing hole for the chick. Assessment of membrane vascularity should determine the speed of further assistance. The use of radiosurgery to cut the membrane in which blood vessels that had not regressed has been described (Olsen and Duvall 1994) and may be applicable in some circumstances although the author has not had any experience with bustard chicks. Chicks that are assisted, but are still strong at the end of the process tend to be more viable compared with chicks that are "tired" because of delayed intervention. The success of assisted hatching is mainly down to timing, but unfortunately this is easier to determine retrospectively, rather than early in the morning or late in the evening when many of these cases are played out! It is the important to provide antibacterial therapy to chicks from assisted hatches. It is well known that the survival rate of chicks that hatch with larger umbilical protuberances have a lower survival rate (Joyner 1993).

At NARC all chicks with assisted hatches are given the following:

- Application of 1% iodine solution to the umbilicus of chicks promptly.
- Administration of amikacin or tobramycin for 72 hours and SC fluids (e.g. Duphalac, Solvay
  Duphar Veterinary, Southampton, UK) or PO electrolytes (Pedialyte, Abbott Laboratories,
  Chicago, USA) for 24-48 hours as newly hatched chicks are prone to dehydration.
- Supplementation of the rearing diet with probiotics (Avipro, Vetark, Winchester, UK) from 0-14 days.

A kori bustard being successfully helped from the shell following an assisted hatch. It is important to maintain a high standard of hygiene as chicks successfully hatched following intervention are more susceptible to problems such as bacterial infections (Photo credit Tom Bailey).

Normal pipping muscel (arrow) of a malpositioned kori bustard that died of anoxia after a prolonged hatching period and unsuccessful ovotomy (Photo credit Tom Bailey).
22.6. Yolk Sac Disorders

22.6.1. Yolk Sac Resorption
The yolk of the egg supplies nutrition to the developing embryo and newly hatched chick as well as antibodies for its passive protection. Before hatching the yolk sac is withdrawn into the abdominal cavity and the chick's navel closes over it. In ratites this remainder of the yolk sac is approximately 22% of the initial egg mass and the complete resorption of healthy yolk sac takes 10-14 days (Deeming 1997). The speed of resorption is further affected by temperature, stress and subclinical infections. Noy et al. (1996), demonstrated in poultry, that providing hatchlings with drinking water early in the post-hatch period improves the utilization of the yolk and facilitates motility of the digestive tract. Studies in chicks and turkeys have shown that access to feed immediately after hatching enhances the development of the intestine during the immediate post-hatching period (Uni 2005). At the NWRC-Taif, the installation of a systematic drinking water supply in the hatchery, combined with hand-feeding houbara bustard neonates directly upon arrival at the rearing unit, without any period of fasting, resulted in the disappearance of the unresorbed yolk sac syndrome. Early care to chicks immediately after hatching is clearly important.

22.6.2. Yolk sac infection
22.6.2.1. Pathogenesis of yolk sac infection
Omphalitis, yolk sac infection (YSI) and retention of the yolk sac are different aspects of the same condition and are a common cause of mortality in bustard chicks (Figures 22.5, 22.6). The infection of the yolk sac can take place via different routes, through the shell, the chorio-allantoic membrane (CAM), the albumen and through the umbilicus at hatching. In other species, such as ratites (Huchzemeyer 1998), bacteria penetrating the shell during incubation can remain localised under the CAM by the antibacterial action of the albumen. When, before hatching, the yolk sac is drawn into the abdominal cavity, any bacteria on and in the CAM can travel along the navel duct and penetrate the yolk sac. Throughout incubation, the growing avian embryo swallows the albumen while continually diluting it with its urine. At a later stage the albumen becomes sufficiently diluted for the antibacterial activity to be unable to control the bacteria under the shell. These can diffuse into the albumen and when they are swallowed, can colonise the intestinal tract and pass via the vitello-intestinal duct to penetrate the yolk sac. If during or after hatching the navel of the newly hatched chick comes into contact with a contaminated surface, the bacteria from such a surface can travel through the navel duct and contaminate the yolk sac, causing an umbilical inflammation. In the yolk sac they find nutrients and an optimum temperature for growth and from there they can spread through the vitello-intestinal duct into the intestine causing enteritis or enter the blood stream through the mesenteric blood vessels leading to hepatitis and septicemia. In these cases, findings at post-mortem examination include umbilical abscession and an inflamed yolk sac. Ratite chicks that do not drink because they are kept at too low a temperature, or that become dehydrated because they are kept at too high a temperature, will satisfy their water requirements from the yolk sac, which becomes inspissated and cannot be resorbed further (Huchzemeyer 1998). This may also be the case in bustards.

22.6.2.2. Diagnostic features of yolk sac infection
If the retained yolk sac is large enough it can be palpated. In ratites, ultrasound has been used to visualise yolk sacs and to monitor the normal regression of yolk sacs in healthy chicks (Blue-McLendon and Homeco 1995). The
use of ultrasound in bustards warrants further attention. If the infection has taken place via the navel around hatch, it is possible to find an inflamed area on the yolk sac wall around the navel duct and around the umbilical region. Abscesses are seen in ratters, but have not been observed in bustards, where inflammation is a common finding. In cases of an infection of intestinal origin, inflammation around the umbilicus and yolk sac is absent. A common finding at post-mortem examination are green-coloured yolk sac contents and while this can sometimes be associated with an infected and autolysing yolk sac, this can also occur in non-infected yolk sacs. The green discoloration occurs when bile pigments enter the yolk sac through the vitello-intestinal duct and in ratters this is believed to be take place because of the abnormal movement of intestinal contents if the intestine is empty, if the chick has not been fed or if it is not eating (Huchzermeyer 1998).

