Ostrich Paediatrics.

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Abstract

The ostrich (*Struthio camelus*) has been farmed intensively in South Africa for over 100 years, and in Australia, the United States and Europe for the last ten years. Although the adult bird is robust and generally easy to maintain and breed, the single most important limiting factor in intensive farming has been chick survivability. This paper discusses the "normal" chick, factors affecting the viability and growth of ostrich chicks, the principles behind successful chick rearing, and the common diseases causing loss to the ostrich industry.

The "normal" ostrich chick.

In Australia, there are four commercial `types' of ostriches; the Australian Grey, the descendant of birds brought to Australia and farmed at the beginning of the 20th Century, noted for its early maturity and prolific egg-laying, but generally a smaller, poorer quality bird; the African Black, a stable cross breed of the South African (*S. camelus australis*) and Barbary ostrich (*S. camelus camelus*), noted for its larger body and feather quality, but slower to mature; the Zimbabwe Blue, the Somali ostrich (*S. camelus molybophanes*), noted for its massive size and late development; and the Canadian Red, the Massai ostrich (*S. camelus massaicus*), similar in many respects to the Zimbabwe Blue. The latter three types have been imported into Australia from Africa (eggs) and from Canada (live birds) in the last 7 years. As well as differences between adults, there are marked differences between chick survivability and growth among the four types.

The ostrich egg when laid averages a weight of 1,500g (900 - 1,900 g). Young birds and Australian Greys tend to lay smaller eggs. Very small and very large eggs can be difficult to incubate, and can result in poorly viable chicks. Over an incubation period of 42 days, a weight loss of 12% - 17% is desired. Weight losses below 9% and over 20% generally fail to hatch or, if they do, produce poorly viable chicks. Other incubation parameters, such as ventilation, temperature, turning frequency, hygiene and positioning can all have an effect on the viability of the newly hatched chick.

The ostrich chick is precocial, that is it hatches fully feathered and independent of its mother for food. Nutrition in the first week of life is provided by the contents of the egg. Calcium for bone development and growth is initially absorbed from the egg shell during incubation. Some other proteins, minerals and vitamins, contained in the albumen, are swallowed by the chick in the last days of incubation. The nutritional value of the yolk is determined to a large extent by the nutritional status of the hen, which therefore has a direct effect on the chick's survivability. The yolk

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sac is resorbed rapidly in the first week, and resorption is complete, on average, in 14 days (range 12 - 20 days) (Blue-McLendon, 1995). Ostrich chicks, however, begin to eat offered feed within days of hatching. They often need to be taught to eat, either by mimicking older chicks or by pecking at shiny objects in the feed.

Like many young animals, the ostrich chick's ability to digest and absorb its diet differs markedly from that of the adult. Fat digestibility is poor until 6 weeks of age and then increases until 17 weeks. Not only will fat digestion and absorption be reduced, but the ability to utilise fat-soluble vitamins (A, D, E and K) in the diet is therefore limited (Angel, 1995). The ability to digest fibre is limited until 12 weeks of age (6% at 3 weeks, 27% at 6 weeks, and 58% at 17 weeks.). (Angel, 1996) Therefore, chick diets must, of necessity, vary greatly from the adult ration. Additionally, the importance of maternal nutrition becomes clear, as many of the fats and vitamins required for early growth must come from the yolk.

No specific work has been done on the development of the chick's immune system. However, it is generally conceded that a functional immune system does not begin to operate until 4-6 weeks of age. Until then, reliance is placed on maternal immunity via the yolk. There have been recent suggestions that there is little transference of such maternal immunity (Allwright, pers. comm. 1996). It is therefore wise to assume that until 6 weeks of age, the ostrich chick should be regarded as immuno-incompetent.

The growth rate of an ostrich chick is a fairly accurate barometer of its health. The weight of the chick at hatch averages 900g. This is, however, dependant on several factors - the type of ostrich (Australian Grey chicks tend to be smaller, averaging 700g), the size of the egg, weight loss during incubation, age of the hen, and stage of the egg laying season. Very small chicks generally do poorer than larger chicks. Over the first 4 - 5 days post-hatch, there will be a drop in weight as the yolk sac is resorbed. After that, there should be a steady growth of 4% - 5% per day for the first 30 days, 2.2% for 70 - 90 days, and then 3.1% from 100 - 120 days (Samson, 1996). Factors affecting growth rates include genetics, nutrition, management (stocking rates, ventilation, temperature, etc.) and health status (Blue-McLendon, 1996). Blue-McLendon (1994) proposed the following weights for African Black and Black-Blue cross ostrich chicks fed ad-lib on a diet that contained 20% protein, 2.5% fat, 12% fibre and an ME of 9,200 kjoules:

Age	Weight (Kgs)
4 weeks	4 (3 - 6.5)
8 weeks	14.5 (10.4 - 19)
12 weeks	30 (25.5 - 34.5)
16 weeks	49 (42.5 - 53)
20 weeks	67 (56.5 - 74)

Table 1. Weight of chicks at different ages, adapted from Blue-McLendon, 1994.

Deeming *et al* (1996) showed that the behaviour of chicks also has an effect on growth rates. His study showed: that chicks that peck at other chicks grew slower than others; that chicks prefer to

eat out of shallow rather than deep dishes; that colour is important in feed selection, with green being the most popular colour; that chicks prefer lucerne chaff to pellets; and that chicks reared in "mixed weight" groups grew more slowly than chicks in "same weight" groups. Black (pers. comm., 1997) makes the point that chicks will selectively eat parts of their ration if differing grains are available, and that this behaviour quickly leads to nutritional deficiencies.

External environmental factors can also influence growth rates, with Angel (1996) demonstrating that chicks reared in summer (average temperature 15.4^{o} - 30.3^{o}C) grew much better than those reared in winter (average temperature -0.65^{o} - -6.2^{o}C). At the same time, Aarons (1996) reports that temperatures over 37.7^{o} C will see a drop in growth rates as chicks spend more time panting, and less time eating. Hicks (pers. comm., 1995) stated that with environmental temperatures over 41^{o}C, feed consumption by chicks could be reduced by as much as 90%.

