TREATMENT OF EBSTEIN’S ANOMALY IN A MEERKAT (Suricata suricatta).

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Abstract
Ebstein’s anomaly is characterized by a downward displacement of the tricuspid valve into the right ventricle due to anomalous attachment of the tricuspid leaflets. An 8-yr-old male meerkat (Suricata suricatta) was presented with sudden lethargy. Thoracic radiograph was consistent with congestive heart failure. An echocardiography revealed an Ebstein’s anomaly. The animal was treated with imidapril chlorydrate. After six mo of treatment, thoracic radiographs still showed right-sided cardiomegaly, however, the animal appeared clinically normal.

Key words: (Suricata suricatta), Ebstein’s anomaly, heart, echocardiography, meerkat

Introduction
Ebstein’s anomaly is characterized by a downward displacement of the tricuspid valve into the right ventricle due to anomalous attachment of the tricuspid leaflets (7). Complications such as right ventricular failure, infective endocarditis, and paradoxical embolism can occur (10).

Material and Methods
A captive born meerkat (Suricata suricatta), aged 8 years, had been submitted for clinical examination. Physical examination was performed under anesthesia with isoflurane. Thoracic radiograph, subcostal thoracocentesis and transthoracic echocardiogram were also conducted.

Results
Case description
The animal lived in a group with two other male captives, born the same day from the same sire. With no prior medical history, he presented with sudden lethargy and difficulty walking. Clinical findings included hypothermia (34.5°C), dyspnea and a systolic murmur over the tricuspid valve. A globular-shaped heart with pleural effusion was observed on thoracic radiographs, suggesting congestive heart failure. Differential diagnoses included right heart failure, pleuritis of bacterial or fungal origin, or thoracic neoplasm. A percutaneous, subcostal thoracocentesis, inserted at the left fourth intercostal space, was performed without any imaging guidance. Two ml of serosanguinous fluid were obtained. The total protein content of the sample was 4.9 g/dL. Unfortunately, no cytological analysis was performed. The protein levels were consistent with a modified transudate which can be consistent with cardiac insufficiency, the most likely differential diagnosis.

A standard transthoracic echocardiogram with Doppler was performed under isoflurane anesthesia, with continuous ECG. M-mode echocardiogram at the ventricular level showed a right ventricular enlargement, with a moderate paradoxical septal motion. Paradoxical septal motion generally occurs as a result of a pressure overload in the right ventricle and an elevation of right ventricular pressure was suspected. Two-dimensional echocardiography revealed a more pronounced right atrial dilatation. The long-axis four-chamber views recorded from right parasternal positions demonstrated that the two atrioventricular valves were abnormal. The mitral valve leaflets were irregularly thickened (maximal thickness of 4.8 mm) and the papillary
muscles appeared hyperechoic and elongated. This “atrialization” of the right ventricle is the typical feature of a variation of tricuspid dysplasia called Ebstein’s anomaly. Moreover, the septal tricuspid leaflet was adhered to the septal wall and seemed to have little movement. The anterior tricuspid leaflet appeared also elongated. Spectral Doppler of diastolic tricuspid inflow showed a normal laminar velocity profile (0.62 m/s and 0.30 m/s for E and A waves, respectively), excluding a diagnosis of tricuspid valve stenosis. An abnormal systolic tricuspid regurgitant flow (maximal velocity of 0.8 m/sec) was identified. This laminar low-velocity tricuspid valve flow was consistent with a low-pressure gradient across the tricuspid valve between the right ventricle and the right atrium during systole and excluded systolic pulmonary arterial hypertension, although it may have been in part related to the potential of systemic hypotension associated with the general anesthesia. A small amount of pericardial fluid (2 mm) was observed cranial to the right outflow tract. The pericardial effusion was considered to be secondary to right sided congestive heart failure and was determined to be clinically insignificant due to the absence of cardiac tamponade or diastolic right atrial collapse. Due to the right heart overload, a dilation of the caudal vena cava without aceites was observed. Based on these ultrasound findings, the final diagnosis was a congenital tricuspid valve malformation called Ebstein's anomaly, associated with a mitral valve dysplasia and an atrial septal defect. Both the atrial septal defect and the tricuspid regurgitation contributed to the severe right heart enlargement.