22.6.2.3. Treatment of yolk sac infection and retention

The yolk sac is inaccessible to the antibodies which, if given time, the chick might be able to produce against the bacteria; it is also inaccessible to antibiotics given to the chick. Thus, the condition cannot be remedied in individual chicks by antibiotic therapy. Surgery to remove the retained yolk sac has been described in ratters, but success rates vary and apparently the optimum time for removal is 10 days. The problem in bustards is that the condition is generally very acute, with chicks often presenting profoundly and acutely ill and generally not in a suitable state for surgery. A treatment used by South African ostrich farmers consists of penetrating the retained yolk sac via the navel canal with a large needle and withdrawing the yolk and instilling a small amount of antibiotic solution (Huchzermeyer 1998).

22.6.2.4. Prevention of yolk sac infection

- Egg and incubator hygiene.
  - To prevent the entry of bacteria via the umbilicus, strict incubator hygiene is necessary as has been discussed.
  - Fumigation/disinfection of eggs after collection/before setting.
  - Disinfection/fumigation of the incubator and hatcher.
  - Removal of infertile eggs and eggs with dead embryos.
  - Disinfection of all surfaces that come into contact with the hatchlings umbilicus during the first few days after hatching.
  - Nest hygiene - nest sites should be covered with clean sand at the start of the season and surface sand should be regularly changed at intervals throughout the season.
  - Environmental dust - environmental dust which is drawn with the air into the incubator is also an important source of contamination. This can be avoided by ventilating the incubation room, not directly from the outside, but through another room, in which the dust can settle. Air can also be ionised to precipitate the dust out of the air.

- Treatment of the navel - at hatch a wound disinfectant should be sprayed on the umbilical region of every chick. In ostriches, yolk sac infection in chicks that hatch with incompletely closed umbilici can be prevented by instilling broad spectrum antibiotic using a teat cannula.

22.6.3. Hernia of the Yolk Sac

Hernia of the yolk sac is straightforward to treat when the hernia is small. Survival of chicks with partially unretracted yolk sacs is higher than those with total unretraction (Ostrowski et al. 1996a). Treatment consists of ligation of the yolk, excision, disinfection and antibiotics (Bailey and Anderson 2000). Fluid therapy and administration of antibiotics are important.

22.7. Gastrointestinal Tract Disorders

22.7.1. Enteritis

Enteritis is an important cause of mortality in bustard chicks and is influenced by many factors including intestinal flora, nutrition, environmental factors and pathogens.

22.7.1.1. Intestinal flora

Birds hatch from the egg with a completely sterile digestive tract and microbes are quickly picked up within the nest or incubator (Klasing 1998). The source of the microflora depends on the hygienic conditions of the environment and the presence or absence of the parents (Klasing 1998). In intensive breeding projects bustard chicks are incubated and hatched from sterilised eggs, incubators and hatches, and after hatching they are reared in a heavily disinfected environment. Under these circumstances it is hard for bustard chicks to acquire a normal intestinal microflora. The normal microflora comprises useful bacteria that protect the intestine from infection with pathogenic bacteria by occupying the available attachment sites (competitive exclusion) and by creating an environment (pH and metabolites) in which pathogens cannot survive. It is interesting to note that ostrich chicks reared on pasture rarely suffer from enteritis, something thought to be because of their contact with soil and the fibre in their grazing (Huchzermeyer 1998).

22.7.1.2. Probiotics

Formulated probiotics can be used to provide some protection to bustard chicks, but ideally complete
protection can only come from providing a complete 'bustard' intestinal flora. The chicks of many avian species have been observed eating the faeces of their parents (Klasing 1998). Coprophagy is considered by many authors to help seed the digestive tract with beneficial microflora from the established flora of the parents' posterior digestive tract (Klasing 1998, Jeffrey 1999, Perlman pers. comm.). McKinney has used 'transfaunation' of bustard intestinal flora in white-bellied bustard chicks following antibiotic therapy. Dangers of feeding faeces from adult bustards to chicks could be the possibility that pathogens could be transmitted in this way. That said, it should be possible to maintain correctly vaccinated maternal 'donors', which have been appropriately screened for disease. However, one should bear in mind that even properly vaccinated breeders can shed pathogens that could threaten neonates; for example PMV-1. The faeces of rabbits or goats have been used successfully in ostriches (Huchzermeyer 1998). Although preliminary studies have assessed the normal aerobic microflora of bustard chicks (Naldo et al. 1998c), more research is warranted. Commercial probiotics have been given to white-bellied bustards at Dubai, but neither changes in growth rates or in Gram stains of faeces were noted.

22.7.1.3. Antibiotics
We are all guilty, aviculturalists and veterinarians, of tending to combat any emerging disease of bustard chicks with the liberal use of broad-spectrum antibiotics. Very often, instead of their desired effect, inappropriate use of antibiotics can suppress the normal intestinal flora and immune system of the chick.

22.7.1.4. Biosecurity and sources of infection
Prevention of infection by following strict hygiene is important. Biosecurity should be designed to keep pathogens out of bustard rearing units. Pathogenic bacteria, including Salmonella, can be transmitted by flies, reptiles, rats, mice that may be attracted to bustard rearing facilities. The failure to establish a normal intestinal flora, or its imbalance by inappropriate use of antibiotics can make the chick vulnerable to infection by pathogens causing enteritis, such as salmonellae and other enterobacteria. It should also be remembered that bustard chicks are coprophagic and any chicks with enteritis should be isolated because transmission within a group can occur rapidly.