Factors affecting chick viability and growth.

Like other birds, the factors affecting the viability and growth of ostrich chicks can be broken into three groups: pre-laying, incubation, and post-hatch (Doneley, 1992).

1. Pre-laying factors.

a) **Genetics**. In one season's production (1996-97) on one farm (total number of chicks - 451), there were significant differences between ostrich types in relation to chick survivability, with Australian Greys having a higher incidence of leg rotation, yolk sac retention and "fading chick syndrome" than other types and crosses (Doneley, pers obs.). Australian Greys also tended to lay smaller eggs, with smaller chicks resulting. In general, in Australian conditions, African Blacks, African Black - Australian Grey crosses (known as F1's) and Blue and Red ostriches do better than Australian Greys. This is undoubtedly due to the limited gene pool introduced into Australia in the early 1900's, and subsequently maintained under feral conditions.

b) **Age of the hen**. Younger hens (<3 years) tend to produce lower numbers of poorer quality eggs and chicks.

c) **Maternal nutrition**. As discussed earlier, there is a direct effect by maternal nutrition on chick viability. On one farm visited by the author, a change in breeder diet from a pelleted ration to a cheaper grain-based ration resulted in significantly smaller chicks with a higher mortality.

d) **Maternal health and body condition**. Obesity, low grade infections, poor body condition, etc. can all have an effect on the chick through the nutritional content of the egg.

e) **Stage of the egg production season**. Eggs laid late in the season tend to have a poorer hatchability and less viable chicks than those laid earlier in the season. This may be due to a depletion in the quality of albumen as the season progresses. Eggs laid early in the season are often smaller than those laid later.

f) **Egg size**. Smith *et al* (1995) claim that egg size as a production parameter has a repeatability of 74%. Large eggs, because of their small surface area to volume ratio, tend to lose insufficient weight during incubation, resulting in weak, oedematous chicks. Conversely, small eggs lose too much weight, resulting in weak, dehydrated chicks.

2. Incubation Factors.

The artificial incubation of ostrich eggs introduces many variables that can have a direct or indirect effect on the survivability and viability of chicks. The incubator temperature, humidity, air flow, and hygiene, and the positioning and turning of eggs, can all affect not just the hatchability of the eggs, but also the viability of those chicks that do manage to hatch in less than ideal conditions. The table below illustrates the effects of various incubation parameters.

Incubation parameter	Variation	Effect
Temperature	Too high	Early hatch external yolk sac malformed chicks oedematous chicks
	Too low	late hatch yolk sac retention
Humidity	Too high	oedematous chicks yolk sac retention
	Too low	early hatch
Ventilation	insufficient	oedematous chicks
Positioning	horizontal after first 1-2 weeks	malpositioned chicks malformed chicks
Turning	too much	toe abnormalities - flexure - rolling malpositioning malformation
	too little	malposition
Hygiene	inadequate	yolk sac retention

Table 2. The effect of various incubation parameters on ostrich chicks

It should be stressed from this table that anything making the development and hatching of the chick less than ideal will have a direct effect on the viability of that chick. For example, malpositions can make hatching difficult, if not impossible, and result in an exhausted chick, as does dehydration and oedema..

3. Post-hatch factors.

Although precocial, the artificially incubated ostrich chick is totally dependent on the farmer for feed and shelter. This, along with the chick's special requirements, needs to be recognised and catered for by the farmer.

a) **Nutrition**. This will be discussed in greater detail further in this paper.

b) **Stressor**s. Anything that diverts the chick's resources away from growing is a stressor. Conditions such as temperature extremes, poor ventilation, hygiene, overcrowding, excessive movement of chicks, external disturbances, etc. will all stress the growing chick, and have an adverse effect on its growth and viability.

c) **Disease control**. Remembering that the ostrich chick is virtually immuno-incompetent, the importance of high standards of disease control becomes obvious. Quarantine and other management factors need to be addressed, as well as reducing the strain on the chick by dealing with the nutritional and stress factors described above.

These factors will be addressed in more detail in the next part of this paper.

Principles of successful chick rearing.

There are many different ways of rearing ostrich chicks, varying from farm to farm and country to country. The effects of climate, vegetation, available feeds, differing levels of experience, and even the financial value of the bird have led to such a wide variety. However, underlying the success achieved by many farms are four common threads to rearing chicks in an economical fashion:

a. Rear chicks in a stress free environment;
b. Feed them properly;
c. Control disease; and
d. Do not use drugs and supplements to hide poor management.

a. Rear chicks in a stress free environment.

When chicks become stressed, body resources are diverted away from growing, leading to immunosuppression and poor performance. As growth rates drop, so does the health of the chicks. Sick chicks become multipliers of disease, and thus can start to affect healthy chicks, and worsen the situation. Maintaining chicks in a stress free environment is therefore essential to maintaining adequate growth rates and a healthy flock. Some of the factors that cause stress to growing ostrich chicks are:

i. **Temperature**. As discussed previously, very low and very high temperatures have an adverse effect on feed intake and metabolism. Many farmers when constructing chick sheds devote inordinate attention to warming chick pens, but pay little or no attention to cooling them. Ideally, chick sheds should be kept at 23^{o} - 25^{o}C. Focal heat sources (lamps, heaters, etc.) can then provide localised heated areas up to 30^{o} - 32^{o}C. This creates a temperature gradient within the pen, allowing the chicks to find the temperature that suits

them. Outside the shed, shade in the runs is essential. Feed and water placed in this shade attracts the chicks, and thus encourages exercise as chicks move from shaded area to shaded area. Aarons (1996) describes 3 nutritional deficiencies that he feels may be the result of heat oxidation of feed left out in the sun. There is considerable expertise in the pig and poultry industries in the temperature control of animal buildings, and this should not be ignored.