**Treatment**

The animal was hospitalized in a warm room (24°C) for 10 days and stabilized with dexamethasone 21-isonicotinate (0.16 mg/kg, i.m., once) and with a solution containing ascorbic acid, thiamine, pyridoxine, niacin. Imidapril chlorydrate treatment was initiated (5 mg/kg, p.o., s.i.d., for two mo then 2.5 mg/kg, p.o., s.i.d.). Six months later the animal appeared clinically normal, however radiographs demonstrated that cardiomegaly was still present. Pleural effusion was not noted.

**Discussion**

Ebstein's anomaly has been described in humans and also in other species (1,4,5,7). In most cases, this congenital cardiac anomaly is suspected when a thrill or systolic murmurs over the tricuspid area are detected in young subjects. This case is quite unusual as Ebstein's anomaly expressed itself as an acute syndrome in an adult meerkat without prior symptoms. The abnormal gait was probably due to the weakness associated with the acute right-sided heart failure. This case is also unusual as pleural effusion is not a common finding in the Ebstein's anomaly. There is only one case in the literature that describes the coexistence of clinically important pleural effusion and Ebstein's anomaly (7). Radiography associated with Ebstein's anomaly may show evidence of cardiac enlargement, particularly of the right atrium. Electrocardiography may indicate atrial disease or right atrial enlargement and right ventricular conduction defects or may suggest left ventricular hypertrophy (4). However, as in this case, echocardiography is the method of choice to diagnose Ebstein’s anomaly on its own or in association with other heart defects (7). Furthermore, contrast echocardiography or color-flow Doppler echocardiography are needed to reveal tricuspid regurgitation (5). In humans, the treatment of Ebstein's malformation is surgical. The technique is based on the concept of mobilization of the restrictive anterosuperior leaflet associated with a longitudinal plication of the inlet component of the right ventricle. Additional procedures include tricuspid valvoplasty associated with longitudinal right ventricular plication and valve replacement (2). Even though Ebstein's malformation remains a surgical challenge, the various approaches that have been used appeared appropriate and successful in many human patients (8). Some authors have presented an alternative approach to the management of severe Ebstein's malformation that focuses on both the tricuspid valve and the right ventricle.8 In humans, Ebstein's malformation has also been treated medically with isotroproterenol, a potent pulmonary vasodilator with inotropic and chronotropic effects.9 In veterinary medicine, surgical attempts have not shown the same success (4). We have managed this case with the use of imidapril chlorydrate. Imidapril is a long-acting, non-sulfhydryl angiotensin -converting enzyme (ACE) inhibitor which has been used in humans in the treatment of hypertension, chronic congestive heart failure, acute myocardial infarction, and diabetic nephropathy (6). This drug decreases cardiac input and output, reducing
the systemic blood pressure without increasing cardiac frequency. In dogs, imidapril is used in subjects with heart failure caused by mitral regurgitation or dilative cardiomyopathy at 0.25 mg/kg, p.o., s.i.d. We arbitrarily increased the dosage of imidapril chloride in this animal because in dogs, the bioavailability of the imidapril and imidaprilate decreases with concomitant administration of food. More experience will be necessary to evaluate this strategy in treating Ebstein's anomaly.