22.7.1.5. Nutrition
Balanced nutrition of the breeder flock as well as of the chick ration is necessary to produce healthy chicks. Fibre is an important component of the rearing diet of ratite chicks, and the undigested fibre is thought to play a role in absorbing toxic metabolites of pathogenic bacteria (Huchzermeyer 1998). Similarly excessive protein in the starter ration appears to predispose ostrich chicks to enteric infections, while the inclusion of mannanoligosaccharides in the form of indigestible cell walls of brewers yeast provides attachment sites for bacterial pathogens and aids their elimination from the alimentary tract (Huchzermeyer 1998). What is clear is that early chick morbidity and mortality because of bacterial infection are important in many houbara bustard projects and modifications to the rearing diet may be an area projects should look closely at. Sudden changes to diets can also adversely affect the normal flora and diet changes should ideally be made gradually. McKinney noted a higher incidence of enteritis in bustard chicks when minced meat was included in the diet.

22.7.1.6. Environmental factors
Environmental factors include:

Heat - Chicks that have been exposed to excessive heat can develop dehydration, impaction, alimentary stasis and superinfection. Captive bred bustard chicks are also susceptible to heat stress when they are moved from environmentally controlled facilities to outside pens in the summer months in the Middle East.

Cold - Similarly a lowered body temperature, most often caused by the overnight failure of heat lamps can result in the reduction of the activity of the immune and digestive system.

Stress - Stress has a negative effect on the functioning of the immune system. Common stress factors encountered by bustard chicks include death of pen mates so that chicks are reared as singles rather than in a group, cold, heat, overcrowding and during transfers from indoor to outdoor rearing facilities.

Behaviour - Bustard chicks are coprophagic and chicks running around on matting in rearing coops will often pick up the faeces of other chicks in their instinctive search for food. Consequently, if one chick becomes infected with pathogens, infection may rapidly spread to other coop-mates because of this behaviour.

Treatment - Treatment with antibiotics can be successful with dealing with outbreaks of enteritis provided the pathogens are identified and sensitivity testing is carried out. Treatment can reduce further mortality, but it is important that in addition to therapeutic measures, the environmental and biosecurity faults are identified and corrected. Additionally, after treatment the establishment of a normal intestinal flora must be encouraged.

22.7.1.7. Bacterial enteritis
Gram-negative bacterial enteritis is a common cause of morbidity and mortality in young bustards (Bailey et al. 1997b). Similar to rats (Bruning 1973), an early sign of enteritis in bustards is dehydration of the legs. Gram-negative bacteria are the most important cause of enteric disease in young bustards, particularly Salmonella spp., Escherichia coli and Pseudomonas aeruginosa (Bailey et al. 1997b) (Figures 22.7, 22.8). These conditions have
been fully discussed in Chapter 18. Wherever possible, typing of *Escherichia coli* and *Salmonella* isolates should be carried out. At NRWC-Taif *Staphylococcus aureus* has been isolated from day-old houbara bustard chicks suffering digestive disorders. Sudden deaths in bustard chicks have been associated with *Clostridium perfringens* (alpha toxin producer) being the only significant finding. Predisposing factors are probably important in this condition because this bacterium can be isolated from normal chicks.

There is an increasing incidence of antibiotic resistance, particularly in the Middle East, and treatment should reflect the results of antibiotic sensitivity tests (Bailey et al. 1998f). All chicks given antibiotics should be given probiotic treatment. A common treatment in ostriches that warrants attention in bustards is the dosing of chicks with yeast tablets, for the mannoligosaccharides to help eliminate pathogenic bacteria (Huchzermeyer 1998). Viral agents have been occasionally isolated from cases of enteritis in bustard chicks including PMV-1 and adenovirus and such diseases should be considered in cases of enteritis that fail to respond to traditional antibacterial therapies. In one Dubai collection, McKinney has observed that fewer deaths are seen in bustard chicks if mince meat is not fed. This may be because the high temperatures cause rapid meat spoilage resulting in enterotoxemia.

### 22.7 Depressed white-bellied bustard chick with *Escherichia coli* septicaemia and enteritis, notice the closed eyes and ‘sleepy’ appearance (Photo credit Tom Bailey).

### 22.8 Buff-crested bustard chick showing enteritis and CNS signs including ataxia, loss of balance and incoordination. *Salmonella* sp. was isolated from clinical samples and the bird responded to antibiotic therapy (Photo credit Tom Bailey).

#### 22.7.1.8. *Fungal Gastritis*

Fungal infections of the digestive tract of bustards are caused by fungi from the environment or normal inhabitants of the intestinal tract which under certain conditions become invasive and pathogenic. Infections with *Candida* spp. can cause stomatitis, proventriculitis and ventriculitis. Candidiasis in bustard chicks is a side-effect of prolonged antibiotic therapy. Other fungi (*Mucor* spp., *Aspergillus* spp., *Rhizopus* spp.) have been associated with outbreaks of gastric mycosis in raptites (Perelman and Kuttin 1992). These diseases have been covered in chapter 18.