ii. **Ventilation**. Ammonia toxicity is well known as an inhibitor of growth, causing anorexia and depression, eye problems and respiratory disease. Wade (1995) quotes air flows of 0.012 cubic feet per minute per pound bodyweight per ^{o}F (this is equivalent to 0.036 cubic metres per minute per 45 Kgs per ^{o}F). In practical terms, however, if the smell of ammonia is very strong when the shed is opened in the morning, and the chicks are lethargic when let out, then the ventilation is inadequate. (Healthy chicks, when let out in the morning, should race down the run, do a little dance, and run back.) As well as the effect on growth rates, poor ventilation frequently leads to a higher incidence of Aspergillosis, which may not become apparent until the carcass is condemned at slaughter. When planning the ventilation of a chick shed, it is wise to bring fresh air in at the chick's level, not at human head height. Pig and poultry industry consultants have a wealth of knowledge on ventilation, and should be consulted wherever possible.

iii. **Stocking density**. Overcrowding is one of the most common management mistakes seen in practice. Overcrowding causes ventilation problems and social problems, leading to severe stress and subsequent poor growth rates and increased mortality. Wade (1995) recommends shed floor space of 0.3 - 1.4 m^2 per chick, the author finds that 1 - 1.5 m^2 prevents overcrowding stress (i.e a pen measuring 5m x 2 m can hold 10 - 15 chicks. Above that number, and mortalities and stunting will start to occur). Exercise runs need to be adequate, and should be long and narrow, rather than square. Allow 15 - 35 m^2 per chick. A run measuring 70-100m x 3-5 m is usually adequate. It must be well shaded, and have feed and water points along its length to encourage chicks to exercise.

iv. **Hygiene**. Given that until 6 weeks of age, the chick is virtually immuno-incompetent, hygiene is of paramount importance. This can not be stressed too strongly to most farmers, many of who have had no previous livestock experience and little or no understanding of bio-security. Hygiene will be discussed further under "Disease Control".

v. **Exercise**. Leg problems are common in ostrich chicks from 3 weeks of age, and appear to be the result of a combination of fast-growing birds, fed on a high energy diet (to encourage fast growth) and then restricted in exercise. The result is tibiotarsal rotation and/or rolled toes. Long runs (see above) must be provided, and the chicks encouraged to use them. As well as feed, water and shade, the chicks may have to be "walked". Ostriches are very gregarious creatures, and will often see people as maternal figures to be followed. Good handlers will walk their charges up and down the pen several times daily. Remember, in the wild, chicks follow their parents all day, covering distances of 15 - 30 kms each day. Except in extreme weather (too hot; too cold; too wet) chicks should be locked out of their pens by day, forcing them to use the exercise run.

vi. **Nutrition**. Inadequate diets are a major stressor, and this will be discussed in more detail later. Medications and supplements can often be a significant source of stress, and will

also be discussed later.

vii. **Movement**. Many farmers have reported a drop or flattening out of growth rates for a few days after chicks have been moved from one pen to another. Undoubtedly, this is a stress response to moving into unfamiliar territory, and having to re-locate feed and water, and possibly re-establish a pecking order. This is one reason for adopting an All In - All Out policy, i.e. chicks go into one pen until they are 6 - 8 weeks old, and are then moved to a larger grower pen until 12 weeks, and then to juvenile pens. This policy can be maintained in mobile sheds, by keeping the shed within the same run and only moving short distances at a time.

viii. **Social stresses**. Like any other animal, ostrich chicks quickly establish a pecking order. While generally this does not cause problems, if management errors are being made, this could compound them. Deeming and Ayres (1994) showed that "mixed weight" groups of chicks grew more slowly than "same weight" groups. Deeming (1996) showed that this was the result of larger chicks pecking at smaller chicks instead of eating. Therefore, chicks should be batched according to size. The author recommends that chicks be placed in a brooder after hatch for up to one week. This allows chicks to socialise and learn how to eat. After that, they can be batched by size, and left together until 6 weeks, when they can then be re-batched by size again. As mentioned earlier, chicks are a very social animal, and the presence of a "maternal" figure, usually the person responsible for the chick shed, can be a very settling influence. This maternal figure needs to move calmly and confidently, talk to the chicks, and allow them to relate to him/her.

b. Feed them properly.

Ostriches, in many ways, have a digestive tract similar to that of many other avian species. They do not have a crop, but the large proventriculus appears to compensate for this. The proventriculus, as well as storage, also acts to begin digestion, and there is some evidence that foregut fermentation may occur there in adults. The smaller, thick walled ventriculus , with the aid of grit, grinds the food up for passage into the duodenum. The small intestine forms 36% of the length of the intestinal tract (in comparison to chickens, where it makes up 90%). The paired caecae and the long large intestine are responsible for fibre fermentation and water resorption. (Note however, that fibre fermentation and utilisation does not begin fully until 12 weeks of age). Passage times in young birds can be up to 36 hours, increasing to 48 hours as the bird gets older. (Angel *et al*, 1996).

As mentioned earlier, there are several differences between chicks and adults in the digestion and absorption of feed. Fat, fat-soluble vitamins and fibre cannot be utilised effectively until the chick is 12 weeks old. In addition, the behaviour of feeding chicks has to be taken into account. In one study of 20 chicks aged 7 - 14 days, it was found that they spent 27.7% of their time pecking at items on the floor, 23.1% walking, 11.2% under the brooder lamp, 3.5% eating from a bowl, and 10.2% pecking at other objects. (Bubier *et al*, 1996). Another study (Deeming *et al*, 1996) found that given a choice, chicks will eat green lucerne chaff in preference to pelleted feed. Numerous farmers have commented on chicks' preference for mashes and small pellets compared to larger pellets and grain mixes. (Doneley, pers. ob.)These behaviours and differences in digestion have to be taken into account when formulating rations for chicks.