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References
NEURODEGENERATIVE DISORDERS IN SNOW LEOPARD CUBS (Uncia uncia)

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Key words: snow leopard, Uncia uncia, neurodegenerative disorders, spinal cord

Extended abstract
This report presents the clinical features and pathological findings of two neurological disorders affecting young snow leopard cubs born in different zoological institutions in Switzerland and France. The first disorder was diagnosed in snow leopard cubs born to three breeding pairs in one Swiss (zoo n°1) and two French zoological institutions (zoos n°2 and 3) between 1997 and 2003. The disorder appeared in two, three, respectively four consecutive litters from each breeding pair and all cubs born in the affected litters developed neurological signs. Beginning at the age of two to four months, the cubs developed locomotional disorders characterized by swayng gait, hypermetria and weakness of the hind limbs, associated with progressive muscle atrophy of the hindquarters. The interpretation of the neurological examination was very difficult, however the spinal reflexes of the pelvic limbs (patellar and flexor reflexes) were present. Due to the progressive nature of the symptoms most cubs were euthanized within one year of age. Necropsies were performed on five animals and did not reveal any gross lesions. Histopathological examination revealed degenerative lesions in the cervical, thoracic and lumbosacral segments of the spinal cord. The lesions were confined to the lateral and ventral columns, with the dorso-lateral and ventro-medial peripheral aspects being most severely affected. The changes were characterized by the dilation of myelin sheaths containing preserved axons, myelinophages or axonal debris, occasional axonal swelling, and decreased density of myelin and axons associated with astrocyte proliferation and perivascular gitter cell cuffs. The severe Wallerian-type degeneration of the axons was better appreciated in the longitudinal sections.

A second neurological syndrome was diagnosed in 3 of 4 cubs born at a different zoological institution in France (zoo n°4) (3). The first cub drowned at two weeks of age during an accidental flooding of the building. At the age of three to five weeks the three remaining cubs started to show clinical neurological symptoms characterized by head and body tremors, swaying gait, followed by inability to stand and paresis of hind limbs. Additional clinical findings were loss of body weight and a shaggy hair coat. Due to the progression of the neurological symptoms all three cubs were euthanized at the age of nine to 11 weeks and...
necropsies performed. Gross examination revealed severe emaciation. The hair coat appeared shaggy due to the presence of small dystrophic, hyperkeratotic hair. No lesions were observed in the other organs. Histopathological investigation of the nervous system revealed lesions characterized by the presence of chromatolytic neurons in the spinal cord, predominantly located in the proprioceptive nucleus thoracicus in the proximal lumbar segments with associated microgliosis. Evidence of neuronal necrosis and neuronophagia was not evident. In one animal there was also distinct myelin sheath dilation and axonal degeneration in the corresponding thoracic and cervical spino cerebellar tracts (dorso-lateral). No changes were seen in the brain, spinal ganglions and peripheral nerves. The first disorder appears to be clinically and pathologically similar to a previously described neurological disorder in snow leopard cubs at the Helsinki Zoo (1). In Helsinki, snow leopard cubs presented with spastic paresis. Pathological lesions from these cubs were characterized by myelin and axonal loss associated with astrocytic proliferation without inflammatory reaction, predominantly in the thoracic and lumbosacral segments of the spinal cord. Furthermore, these cases are similar to the degenerative myelopathy in cheetah, an emerging disease that severely affects the European cheetah population (5,6). To date, the cause of the degenerative lesions in these two species could not be determined. The second syndrome is clearly distinct. Not only are the age of onset, the clinical symptoms and histopathological features distinct, but also the fact that this was an isolated litter from a breeding pair that had already produced several normal litters. This contrasts the situation in the three other institutions where afflicted litters were repeatedly produced. Neurodegenerative diseases characterized by spinal white matter degeneration or neuronal chromatolysis have been described in human and in several animal species, including cheetahs and snow leopards, but the cause is mostly unknown (1,2,4,5,6). Some of these diseases have been shown to have a hereditary basis, but nutritional deficiencies, infectious diseases, intoxication or metabolic disorders were suggested as other aetiologies. The genetic basis of captive snow leopards is narrow. Going back one to five generations, a preliminary pedigree analysis showed that all affected snow leopards from Switzerland and all three French institutions have common ancestors. However, a pure genetic cause is unlikely as the same ancestors appear also frequently in the lineage of unaffected snow leopards of other institutions. It has also to be noted that the snow leopards in the zoos 1, 2, and 3 were fed with chicken only. Zoos n°2 and 3 changed the diet to a variety of different meats supplemented with vitamins and trace elements. The cubs born in these zoos after the diet change did not develop neurological signs, whereas zoo n°1, that continues the feed chicken, didn’t produce any normal litter. This may indicate a possible nutritional factor as cause of the spinal cord degeneration, but further investigations into the clinical, pathologic, and management aspects are needed to elucidate these disorders.