#### 22.8. Mechanical Gastro-intestinal Tract Conditions

### 22.8.1. Impaction

#### 22.8.1.1. Clinical description

Mechanical gastro-intestinal tract conditions are not uncommon causes of death and morbidity in juvenile bustards. In a study conducted at NARC, 16% of mortality and 4% of morbidity over the first 180 days was attributable to mechanical gastro-intestinal tract conditions (Bailey et al. 1997b). Impactions of the proventriculus and ventriculus, and perforation of the gastro-intestinal tract following foreign body impactions, have been reported as a cause of death in Australian (*Ardeotis australis*), great and houbara bustards (Grummitt 1979, White 1985, Ostrowski et al. 1996a).

Impactions are usually caused by bustard chicks ingesting foreign matter, which accumulates in the proventriculus and blocks the entrance to the ventriculus. With the entrance to the ventriculus blocked, no food can pass through, the ventriculus stops contracting and the bird will die of starvation. Some ingested foreign bodies will also perforate through the proventriculus or ventriculus causing problems with peritonitis and septicaemia. *Clostridium perfringens* can be isolated from chicks that have died due to severe impactions.

Most commonly the ingested material consists of substrate, such as grit or gravel or sand, although chicks have also been known to ingest fragments of loose palm.
material from the walls of old enclosures and twigs from outdoor enclosures. Cases of impaction have also occurred when chicks have eaten frayed pieces of carpet that they were being reared on. Buff-crested bustards appeared to be more susceptible than the other species to gizzard impactions and foreign-body obstructions of the upper and lower digestive tract (Figure 22.9). Impactions in bustards can be associated with a move to a new pen where chicks will consume ‘new’ objects that were not present in the original environment. Blockage with grass fibres has been in seen in chicks moved from rearing unit to outdoor vegetated pens and white bellied bustards have even been noted to become impacted with egg shells.

Partial sand impaction has been seen occasionally in juvenile bustards and is caused by the ingestion of sand from the litter. In the cases seen at NARC the sand had passed through the proventriculus and ventriculus and had accumulated in the small intestine. Partial impaction has also been seen occasionally in juvenile bustards and again the small pieces of gravel had passed through into the small intestine. In the cases seen the birds were dosed with vegetable oil at a dose of 1 ml/kg.

Ventricular impaction has also been reported in bustard chicks secondary to iatrogenic pharyngitis caused by damage subsequent to force-feeding.

22.8.1.3. Post mortem findings
The presence of accumulated foreign objects or material in the proventriculus or ventriculus is indicative of impaction.

22.8.1.4. Treatment and prevention
The surgical removal of impacted material in ratite chicks by a ventriculotomy operation has been described in Chapter 10 and a similar operation has been successfully used to remove metallic foreign bodies from sub adult kori bustards (Bailey et al. 2001f). Gastric lavage is recommended in ratite chicks followed by supportive therapy (fluids, warmth and gastric stimulants such as metaclopramide or cisapride).

“Overload ventriculus” has been reported as a cause of death in hand-raised great bustards fed without grit (Seidel 1995). To avoid this condition, Grummt (1979) recommended that bustard chicks should be fed small gravel particles the day after hatching until they learn to pick up stones by themselves.

It is important to ensure that the rearing environment is not contaminated by material that can obstruct the alimentary tract of bustard chicks. Impaction with grit has not been observed in bustard chicks reared at NARC, despite the fact that chicks are reared on a fine grit substrate after 4 days of age. Impactions with grit have occurred in houbara bustards reared at the NWRC-Taif, and chicks are reared on sand with a layer of fine plastic mesh on top in order to prevent them from ingesting excessive amounts of substrate (Ostrowski et al. 1996a). In ratites it is considered that stress, such as the movement to new pens, is associated with this condition (Frasc and Khan 1977).

22.8.2. Cloacal Prolapse
Cloacal prolapse occurs sporadically in individual bustard chicks. In young birds this condition is often associated with severe diarrhoea, impaction, nutritional deficiencies and tenesmus. In ratites cloacal prolapse is associated with cryptosporidiosis (Huchzermeyer 1998) and histomoniasis (Iordanidis et al. 2003), while in bustard chicks the cause is often not known. Purse string sutures are usually satisfactory in the cases seen in bustards as long as the primary cause, such as diarrhoea is dealt with.

22.9. Musculoskeletal disorders

22.9.1. Metabolic Bone Disease
Metabolic diseases of the bones are common problems in birds, particularly long-legged species such as bustards (Naldo et al. 1998a). Musculoskeletal disorders and soft tissue-related traumatic injuries were the most commonly diagnosed clinical conditions in juvenile bustards accounting for 66.6% of the total findings.
(Bailey et al. 1996a). Musculoskeletal problems were seen in 100% of parent-reared white-bellied bustard chicks at Jacksonville Zoo (Cox 2004). Nutritional bone disease was the single most important musculoskeletal disorder in captive juvenile bustards accounting for 24% of total findings at NARC between 1993 to 1995 (Naldo et al. 1998a). Metabolic bone disease may be seen as splayed legs, fractures, folding fractures, rickets, beak malformations, crooked toes, bowed legs, tibiotarsal rotation and slipped tendon (Figure 22.10). An excess of either calcium or phosphorus results in the sequestration of the other in the form of an insoluble calcium phosphate (Smith and Roudybush 1997). In the intestine this results in less absorption and availability of the element found in low concentrations. The ration of calcium to phosphorus in diets for growing birds needs to be in the range of 1:1 to 2:1. Calcium deficiency leads to nutritional secondary hyperparathyroidism in bustard chicks. Typically folding fractures occur from 3 weeks of age onwards, coinciding with the time period when bustard chicks become more active. This disease can be corrected with intensive calcium and vitamin D3 therapy. The high incidence of musculoskeletal disorders in captive houbara, white-bellied and buff-crested bustards occur between the first and 13th week of age (Naldo et al. 2000a). This coincides with the time when the growth rates in the length of long bones are at their peak for these species and when the bones are susceptible to pathological disturbances. In comparison, musculoskeletal disorders occur in kori bustards until 26 weeks and in this species the long bones still have a high growth rate until 27-31 weeks (Naldo et al. 1998a, 2000a). Similar late-stage problems have been seen in Heuglin’s bustards in post-rearing enclosures. Naldo et al. (2000a) found that the growth rate of the tarsometatarsus in young kori, houbara and white-bellied bustards is greater than the domestic fowl, but lower than ostriches (Struthio camelus) and sandhill cranes (Grus canadensis). The mean growth rate of the tarsometatarsus at 3-4 weeks in houbara, buff-crested, white-bellied and kori bustards is 1.47, 0.85, 1.35 and 1.84 mm/day respectively. The mean growth rate of the tibiotarsus at 3-4 weeks in houbara, buff-crested, white-bellied and kori bustards is 1.44, 0.95, 1.47 and 1.8 mm/day respectively. Another interesting finding from the work of Naldo et al. (1997b, 2000a) is the fact that ossification of the long bones and maximum growth rate of bones is achieved at an earlier age in houbara bustards compared with smaller species such as the buff-crested and white-bellied bustards. This early bone development in houbara bustards may be related to the need of juvenile birds to migrate and fly long distances from their breeding grounds.