Several manufacturers now produce a pre-starter ration (0-8 weeks) and a starter ration (8 weeks - 4 months). Typically, these contain approximately 23% and 19% protein (respectively), 12.5 MJ/Kg and 11.5 MJ/Kg (respectively) and 1.4% Calcium and 0.43% Phosphorous (Cilliers *et al*, 1994). Several writers have advocated reducing protein levels to avoid angular leg deformities (ALD's), but Angel (1994) showed there were no significant differences in the incidence of ALD's between chicks fed on 14.5% protein and those on 17% protein, but that there were significant differences in growth rates between the two groups. The author's experience is that energy levels and exercise are more important in the pathogenesis of ALD's.

From a practical viewpoint, pre-starter rations are generally small pellets or a mash. This is preferable to grain mixes, where chicks have been known to pick out their "favourite" grains and discard the rest, throwing the balance of the ration out. They can be supplemented with finely chopped lucerne chaff (starting at 4% in young chicks, and gradually increasing as the chick gets older). This fibre, while not useable as an energy source until 12 weeks, helps to "train" the intestinal tract and encourages the growth of the bacteria needed for the fermentation process. It also attracts the chicks to the feed. Grit can be provided, in the form of small pebbles up to 3mm in diameter. This grit should be mixed in with the feed, or scattered over the floor of the shed and run. The practice of providing grit in a separate container should be discouraged because of the risk of proventricular impaction.

Placing the chicks in a brooder for their first week encourages them to recognise and eat their feed. Brooders can be constructed in the pen, and act to confine the chicks while they learn to eat and socialise. After this first week, when the weight gains are on the rise, chicks can be given free range to outside runs. These runs should be sown with short grasses and lucerne. As the chicks have "learnt" to eat the offered feed, they rarely have problems with fibre impaction , but this needs to be monitored closely for the first few weeks. Ample low feed dishes scattered around the run, under shade, also help to prevent this problem.

Ostrich chicks require large amounts of fresh cool water. Their water intake is generally 2.6 times their dry food intake, with more required as salt and fibre intake increases (Angel, 1996). Water quality is important. Total Dissolved Solids (TDS) should not exceed 3,000 ppm. Composition of these TDS is equally important, and should follow guidelines laid down for poultry. Bacterial contamination can be significant, and outbreaks of *Pseudomonas* septicaemia can often be traced back to contaminated drinking water (Doneley, pers. ob.).

c. Control disease.

Without doubt, if the first two principles discussed above are been followed, then disease control has already being implemented. There is no doubt that a well-fed, non-stressed chick is better able to resist disease. In fact, several authors believe that virtually all disease problems in ostrich chicks can be traced back to faults in management or nutrition (Madeiros, 1996)

Disease control in ostrich chicks needs to revolve around two key goals:

1. Disease-causing organisms should not enter the chick facility in the first place; and
2. If there is an outbreak, control measures need to be in place to quickly recognise

the problem, and then to isolate and contain it.

Farm management needs to reflect these goals.

The chick facility on a farm should be regarded as a biosecure site, second only to incubation facilities. Disease can enter the facility via:

a. contaminated personnel, clothing, boots and equipment;
b. chicks;
c. free-flying birds, insects and rodents; and
d. mechanical vectors such as wind, drinking water, feed and water run-off from nearby contaminated sites.

Only those personnel who need to be there should enter. Traffic flow on the farm should always dictate that no one can move from grower or breeder pens to the chick facility without showering and changing first. Fresh clothing (overalls) and boots should be provided for all visitors. Footbaths can be used, but their effectiveness is questionable, and may serve more as a physical reminder of the need for biosecurity than they do as an actual protection. Separate equipment should be provided for the facility, and it should always remain there.

Many farmers in Australia still purchase chicks from different sources. Minimum standards for purchased chicks should be set, according to the age and type of ostrich. Strict quarantine and testing procedures must be implemented, with the testing geared towards those pathogens identified as being the most significant. Farmers should be encouraged to limit their purchases from only one or two sources, and to purchase sufficient chicks at a time to form complete batches. Regardless of source, chicks need to be batched, and traffic flow in the facility should move from most susceptible (youngest) to least susceptible (oldest).

Insect and vermin control need careful attention. Careful and methodical cleaning of pens and careful storage of feed needs to be given primary consideration. Electronic mosquito “zappers”, fly bait stations, etc. should be used to minimise insect vectors.

The facility needs to be sited where wind and water runoff are unlikely to introduce possible pathogens. Only good quality feed and water should be used, and water quality should be tested on a regular basis.

In the event of a disease outbreak occurring, it is essential that systems already be in place to deal with it. Staff must be trained in the early recognition of illness, and be familiar with the “normal” - behaviour, appetite, droppings, etc. Once a problem has been detected steps must be taken immediately to identify it, isolate affected chicks, and contain the spread of the disease. The services of an experienced ostrich veterinarian and a well-equipped veterinary laboratory are essential. Sick pens should be part of the facility design, so that affected birds can be removed from the remainder and isolated. An All In - All Out batching system will help to contain an outbreak to a minimum. Cleaning and disinfection, using suitable chemicals, should be implemented, with care being taken that runoff from the cleaning does not wash into adjoining pens.

Even in the best facilities, a build up of potential pathogens is certain to occur in each pen.

Flooring will play a major role in this. Flooring can be concrete, sand, compressed road gravel (or similar material), or dirt. The latter two surfaces are virtually impossible to clean. Concrete floors, while relatively easy to clean, may attribute to some leg problems, and need an overlay (outdoor carpet, synthetic non-slip flooring materials) to prevent heat loss at night. These overlays need to be cleaned on a daily basis. Sand can absorb urine, and raking it helps to minimise the build up of pathogens. However, it is often associated with eye problems and impaction. The use of hay or straw should be discouraged, because of the potential for Aspergillus to flourish, and the risk of impaction. Mobile pens offer the opportunity to have fresh pasture as a floor, but require plenty of room. There should be sufficient pen and run space to cater for one or more pens to be spelled at any one time. Pens should be spelled for 30 - 60 days.

d. Do not use drugs or supplements to disguise poor management.