References
MYELOPATHY IN CHEETAH (Acinonyx jubatus) CUBS TREATED WITH CITIDINEDIPHOSPHOCHOLINE (CDP-CHOLINE)

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Abstract
Cheetah status: two adult male and one 6 years old female "Giza", who gave birth to 7 kittens on 29th Feb 2003 in her 3rd brood. In one of the 10 weeks old cubs there was observed ataxia for the first time. Preliminary diagnosis based on bacteriological, parasitological and neurological methods. All found pathogens were treated adequately. Additionally, antiviral drug was administered for several days. In order to stop the progressive development of ataxia a neuroprotective drug (CDP-choline) was introduced orally. Neurological reflexes were controlled daily until the improvement of the animals' mobility was achieved.

Key words: Acinonyx jubatus, ataxia, neuropathology, Citidinediphosphocholine (CDP-choline), myelopathy.

Introduction
Out of a seven new born cheetahs in the Warsaw Zoo, one of the females developed gait disorder in her sixth week of life. Within few weeks the disease spread over the whole litter. Symptoms observed in this group resembled those, described already under term of "ataxia". It consists in a range of gait disorders from slight sway-back to the degree when the animal can't walk and is unable to stand up at all. Ataxia itself is not a disease – it is only a symptom, however the aetiology remains unknown. Many various factors are suspected such as: metabolic (copper deficiency), infectious (viral or protozoan). The disease concerns certain structures within the Central and Peripheral Nervous System (1,2,3,4,5,).

Materials and Methods
On 27th February 2003 there were seven cheetah cubs (Acinonyx jubatus) born in the Warsaw Zoological Garden in Poland: four females and three males. They were the third litter of the same parents couple (International Studbook numbers: female – 3902, male - 3668). Two previous litters were brought up with no problems whatsoever. The mother, together with the whole litter was kept in grassy outdoor enclosure of approximately 1000 m². They had the direct access to one heated pen. Animals were fed a diet of rabbits and beef with vitamin and mineral supplements. Due to ascariasis they were regularly dewormed (pyrantelum). The first cub showed slight ataxia in sixth week with progressive course. Following weeks ataxia appeared in the rest of the litter. Due to evident neurological background the Citidinodiphosphocholine (CDP-choline) was introduced. After death of four cheetah cubs, which wasn't related to primary disease, their cerebellum, brainstem and spinal cord were taken and fixed in 4% buffered formalin. For light microscopic study the transverse sections from many levels of spinal cord and the coronal sections from cerebellum, pons and medulla were taken embedded in paraffin and stained with standard methods.
Results

Clinical history
In their seventh week of life cubs became listless and body weight gain slowed down. The body temperature remained normal. Just several days later first bilateral watery ocular and then mucopurulent nasal discharge occurred. The signs subsided within 10 days. In their second month first symptoms of disease were observed (ataxia). Due to suspected viral background cubs were treated with acyclovir (5). There was no improvement whatsoever.