22.9.2. Tibiotarsal Rotation

22.9.2.1. Clinical description

Tibiotarsal rotation occurs in bustard chicks of up to 120 days of age and usually affects individual birds (Naldo et al. 1998a) (Figures 22.11, 22.12). Varus deformity is the most common finding. The tibiotarsal bone rotates laterally above the hock joint up to 90° from normal, rotating the foot (tarsometatarsus and toes) laterally to the outside. This rotation takes place rapidly, usually within a couple of days. This condition may result in slipped tendon. Torsion of the femur can be concomitant with tibiotarsal rotation. Although developmental abnormalities involving the long bones of hand-reared precocial birds are commonly attributed to high energy diets, high protein diets, and rapid growth rates (Serrafin 1982, Leeson and Summers 1988, Kirkwood et al. 1989), rotational deformities of long bones can also result from trauma to the growth plates (Kirkwood et al. 1987). Such growth plate trauma can result from inappropriate capture or handling techniques, and also during apparently routine capture or handling of chicks (Charles Deeming cited in Anderson 1998b). Buff-crested and white-bellied bustards appear to be particularly susceptible to this condition. In broiler chickens, leg deformities can result from malfunction of the growth plates of the proximal or distal tibiotarsus, which causes uneven bone growth and bowing and twisting of the tibiotarsus (Poulos et al. 1978). Naldo et al. (1998a) reported a narrow epiphysis and uneven growth plate in the tibiotarsus and tarsometatarsus of a buff-crested bustard with a valgus deformity.

22.10

Dropped wing in a white-bellied bustard with a fractured ulna. This bird had metabolic bone disease (Photo credit Tom Bailey).
22.2.2 Causes

Like so many bustard diseases, this condition has been incompletely investigated and the causes are not known. In ratites this is a multifactorial disease and there is a link with the rate of growth and consequently lowering protein levels in the ration has been used as a way of reducing the growth rate and reducing the incidence of this condition in ostrich flocks (Huchzermeyer, 1998). Bezuidenhout et al. (1994) found increased serum zinc values and decreased serum calcium and phosphorus in ostriches with tibiotarsal rotation resulting in poor mineralisation. Consequently, it is also thought that a poorly-balanced diet and enteric infections causing malabsorption of minerals such as phosphorus may also be contributing factors (Huchzermeyer 1998). Seidel (1995) considers that perosis in bustards is caused by deficits of manganese, zinc and excessive dietary protein. At Berlin no perosis has been seen in bustard chicks since the following measures were introduced during the critical period of around 15 days; 1) reduced dietary protein to <15%, 2) supplementation of MnSO4 (30mg/kg food) and 3) giving no additional calcium and phosphorus (Seidel 1995).

The tibiotarsal bone of bustard chicks grows rapidly and is consequently subject to strong mechanical forces from the associated muscles and from the weight of the growing chick. Growth plates are highly metabolically active and are supplied with nutrients by blood vessels (Wise and Jennings 1973). Any disturbance, such as trauma or other insults can cause a local disruption of the blood supply, resulting in slowing of growth on one side of the plate and rapid bending in the opposite direction. Genetic factors are also thought to be important and some ratite parents produce chicks with a higher incidence of leg rotation in their offspring (Huchzermeyer 1998). Reviewing parentage records is important when reviewing this condition in bustards so that any parents producing a higher incidence than flock average of chicks with deformities should be excluded from the breeding programme. In ratites lack of exercise is also considered to be a causative factor of tibiotarsal rotation.

22.2.3. Exercise

The role of exercise in the development of tibiotarsal rotation is controversial. Some authors (Allwright 1994, cited in Huchzermeyer 1998) consider that chicks should be reared in small cages, to prevent exercise associated leg injuries, such as stumbling over feed troughs. Other authors consider that a lack of exercise, along with weakness of the adductor muscles are implicated in the aetiology of tibiotarsal rotation in ratites (Guitteny 1986, Labonde et al. 1994). Common sense would suggest that a degree of exercise should be beneficial by strengthening the growing bones as well as reducing the rate of growth.