Veterinarians should not encourage the indiscriminate use of drugs on an ostrich farm. If problems on a farm are persistent, then a farm visit should be mandatory to look at management, nutrition and hygiene. This needs to be emphasised to the farmer. As the Australian industry moves towards the commercial phase, antibiotic and other residues in meat are becoming important. Madeiros (1996) feels that of chick deaths under 30 days, 40% are due to poor management, 30% are due to poor egg quality, and the remaining 30% are due to poor incubation/hatching techniques. This author feels that more weighting should be given to poor management, but agrees with the basic causes. Very few, if any, problems with ostrich chick rearing are not directly attributable to management errors.

When the ostrich industry was in its heyday, many entrepreneurs saw the opportunity to provide supplements to farmers. Many of these supplements have no scientific rationale behind them, but when ostrich chicks were worth thousands of dollars, anything was worth a try. In the current economic climate, farmers need to evaluate all supplements critically, and assess whether the return from the use of a particular supplement justifies its cost. Veterinarians can assist in this process by designing on-farm trials, tailored to that farm's management practices.

The above principles should be used by farmers when planning their enterprise, and by veterinarians seeking to critically evaluate a problem farm. There are almost as many ways of achieving each principle as there are farms, but the basic concept behind each one remains constant.

Diseases of ostrich chicks.

Ostrich chicks, being immuno-incompetent, are prone to a wide range of diseases and other disorders. These can be categorised as follows.

1. bacterial disease
2. viral disease
3. fungal disease
4. parasitic disease
5. nutritional disease
6. orthopaedic problems
7. miscellaneous problems

1. Bacterial disease.

Most of the bacteria that cause problems with ostrich chicks are generally considered to be secondary pathogens. However, given the immune status of the chicks, occasionally they can be primary. Many infections are associated with less than ideal management.

a) ***Pseudomonas aeruginosa***. *Pseudomonas* is a water-borne pathogen, common in most water sources. It grows particularly well in plastic and PVC pipes and containers. Clinical signs seen in affected birds include conjunctivitis, corneal ulceration, dyspnoea, pharyngitis, swollen joints, and sudden death. Generally, the infection spreads quickly through in-contact birds. On autopsy, pharyngitis (with abscessation), air sacculitis, pneumonia and hepatic abscessation are common. Culture of *Pseudomonas* in a veterinary laboratory is relatively easy.

Treatment is generally frustrating and unsuccessful. Enrofloxacin and gentamycin have been used, but if the septicaemic form is present, death generally results. Work with autogenous vaccines has been inconclusive (Foggins, 1995).

Isolation of affected birds, moving unaffected birds to a clean pen (where possible), and disinfection are the only possible control measures when dealing with an outbreak of Pseudomonas septicaemia. Suitable disinfectants include sodium hypochlorite, quaternary ammonium compounds and glutaraldehyde. Attention needs to be paid to flushing water pipes before filling containers, and routine disinfection of water containers. Chlorhexidine (Avi-Clens^{R} - Vetafarm) is not effective against *Pseudomonas*.

In Zimbabwe, farms which have experienced an outbreak of Pseudomonas septicaemia are not allowed to sell chicks under 12 months of age, unless directly for slaughter. (Foggins, 1995).

b). ***E. coli***. Colibacillosis is one of the more common diseases seen in intensive chick rearing. *E. coli* is a normal component of the chick's gastrointestinal flora, but under conditions of stress and poor hygiene it can become an opportunist pathogen. Transmission is via ingestion, either of contaminated feed or water, or faeces from infected birds. Poor hygiene can result in the introduction of unfamiliar strains into the incubation complex or the chick sheds.

In newly hatched chicks, *E. coli* can cause navel infections and subsequent omphalitis (see later). Older chicks frequently develop diarrhoea and/or green urates. This can sometimes be seen adhering to the chicks' perineum. Autopsy shows enteritis and often multifocal hepatic granulomas. On histopathology, there are usually multifocal area of hepatic necrosis. Often there are no bacteria associated with these hepatic lesions, and it is possible they are the result of endotoxaemia.

The author's experience is that culture and sensitivity to determine the appropriate antibiotic, combined with stringent hygiene, best management practices and isolation of affected chicks is the best approach to controlling an outbreak. Autogenous vaccines (Ausvac - Australia) have been used, with variable results.

c) ***Salmonella***. Salmonella has been reported world wide as causing sporadic outbreaks of disease. Transmission has been described as both horizontal (via either clinically affected chicks or asymptomatic carriers) and vertically. Because of the presence of carrier birds, vertical transmission, reservoirs such as mammals and birds, and the organism's ability to survive in the soil for long periods, this disease may be difficult to eradicate.

Death can be sudden, or be preceded by diarrhoea, weight loss and lethargy. Diagnosis is by culture.

Treatment is indicated by the sensitivity testing, but consideration must be given to the creation of a carrier state.

Strict quarantine protocols are necessary to prevent its entry onto a farm.

d) ***Clostridia***. *C. perfringens* is often implicated as the cause of sudden death in young chicks. In most cases, there are few ante-mortem clinical signs. Post-mortem shows varying degrees of haemorrhagic enteritis. Care must be taken not to confuse clinical disease with post-mortem autolysis and proliferation of what is normal intestinal flora.

Clostridia is a common organism on farm soil, particularly if large animals have grazed there previously. The organism can remain viable in the soil for long periods of time. It is also often cultured from the droppings of healthy chicks. This would indicate that it is a commensal pathogen, causing disease only when other factors come into play. Some of these factors include an abrupt change in diet, concurrent enteritis, starvation, stress, poor hygiene and overuse of antibiotics.

Treatment of the individual is usually unsuccessful, but some success has been reported in controlling outbreaks with the use of zinc bacitracin (30 g/tonne feed) (Shane, 1996).