As the response to cases described in world resources (particularly results of pathology), the group of scientists from Polish Academy of Science [Polish abbreviation: PAN] has proposed therapy with new drug Citidinediphosphocholine (CDP-choline). The drug has a neuroprotective action and was administered orally. First three of cheetah cubs with the disease symptoms were treated with CDP-choline. Because animals were touched by disease at a different time and the progress of symptoms differed from one to another, at the moment of the drug introduction the two of cubs were falling over and the third was in a state of sway-back. During following weeks the four others developed gait disorders of different degrees. The decision was made to introduce the drug to the rest of the litter. In order to monitor the therapy impact on ataxia, animals were constantly neurologically examined. Examinations consisted of: sense of pain in fore and hind fingertips, examination of radicles sensitivity, knee reflex, abdominal lower reflex and muscle strength of hind limbs as well as evaluation of the ataxia degree. All of that allowed to objectively observing progress in the treatment.

In 22nd week of their life, cheetahs developed another disease which was not related to the primary one (with ataxia). Its course was so acute, that in spite of almost immediate pharmaceutical intervention four animals were lost in deep generalised shock. However, with the great devotion of involved staff, three other were resuscitated. After recover from that unfortunate episode, increase of ataxia was observed. Again CDP-choline was introduced. Gradual and nearly constant gain of the gait ability was clearly seen until complete recover of two of cheetah cubs was achieved. The third one, that suffered the longest from the disease, has recovered up to the degree that allowed ceasing the treatment.

Post mortem
The most distinct changes of cerebellum appeared in cortex and white matter of paramedian and ansiform lobuli but the archicerebellum was less affected. Changes consisted of demyelination of white matter, predominantly in cortical folia, severe Purkinje cells loss and rarefaction of granule cells in cerebellar cortex with axonal swellings and formation of torpedoes in the granular layer or nerve fiber degenerations in the cortex, white matter and cerebellar nuclei associated with hyperplasia and hypertrophy of astroglial cells.

Mudulla and pons showed spongineous changes and demyelination of white matter in spino-cerebellar tracts, inferior cerebellar peduncles and focally in middle cerebellar peduncles. Dorsal funiculi and pyramids were unchanged. Inferior olivary nuclei were rather small and showed chromatolysis with loss of nerve cells.

Spinal cord lesions: myelinated fibers degeneration, vacuolation and mild astrogliosis of lateral and ventral columns, especially in ascending spinocerebellar tracts and in paramedian parts of ventral funiculi (peripheral parts of lateral funiculi). In one case changes occupied whole of lateral and ventral columns. Dorsal funiculi were less affected than lateral and ventral columns. The changes in white matter were pronounced mainly at the thoracic level of spinal cord. Chromatolysis of nerve cells of gray matter was present in ventral horn and thoracic Clarke’s nucleus. Additionally, there was nerve fibers degeneration and vacuolation of myelin in spinal roots. There were also alterations and lack of ganglion cells with concomitant increase of satellite cells in ganglia of the dorsal root. The changes in spinal roots and ganglia were most prominent at the lumbar level of spinal cord.

Discussion
As already mentioned above, ataxia is not a disease – it is a symptom. In fact the aetiology remains unknown. Certain authors suspect copper deficiency to be responsible for the condition (1). It doesn’t seem to be likely because copper is involved in many co-enzymes and supportive substances in an organism. That is why its deficiency leads to multiple pathologies such as: disturbed erythropoiesis, collagen and myelin production and so on. As the result, copper
deficiency should be clinically manifested by multiple syndromes and not only by isolated one - that is ataxia. Moreover, copper, together with other minerals and vitamins supplemented Warsaw cheetahs. Two previous litters (free of ataxia) were fed the same way, with exactly the same supplementation. Other authors claim viral background with Herpes virus involved (5). Following those suggestions the cubs were medicated with acyclovir for 12 days. Nevertheless, the progress of the disease was not inhibited. Also neuropathology reveals no inflammatory lesions – so typical for viral diseases. Because aetiology remained unknown, symptomatic treatment drug (neuroprotective) was introduced. The drug in short is called CDP-choline. Young cheetahs were medicated for 96 days. Neurological condition of all seven cubs was improving. Two ones with so heavy ataxia that they couldn’t walk at all, with time passing under introduced treatment, have reached quite satisfactory condition – they become able to cover a distance of twelve meters. Other five were recovering too but unfortunate additional disease prevented from doing further observations in whole lot. Today, of three living animals the gait disorder in form of ataxia persisted only in one individual and yet it is so slight, that remains unnoticeable for visitors. What’s even more: after caesurae of CDP-choline there is no symptoms escalation.