22.2.4. Correction

Successful surgical treatment by osteotomy, derotation and fixation by pins has been conducted in white-bellied and houbara bustard chicks. However, while this has been successful in a small number of chicks, most chicks with this problem have to be euthanised.

A successful orthopaedic treatment developed at NWRC-Taif fully corrects the varus tibiotarsal malposition within a few weeks, if it is implemented as soon as the tibiotarsal rotation is noticed (Stiwenart 2005), and in chicks that are being raised on a thick sandy substrate.

22.2.5. Prevention

- Recommendations from the ratite industry include:
- Not breeding from birds with a history of leg rotation or from birds that have received surgical correction.
• Well-balanced diet of parent flock and chicks.
• Avoid intestinal infections.
• Providing an injury-free environment. It is preferred to keep small chicks in smaller areas because in large exercise spaces there is a tendency for uncontrolled running with consequent stumbling or falling, which may be a factor leading to tibiotarsal rotation.

The incidence of nutritional bone disease was reduced dramatically at NARC after 1994 and reflected simultaneous changes to the rearing environment and rearing regimes that included improving diet quality, increased exercise levels, giving chick’s access to sunlight at an earlier age as well as supplementation with Vitamin D3.

22.9.3. Chondrodystrophy

22.9.3.1. Clinical description

Chondrodystrophy is defined as a generalised disorder of the growth plates of the long bones that results in impaired linear growth, whereas mineralisation and appositional growth remain normal (Wise 1975). Chondrodystrophy has been reported in great bustards (Gewalt and Gewalt 1966, Grummt 1979, Seidel 1995, Sukhanova 1992) and Australian bustards (White 1985) where it occurs at 12-16 days of age (Gewalt and Gewalt 1966, Seidel 1995). Naldo et al. (2001) described three houbara bustard chicks with shortened metatarsal bones as a result of chondrodystrophy (Figures 22.13, 22.14). Two of these birds developed varus deformities and fused tarsometatarsal trochleae that resulted in swollen intertarsal joints, luxated gastrocnemius tendons and dislocated phalanges (Naldo et al. 2001).

Measurements of long bones of the three birds described by Naldo and Bailey were taken from radiographic films and were compared with measurements from radiographs of seven apparently normal birds of similar ages (Table 22.2). The tarsometatarsi and tibiotarsi of bird A were 30% and 20% shorter, respectively, compared with the same bones from normal birds. The lengths of the humerus, radius, and ulna of bird A were within the normal ranges of these bones (Naldo et al. 2000a). The tarsometatarsi of birds B and C were 22% and 29% shorter, respectively, compared with the tarsometatarsi of normal birds. The lengths of the tibiotarsi, humerus, radius, and ulna of birds B and C were within the normal ranges (Naldo et al. 2000a).

Table 22.2. Length in millimetres of tarsometatarsus and tibiotarsus of three houbara bustards with shorter legs in comparison with seven normal birds (Naldo et al. 2000a) a.

<table>
<thead>
<tr>
<th>Site</th>
<th>Age (weeks)</th>
<th>Bird A</th>
<th>Bird B</th>
<th>Bird C</th>
<th>Normal birds a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tarsometatarsus</td>
<td>4</td>
<td>37.8</td>
<td>46.3</td>
<td>42.5</td>
<td>59.2 ± 1.93c</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(50.0 - 65.0)d</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>58.5</td>
<td>ND</td>
<td>56.2</td>
<td>83.2 ± 2.86</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(69.2 - 91.4)</td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>65.6</td>
<td>ND</td>
<td>ND</td>
<td>93.9 ± 3.05</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(83.6 - 102.5)</td>
</tr>
<tr>
<td>Tibiotarsus</td>
<td>4</td>
<td>50.3</td>
<td>61.0</td>
<td>67.5</td>
<td>71.3 ± 1.90</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(63.3 - 77.8)</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>77.8</td>
<td>ND</td>
<td>93.0</td>
<td>99.2 ± 2.78</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(85.3 - 107.3)</td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>101.0</td>
<td>ND</td>
<td>ND</td>
<td>126.7 ± 4.47</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(111.5 - 139.4)</td>
</tr>
</tbody>
</table>

a measurements were taken from radiographic films; b three females, four males; c Mean ± standard error of the mean; d Minimum - Maximum; ND = no data.
played a role in this deformity. The three houbara bustards were produced by the same female parent. Two chicks shared the same father, while the male parent of the third bird was unknown. The parentages of both female and male parents were not known, although they were suspected to be related, due to the small number of breeding birds.

### 22.9.3.3. Prevention

Measures that proved effective to prevent this condition in Australian bustards (White 1985) included reduction of protein in the diet during the critical period of 12-16 days of age, an increase in green foods, supplementation with magnesium sulphate (30mg/kg food) and no calcium/phosphorus addition to the diet. However, at NWRC-Taif, chondrodystrophy has been found in a houbara bustard chick fed on a reduced food intake for the three first week of life. As a genetic link is suspected at NARC, surviving chicks should be excluded from breeding programmes.

### 22.9.4. Splayed Legs

If caught early splayed legs are easily treated by hobbling the legs together with adhesive conforming bandage (Vet wrap, 3M Animal Care Products, USA), leaving enough freedom of movement to allow the chick to walk (Figures 22.15, 22.16). For prevention of splayed legs, it is important to provide a warm, non-slip surface and to watch the newly hatched chicks closely so that if problems do occur legs can be hobbled promptly. Naldo et al. (1998a) considered that several factors including incubation conditions and the condition of the floor of the hatching unit caused this condition in buff-crested and white-bellied bustards. Similar observations were noted in houbara bustard hatchlings at NWRC-Taif. Flammer (1986) reports that this condition is sometimes responsive to injections with vitamin E and selenium in psittacines, but in our experience at NARC experience taping the legs has proved successful.