Prevention should revolve around management. There are many anecdotal reports of vaccination using cattle 5-in-1 vaccines, but no trials have been conducted to assess its efficacy.

e) ***Megabacteria***. This has been reported as a cause of gastritis in Australia, Italy, and Africa. It caused severe gastritis, allowing secondary septicaemias to develop. Insufficient cases have been reported to comment on treatment, but it is likely that oral amphotericin-B (Megabac-S, Vetafarm) would be successful.

f) ***Campylobacter***. This has been reported commonly in Australia, often secondary to "Ostrich Fading Syndrome." As it is pathogenic to humans, and poultry meat is regarded as being a possible source in human infection, its potential in a commercial ratite industry should not be overlooked. It causes enteritis and hepatitis, and is transmitted by infected carriers, vermin and free-living birds. Treatment with erythromycin is usually successful. Good hygiene and chlorination of drinking water can limit its spread.

2. Viral Disease.

In Australia to date, only a few viral diseases have been isolated (coronavirus in Victoria, a retrovirus in Western Australia.) Nevertheless, viral diseases are important overseas, and it is necessary to be aware of the potential of the introduction of viral diseases to this country. Below are some of the more common viral diseases, but it must be stressed that this is by no means a complete list of viruses affecting, or likely to affect, ostriches.

a) ***Avian Influenza***. Avian Influenza virus is present in Australia, with free-living and migratory birds acting as a reservoir. Outbreaks are rare, and ostriches are yet to be reported as affected in this country. Outbreaks in South Africa and Israel have been reported as causing 80% - 90% mortality. Infection can be direct (carrier bird mingling with flock) or indirect (contaminated feed, equipment, transport, etc.). Infected birds show acute mortality with dyspnoea and polyuria, with biliverdinuria. Younger birds appear to be more susceptible than mature birds. Post mortem examination shows multifocal hepatic necrosis, splenomegaly, nephrosis and air sacculitis. In the event of an outbreak in Australia, control measures would be left to the local government veterinary officer, and would entail quarantine, testing and destruction of affected birds.

b) ***Newcastle Disease***. This viral disease is exotic to Australia, although lentogenic (mild) strains have been isolated from ostriches in Australia. Allwright (1996) reports a definite age-related susceptibility, with birds under 6 months being at greatest risk. In these birds the most common clinical sign is acute death (up to 80% mortality), although some will develop neurological signs (torticollis, inco-ordination, recumbency). Adult birds develop a swollen head, neurological signs and a productive cough. In young birds pathology is generally limited to the brainstem and cerebellum. Testing and control measures of this disease would primarily be up to government veterinary officers.

c) ***Coronaviral enteritis***. There have been reports of coronavirus associated with enteritis in young chicks in Victoria (Gestier, pers. comm.). This is consistent with reports from South Africa. Clinical signs vary from acute outbreaks of enteritis to chronic “fading” chicks. Secondary clostridial necrotic enteritis is not uncommon. In South Africa it is thought that newly purchased ostriches are the most common source of infection. Good biosecurity and management appears to be the only means of preventing or containing an outbreak.

d) ***Adenovirus***. Adenovirus has been isolated from ostrich chicks showing high mortality in Okalahoma and adjoining states. Adenoviruses can be transmitted vertically and horizontally. Affected chicks show marked depression and malodorous chalky-grey diarrhoea, with mortality up to 90%. On post-mortem, multifocal enteritis, air sacculitis and pulmonary congestion are seen.

e) ***Retrovirus***. Western Australian workers have identified retroviral particles in tissues from some chicks affected with the so-called “Ostrich Fading Syndrome”. At the time of writing, it is generally believed that this retrovirus may be one of many contributing factors to the syndrome (Black, pers. comm.).

f) ***Ostrich pox***. Many Australian farms have been affected by ostrich pox, with cutaneous lesions on the featherless areas on the face. Eyelids can be badly scarred, with resultant eye problems. Spread is via biting insects or pecking amongst chicks. Most lesions

spontaneously resolve. Fowl pox vaccine appears to be effective in controlling and preventing outbreaks.

g) ***Borna Disease***. Borna disease virus has been reported to cause high mortalities in ostrich chicks in Israel. Insect vectors are the likely mode of transmission. Affected chicks progressively become paralysed until death from dehydration occurs. No specific lesions are seen on post-mortem. The Israelis use an ELISA test to detect the virus in brain tissue. Supportive therapy and hyperimmune serum can be beneficial in early cases.

3. Fungal disease.

In Australia, Aspergillosis and Candidiasis are the two most important fungal diseases. In Israel and the USA, zygomycosis is also reported as a problem.

a) ***Aspergillosis.*** *A. fumigatus* is the most common cause of aspergillosis, although *A. flavus, A. niger* and *A. nidulans* are also occasionally isolated. (Love, 1995) Although in Australia, aspergillosis is seen as a chronic respiratory disease of mature birds, it can also appear as an acute respiratory disease in young chicks ("brooder pneumonia"). Infection can be transmitted via exposure of the embryo to fungal spores (through cracked or drilled shells - some farmers will drill holes in eggs that are losing insufficient weight), or by the chick inhaling large quantities of spores in a contaminated hatcher or brooder shed. The number of spores inhaled and the state of the chick's immune system will determine the onset and severity of clinical signs. In many cases, the author believes that sub-clinical infections are acquired as chicks, and do not become apparent for many months, or even years, until the bird is placed in a stressful situation. In those cases where high levels of spores are present in the chick's environment, and/or the chicks are under a lot of stress, a more acute syndrome presents. These chicks fail to thrive, are dyspnoeic and die within a few days or weeks. Post-mortem reveals numerous fungal granulomas in the lungs and air sacs. Treatment is not warranted due to the poor response rate and high cost of treatment. Prevention should revolve around providing adequate ventilation and disinfection with a suitable compound.

b) ***Candidiasis***. This fungal infection of the upper digestive tract reflects management problems - immunosuppression, malnutrition or prolonged antibiotic usage. White to brown plaques on the oral and pharyngeal mucosa, sometimes with a pseudomembranes, are easily detected. Affected chicks are typically under stress, anorectic, and are failing to thrive. Treatment with oral nystatin and chlorhexidine (Avi-Clens, Vetafarm) is usually successful, but underlying causes must be eliminated.