References

PRESUMED LEVAMISOLE INTOXICATION IN FOUR CHEETAH CUBS
(Acinonyx jubatus)

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Key words: Cheetah, Acinonyx jubatus, levamisole, intoxication, adverse drug effect

Extended abstract
Four 3 mo old cheetah littermates were dewormed with levamisole hydrochloride according to the regular deworming regimen of the Peaugres Zoo. Levamisole was administered subcutaneously at a dosage of 5 mg/kg. Shortly after the injection, all four cubs showed severe respiratory distress and seizures, and died within twenty minutes despite attempts at resuscitation. At necropsy, the only consistent gross finding in all four cubs was a marked pulmonary oedema. Furthermore thymic haemorrhages were found in one animal. Histologic lesions in the lung included congestion with leucocytostasis, diapedesis of neutrophils into the alveolar lumina, prominent alveolar and interstitial oedema with alveolar foamy macrophages, small intraalveolar hemorrhages, and bronchial mucous hypersecretion.

Levamisole, the Levo-Isomer of dl-Tetramisole, is an antiparasitic drug of the imidazothiazole group that is widely used in the treatment of nematodes as well as an immunomodulator in both humans and animals including mammals, avians, reptiles, and amphibians. It is structurally and physiologically similar to nicotine, and therefore, is able to activate cholinergic receptors in ganglia, in the neuromuscular junction, and in the central nervous system (5, 7). Levamisole has a narrow safety margin, and numerous adverse reactions have been reported in many species including vomitus, diarrhea, hypersalivation, muzzle foaming, dyspnea due to bronchospasms and pulmonary oedema, urination and defecation, depression, tremor, ataxia, paresis, excitation, clonic convulsions, immune-mediated haemolytic anemia, thrombocytopenia, bone marrow depression, cardiac arrythmias, erythroderma, erythema multiforme, toxic epidermal necrolysis and unexplained deaths (1, 3, 4, 5, 6, 8, 9, 10, 12, 13). Information on pathologic lesions in levamisole intoxications are few and include congestion of the splenic red pulp, neutrophilic infiltration of the lung parenchyma, marked subepicardial petechia, enteritis, acute hepatic degeneration with subcapsular hemorrhages, massive necrosis of the liver, hemorrhages in the thalamus, and perivascular cuffing with mononuclear cells in the CNS (5, 11). In the present case, pathologic lesions were restricted to the lung and to the thymus.

Based on the acute onset and the nature of clinical symptoms as well as the pathologic findings, the death of the cheetah cubs is presumed to be the result of levamisole intoxication. Symptoms of levamisole intoxication are reported to begin within 5 to 15 minutes after administration and reach a peak at approximately 30 minutes (7). The reason for the intoxication in the present case is not known. In dogs and cats, fatalities have been described with fourfold overdosage of levamisole, and the molecule is considered to be more dangerous when administered parenterally than when given orally (3). In these four cheetahs, the dosage was 5 mg/kg subcutaneously which is within the recommended range in dogs and cats. The injection solution had been prepared by tenfold dilution with sterile physiological solution ten days prior to use in the four cubs and had been stored at room temperature. The same solution had been used previously in other animals.
without any adverse effects. The storage at room temperature should not have any influence on the compound, since the recommended storage temperature for levamisole hydrochloride lies between 15 and 30°C (10). It may be possible that an individual susceptibility is responsible for the fatal reaction. In view of the narrow safety margin and the numerous adverse effects reported, levamisole should be used in non-domestic species only if the potential benefits outweigh the risks of the treatment.