### 22.9.3.2. Causes

Several factors are known to cause chondrodystrophy in domestic poultry. In chickens and turkeys, manganese, copper, biotin, and choline deficiencies cause chondrodystrophy of the distal tibiotarsus and proximal tarsometatarsus, resulting in displacement of the gastrocnemius tendon from the intercondylar groove (Riddell 1992).

Naldo et al. (2001) considered that inbreeding may have
Twisted or rolled toes occur in raities when breeder hens experience nutritional deficiencies (Huchzermeyer 1998). Gewalt and Gewalt (1966) suggested that rolled toe develops secondary to perosis and in other species riboflavin (vitamin B2) deficiency and embryonic malposition can cause this condition (Anderson 1983). Micronutrient analysis of vitamin B2 levels in infertile buff-crested bustard eggs was conducted at NARC and revealed adequate levels of this vitamin (Anderson 1998a, 1998b, 1998c), assuming that this species has similar requirement as domestic poultry. While treatments using splints and bandages have been suggested for raities (Blue-McLendon 1993), because of their size it is often hard to apply bandages to the smaller species of bustard chicks and this condition, when mild usually corrects itself as the chick develops. Exercise on a thick sandy substrate improves this condition very well.

### 22.9.6. Slipped Wing

The term slipped wing is used to describe a condition in growing chicks where the increased weight of the rapidly developing primary feathers causes an overextension of the carpal joint and the outer wing starts to droop. If left untreated the wing twists outward at the carpal joint resulting in a permanent deformity. Slipped wing is also known as ‘angel wing’ or ‘dropped wing’. This condition has been seen in all species of bustards reared in captivity. Preventive measures should be taken as soon as the chick’s wings look as if they are going to start to droop. Micropore tape is used to tape the wingtip to the upper wing for support for 2-4 days, with the tape changed every couple of days as the chick grows (Figure 22.18). Although high growth rates and high energy/high protein diets are commonly blamed (Seidel, 1995), non-dietary factors may also be involved. White (1985) reported that slipped wing problems were eliminated in captive-reared Australian bustards when the brooder temperature was reduced periodically during the day to mimic female brooding behaviour. Angel wing is a common occurrence in hand-reared bustard chicks, typically occurring at day 10-14. Taping the affected primaries in a natural position at the first sign of the outward turning will permanently correct the deformity. A definitive cause of the problem has not been determined (Boylan et al. 2001).

### 22.9.7. Osteomalacia

The lack of either calcium or phosphorus in the ration can lead to the development of soft bones (osteomalacia). This condition can lead to serious deformities in other avian species, but in bustards this usually leads to multiple fractures of the long bones of the legs and wings. Interestingly, Gewalt and Gewalt (1966) observed that the bones of great bustards are “brittle” and noted that the brittleness was more pronounced in captive reared birds, which was considered to be a result of calcium deficiency. Multiple
folding fractures of the tarsometatarsus, tibiotarsus and femur have been seen in houbara and white-bellied bustards.

22.9.8. Fractures
Long bone fractures accounted for 25% of musculoskeletal disorders in the four species of bustard chick reviewed by Naldo et al. (1998a). Fractures occurred at 61-120, 31-120 and 31-180 days of age in buff-crested, houbara and kori bustards respectively (Naldo et al. 1998a). Fractures of the radius and ulna occurred most commonly and in this study the majority (53%) were found incidentally during a radiography study of skeletal development. This suggests that most fractures probably go undiagnosed in growing bustard chicks and heal by themselves. Most of the fractures were healed or healing fractures and there was a higher frequency of fractures on the distal ulna/radius and metacarpes of the pinioned wing. Naldo et al. (1998a) considered that the pinioned wing was more prone to fractures because of the lack of protection provided by the primary feathers. Healed fractures are often found incidentally in white-bellied bustard chicks. Group rearing of some species, like white-bellied bustards may encourage excessive jumping and flapping, which may predispose chicks to fractures. Trimming wing feathers of chicks prior to a move to a naturalistic aviary may help to reduce the incidence of wing fractures.

22.10. Other Disorders

22.10.1. Adenovirus hepatitis
Hepatitis associated with adenovirus has been reported in one bustard chick at NARC. This has been described in Chapter 16 (Figure 22.19).

22.10.2. Ants
Samsum ants (Pachycondyla semauerensis) have occasionally been found on dead bustard chicks that were left with their mothers in naturalistic aviaries (Figures 22.20a/b). Samsum ants are omnivorous and have a sting and the venom is responsible for allergic reactions in humans. It is not known if these ants were responsible for the death of bustard chicks or whether they were just an incidental finding.

Fire ants (Solenopsis geminata) have been recorded as entering a hatcher, through the external pip hole and were thought to have killed a hatching kori bustard chick. These are aggressive ants with a sting that can leave a marked induration on human skin for up to 2 weeks (Vojin Sljivic pers. comm.).

22.10.3. Avipox virus
An avipox virus disease outbreak was observed at the NWRC-Taif in six-week-old Chlamydotis macqueenii chicks hatched from female houbara bustards properly vaccinated with a Canary poxvirus attenuated live vaccine. Most of the lesions were cutaneous, but some individuals developed a diphtheritic form of the disease that affected the respiratory tract. Avipox virus in adult birds is reviewed in Chapter 16. Avipox virus infections have been seen in chicks of all species of bustards maintained at NARC (Figure 22.21).