4 Parasitic disease.

In Australia, losses in chicks due to parasites are rare. External parasites, such as lice, are usually not acquired until the chicks are mature enough to mix with adult birds. Wireworm (*Libyostrongylus douglassi*) is still uncommon. Cryptosporidia is seen occasionally, associated with "Ostrich Fading Syndrome". South African and American workers (Craig & Diamond, 1996) describe a wide range of parasites affecting ostriches, but only a few are seen in Australian conditions.

a) ***Wireworm****. L. douglassi* is present in Australia, but is believed to be uncommon. The worm is asymptomatic in adults, and eggs and larvae passed in the faeces can remain infective for several years. Chicks that have access to faeces or contaminated pasture can develop a diphtheritic proventriculitis ("vrotmaag" - rotten stomach). Affected chicks show wasting, anorexia, anaemia and death (up to 50% mortality). Treatment with fenbendazole, levamisole and ivermectin is generally effective. Prevention should revolve around not allowing chicks access to infective material. For this reason, the feeding of adult droppings to chicks as a probiotic should be discouraged. Newly purchased birds should be quarantined for at least 30 - 35 days (the pre-patent period for this parasite), and during this time their droppings should be repeatedly tested (by faecal culture and larval identification). Birds testing positive should be treated and their droppings re-tested. All droppings should be collected and burnt every 48 hours.

b) ***Cryptosporidia***. Several clinicians in Australia have isolated *Cryptosporidia spp*. from the intestinal wall of "fading chicks". In these cases it appears to be secondary to other stressful factors, and causes enteritis, weight loss and eventually death. Recent work in the USA (Clubb *et al*, 1996) suggests that paromomycin may be a suitable treatment.

5. Nutritional Disease.

Nutritional diseases result from either deficiencies or excesses of nutrients. In intensive ostrich farming, these imbalances can occur readily when improper feed is used, feed is stored incorrectly or for too long, supplements are added, or when antimicrobials are used inappropriately. As research is still being conducted into the nutritional requirements of ostriches, imbalances are all to frequent. Some of the more common of these imbalances are:

a) ***Calcium - phosphorous imbalances***. Rickets particularly occurs when grain based diets are fed to chicks. The chicks eat the grain, but ignore the pelleted supplement meant to balance the diet. Other imbalances occur when too much lucerne (high in calcium) is consumed, calcium supplements added without proper calculation of requirements, or when feeds high in oxalates are fed (e.g. Buffel Grass)The result is bowed legs, weak bones, poor growth rates, etc. It may be a contributing factor to some cases of leg rotation.

b) ***Hyperkeratosis***. Thickening and cracking of the feet, and the skin on the neck and the head is seen commonly, although intermittently. This has been attributed to zinc/pantothenic acid deficiencies, mycotic infections, Vitamin A deficiencies (resulting from improper storage), or zinc deficiency. In all probability it is likely that the problem is multi-factorial, but in most cases, it does respond rapidly to biotin supplementation.

c) ***Cardiomyopathy***. Vitamin E responsive cardiomyopathy has been diagnosed on several occasions in ostriches, including juveniles. Care must be taken in the diagnosis of this condition, as post-mortem autolysis of myocardium has been confused with cardiomyopathy.

6. Orthopaedic problems.

With the rapid growth rate of the ostrich chick, it is not surprising to see a high incidence of growth disorders. A survey in Queensland in 1994 showed 80% of chick losses over 3 months of age

were attributable to leg abnormalities (Moore, pers. comm.)

a) ***Angular Limb Deformities***. More commonly known in Australia as “turned leg”, ALD’s are commonly encountered. Lateral rotation of the tibiotarsus and/or tarsometatarsus can start to occur as early as 3 weeks of age, but never after 6 months. Many theories are put forward as to the cause, including genetic, nutritional, exercise, and flooring. The author’s belief is that the cause most commonly lies with a with a genetic predisposition to grow rapidly, combined with a high energy diet (allowing rapid growth), then compromised by insufficient exercise. Other workers have shown that protein levels have little effect on the incidence of ALD’s, and that heated floors will increase the incidence. This is thought to be due to direct damage to the growth plates by the heat, but it may also be due to a reluctance of the chick to get up and exercise. Some farmers believe that smooth concrete floors are a contributing factor, but this is unlikely. Regardless of the cause, most workers agree that treatment is rarely successful, and prevention is preferable. Growth rates need to be monitored, and if chicks are growing too rapidly (as evidenced by an increased incidence of ALD’s) dietary energy should be reduced and/or exercise increased.

b) ***Splay leg***. Coxo-femoral subluxation resulting in inability to adduct the legs is uncommon. Oedematous chicks and chicks with large yolk sacs may be more susceptible. Hobbling of the legs at an early stage is usually successful. This can be done by joining velcro leg bands on each leg with a suitably sized heavy duty rubber band.

c) ***Rolled toes***. This author recognises two rolled toe syndromes. The first is seen at hatch, or within a few days. This is almost certainly due to incubation factors, such as type and frequency of egg turning. The second starts to occur from 3 weeks of age, and is probably a variation of ALD’s, although some workers suspect that nutritional deficiencies may be involved. Instability of the interphalangeal joints results in the toe rolling outwards. Left untreated, it often results in a club foot. Corrective splinting applied early in the course of the problem usually (not always) results in straightening of the toe. In the first syndrome, adhesive tape wrapped around the toe in the opposite direction to the rotation is usually sufficient. More advanced cases, especially those seen after 3 weeks, require an inverted `L’ splint, with the long bar under the major digit and the short pointing medially from the tip of the digit. As the foot is placed to the ground the short bar strikes the ground first and pushes the toe back to a more normal position.

d) ***Slipped tendon***. Lateral dislocation of the gastrocnemius tendon as it passes over the caudal aspect of the tibiotarsal-tarsometatarsal joint (hock) is uncommon. It has been attributed to genetics, nutritional deficiencies (manganese, copper, biotin and choline), and improper substrate. Treatment requires surgery to repair torn tendon sheaths, but in the author’s experience is rarely successful. As soon as the chick starts walking again, the repaired area tears again. Given the economic value of chicks, affected birds should probably be euthanased.