22.10.4. Bradygnathism
Bradygnathism of the maxilla has been seen in kori bustards, but the cause is not known (Figure 22.22).

22.10.5. Cataracts
Standard incandescent bulbs should not be used, at least three chicks reared under incandescent bulbs at NARC in 1993 developed cataracts. No cases of cataracts were observed subsequent to this with chicks reared under ceramic dull-emitter bulbs.

22.10.6. Chlamydomophilosis
A chlamydophilosis outbreak in three-week-old Chlamydotis macqueenii chicks living in outdoor pens was reported at NWRC-Taif. The birds presented with severe clinical signs including blepharo-conjunctivitis, tracheo-bronchitis and pneumonia, as well as poor quality feathers. Successful treatment comprised enrofloxacin for a two-week period according to Gerlach (1994), as well as mucolytic drugs. Chlamydotophilosis in adult birds is reviewed in Chapter 17.

22.10.7. Colibacillosis
At NWRC-Taif, a colibacillosis outbreak involving a pathogenic Escherichia coli strain occurred in chicks aged one to five-weeks that were fed a deficient diet. Lesions consisted of pericarditis, hepatoperitonitis, splenitis and septicemia. Colibacillosis in adult birds is reviewed in Chapter 17.

22.10.8. Dehydration Emaciation Syndrome
This has been described in chicks that became thin and dehydrated because of an un-noticed increase in the ambient temperature of the rearing room that lead to the chicks becoming dehydrated (Anon 1993).

22.10.9. Feather Picking
McKinney has observed feather picking and bullying in white-bellied bustards reared in a group setting outdoors. In extreme cases feather picking can be debilitating, leading to bacterial dermatitis and septicemia.

22.10.10. Reovirus associated Tracheitis and Pneumonia
A collection in Dubai lost four Heuglin's bustard chicks with a tracheitis-pneumonia syndrome from which reovirus and Pseudomonas aeruginosa were isolated. After these losses all bustard chicks at this collection are vaccinated against reovirus and this syndrome has not been subsequently seen.
22.19 White-bellied bustard with hepatitis. An adenovirus was isolated from this bird (Photo credit Tom Bailey).

22.20a-b a) Fire ants (Solenopsis geminata) have been recorded as entering a hatcher, entering through the external pip hole and were thought to have killed this hatching kori bustard chick. b) Note the intense reaction (arrow) on the close up of the leg (Photo credit Tom Bailey).


22.22 Bradygnathism of the maxilla in a juvenile kori bustard (Photo credit Tom Bailey).

22.23 A two week old kori bustard chick with sinusitis, rhinitis, and conjunctivitis. *Pseudomonas aeruginosa* was isolated from nasal and sinus aspirates (Photo credit Tom Bailey).

22.24 Ruptured liver (arrow) in a houbara bustard chick following trauma (Photo credit Tom Bailey).
22.10.11. Sinusitis, Blepharitis and Conjunctivitis
Chicks are susceptible to the full range of ocular and respiratory pathogens recorded in adult birds (Chapter 19). Pseudomonas sp. is commonly implicated in cases of sinusitis and a sneezing rhinitis-conjunctivitis syndrome (Figure 22.23). Blepharitis and conjunctivitis has been observed in bustard chicks exposed to disinfectant fumes from coops that had been cleaned with Virkon (Antec International, Suffolk, UK), but had not been rinsed with water. McKinney has seen bustard chicks with a conjunctivitis-sinusitis syndrome that were serologically positive for Mycoplasma gallisepticum.

22.10.12. Toxicity
McKinney has observed secondary brodifacoum toxicity in a 1 month old houbara bustard that ingested three poisoned mice.

22.10.13. Trauma
Trauma is a common cause of injury in bustard chicks and the prevention of trauma has been discussed in Chapter 3. (Figure 22.24).

22.11. Chick Quality
Deeming et al. (1996) recommends grading of ratite chicks when they hatch. Grading of ratite chicks on removal from the hatcher according to appearance and size can significantly improve the survival of birds in the first 2-3 weeks post-hatching. Top grade birds come from the best quality eggs incubated under optimum conditions. Deeming et al. (1996) uses the following criteria to grade ostrich chicks, which could be adapted for use in bustard chicks:

**Top grade**
- alert.
- bright-eyed.
- sit up with their legs tucked below them.
- clean down, free from any dried material.
- navel sealed, no protruding yolk sac or blood vessels.
- no visible deformities.
- no signs of distress (panting etc).
- Chicks with unhealed navels, oedema, and an inability to sit or that have splayed limbs should be down-graded.

22.12. Conclusions
As most mortality and morbidity of bustard chicks occur during the first 30 days of life, health care should be focused on this period. Musculoskeletal, bacterial and mechanical-gastro-intestinal conditions cause most morbidity and mortality in bustard chicks. Inbreeding may also be a factor contributing to the morbidity and mortality of captive-bred and reared, bustard chicks but further investigation is needed.

Modifications to the management of chicks and the rearing environment can reduce the incidence of chick mortality. At NWRC-Taif, after a few years, after implementing a reliable individual monitoring programme of the neonates, combined with improving the hatching management, the feeding system, the hygiene throughout the incubation and rearing processes, as well as reducing trauma, resulted in a significant decrease of the morbidity and mortality rate (from 25% to 5%) during the first month of age. Procedures carried out at NARC to maximize the health of bustard chicks have been reviewed in Chapter 3.