7. Miscellaneous problems.

a) ***Yolk sac retention***. This needs to be distinguished from yolk sac infection (see below). Immediately after hatching, the chick is dependent on the yolk sac for nutrition and maternal immunity. As the yolk is utilised, the yolk sac becomes smaller. By 14 days, on

average, it is marble-sized or smaller. By 21 days it is usually smaller than a pea. Chicks that hatch under sub-optimal conditions (insufficient/excessive weight loss, inadequate ventilation, etc) do not absorb their yolk sac as well, resulting in yolk sac retention. These are usually aseptic. Typically, these chicks will have irregular growth rates for the first 10 - 18 days, and will then markedly deteriorate and die within 2 days. The retained yolk sac is often palpable by an experienced handler. The cause of death is unclear, with some workers suggesting a “toxic metabolite” is released by the yolk, while others suggest the retained yolk sac interferes with respiration and causes death by suffocation. Early recognition and surgical removal, combined with intensive supportive therapy, can save many of these chicks. However, given the value of the chicks and the high mortality rate, surgery is unlikely to be considered as a future option. High numbers of retained yolk sacs (>2%-5%) warrant investigation.

b) ***Yolk sac infection***. Unlike retention, omphalitis is a septic problem. Chicks that hatch out weak due to incubation problems, or whose navels have not closed completely, are prone to infection. This infection typically arises from contaminated hatchers or brooders, although transovarian, oviductal or cloacal contamination can be important. Infected chicks will be depressed and anorectic, showing signs from as early as 3 days and as late as 3 weeks. Antibiotic therapy without surgical removal of the yolk sac is pointless. (see above comments on the practicality of surgery.) Prevention by maintaining strict hygiene in the hatchers and brooders, and prophylactic application of betadine ointment and/or bandages to the navel at hatch is usually beneficial. Infection rates above 2%-5% warrant investigation.

c) ***Proventricular impaction***. Many farmers will diagnose a chick mortality as “impaction” and regard that as the primary diagnosis. Impaction is rarely primary; there is nearly always an underlying cause that may not be readily apparent. (Some chicks, when placed in a new environment for the first time, will overeat the substrate - sand gravel, stalky grass, etc. This could perhaps be described as primary impaction.) Stress, disease and poor management can lead to the ingestion of inappropriate material (foreign bodies, sand, gravel, fibrous material), which then forms a tightly bound mass in the proventriculus. At the same time, illness, dehydration or dietary deficiencies can cause weakened gizzard contractions. The end result is reduced passage of ingesta through the gizzard, and the mass in the proventriculus dries out and enlarges as more material is added. The chick loses weight, its appetite declines and faecal production drops. In days, weeks or months the chick will die, basically of starvation. Diagnosis can be made on the basis of weight loss, aimless pecking at food, reduced faecal output and palpation of the impacted mass in the proventriculus. Treatments described include proventriculotomy, flushing the proventriculus with running water (Putter, 1996), or oral drenches with pysllium or mineral oil. Supportive treatment including fluids and antibiotics, as well as determining the primary cause, is essential. The author’s preferred treatment for sand/gravel impactions is oral electrolytes (10% of the chick’s bodyweight, divided into 2-3 daily drenches) combined with pysllium or Tympanyl to break down the impacted mass. After 3-4 days, paraffin oil (10mls/kg) is given to start pushing the mass through the gizzard. Fibrous impactions invariably require surgery, and as such may not be economically viable.

d) ***Cloacal prolapse***. Enteritis, sand ingestion, cryptosporidia and impaction are frequent

causes of cloacal prolapse. The exposed tissue should be cleaned, gently debrided as appropriate and then replaced. The prolapse is then kept in with a simple purse string suture, and an antibiotic - anti-inflammatory ointment applied if necessary. This can usually be done with physical restraint alone. The primary problem can then be addressed.

e) ***Oedematous chicks***. So-called "wet chicks" are the result of insufficient weight loss during incubation (humidity too high) or inadequate ventilation. The oedema is predominantly over the thighs and the back of the head, although the whole body may be affected. These chicks are prone to a range of problems, and rarely do well. Some farmers have found that furosemide given at hatch can cause a rapid reduction in the oedema, but this treatment should be approached with caution. (However, there may not be much to lose given the current value of ostrich chicks.)

f) ***Intestinal accidents***. Intussusception and torsion are infrequently found at post-mortem as a cause of death. Enteritis, impaction, and abrupt feed changes may be predisposing causes.

g) ***Fading chicks***. Some workers feel that Ostrich Fading Syndrome (OFS), as reported in Australia, is identical to Fading Chick Syndrome seen elsewhere in the world; others feel there are slight but significant differences. Both syndromes are seen in chicks from 3 weeks on. Typically affected chicks become lethargic and anorectic. Weight loss, pale mucous membranes and ascites then develop. A non-regenerative anaemia and hypoproteinaemia is usually present. Gross pathology usually shows ascites, proventricular impaction (usually with ingesta), small white - yellow lesions on the liver, and often numerous other non-specific changes. OFS is characterised by a non-suppurative enteritis and often a multifocal hepatic coagulative necrosis. Numerous different pathogens, usually secondary, are identified, suggesting immunosuppression. Epidemiological studies in Australia suggest an infectious aetiology, but a consistent pathogen has yet to be identified. (Button *et al*, 1996) Aarons (1996) suggests overheating as a possible cause, and the "outbreaks" in Australia certainly coincided with the hot summer months. However, until a definite aetiology has been determined, it would be wise to assume that an infectious agent is involved, and utilise biosecurity to keep any possible infection out.

This paper has attempted to summarise much of the current knowledge on ostrich paediatrics in Australia at the time of writing. It is, however, a constantly changing field as more research and more knowledge comes to light.

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