References

RADIUS CURVUS SYNDROME IN AN AFRICAN WILD DOG (Lycaon pictus).

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Abstract
An almost 4-month-old male African wild dog pup showed severe lameness and a swollen distal radius/ulna of the right leg. A clinical examination under anaesthesia showed no abnormal movement of the bones, but 2 weeks later the affected leg showed severe curving of the radius in a craniomedial direction as well as some cranial curving of the ulna. X-rays showed a healing fracture of the distal radius, about 4 cm above the growth plate, as well as an ulnar fracture just above the growth plate. It looked very similar to the radius curvus syndrome in domestic dogs. We performed an ulnectomy, removing a 3 cm long bone piece from the shaft of the ulna. No wedge osteotomy of the radius was done. Some post-operative wound healing problems were encountered, but 3 weeks after the operation the wound was completely healed and the dog improved by the day. 2 months after the operation, the pup was anaesthetised again for a check-up. X-rays showed a completely healed fracture of the radius, as well as a completely healed distal ulnar fracture. At the ulnectomy site the ulna had completely regrown. The radius was still a bit curved and the leg appeared to be shorter. 10 months after the operation the leg appeared to have straightened further and limping was so slight that it had become quite difficult to distinguish him from the other dogs. The proximal ulna was enlarged. The elbow joint showed a normal range of motion with no crepitation. X-rays showed consolidated bone formation at the resection site. An osteophyte on the cranial radial head indicated some arthrosis in the elbow. Only slight cranial and medial bowing of the radius was visible and there was no valgus deviation of the carpus, clinically or radiologically. Comparing the length of the affected leg with the other foreleg, we found that it was 1 cm shorter. Although a wedge osteotomy of the radius would have given a better and more predictable result with regard to the curved radius, the difficulty of aftercare of the pup, who was put back in the group immediately after surgery, made us choose the simplest method. The clinical result was, however, excellent.

Key words: African wild dog, Lycaon pictus, distal ulnar trauma, radius curvus syndrome, ulnectomy

Introduction
Although African wild dogs are supposed to be distantly related to other members of the Canidae family, their anatomy appears to be comparable to that of other Canidae and even to that of the domestic dog. By performing an operative method of correcting a curved radius in the way that it is done in domestic dogs, we managed to get a severely lame wild dog back on his feet and functioning almost normally again.

Material and methods
On 18 March 2003, a keeper reported that an African wild dog pup born on 24 November 2002 in a litter of 12 was having obvious trouble with his right foreleg and limping badly. The pup was anaesthetised with a combination of 50 mcg medetomidine/kg (Domitor®, 1 mg medetomidine HCl/ml, Pfizer Animal Health, Capelle a/d IJssel, the Netherlands) and 3 mg
ketamine HCl /kg (Ketamin®, 100 mg ketamine HCl/ml, Alfasan, Woerden, the Netherlands). Clinical examination revealed a swollen distal part of the lower leg just proximal of the carpus. The skin in this area showed some small wounds. No abnormal movement of the radius, ulna or carpal bones could be detected. Body temperature was 39.5. The diagnosis was: trauma caused by biting, which had led to inflammation and infection.

Amoxycillin was given by injection, in a dose of 20 mg/kg (Albipen LA®, 100 mg amoxycillin anhydrate/ml, Intervet, Boxmeer, the Netherlands) together with 2 mg ketoprofen/kg (Ketofen®, 10 mg ketoprofen/ml, Merial etc.) and a course of treatment with amoxycillin and clavulanic acid was prescribed in a 12.5 mg/kg bid dose orally (Synulox®, 200 mg amoxycillin and 52.5 clavulanic acid per tablet, Pfizer Animal Health, Capelle a/d IJssel, the Netherlands).

The pup improved slightly during the next few weeks, and on 2 April the animal was anaesthetised again as part of a general check-up when the pups were being blood sampled, vaccinated, dewormed and treated for dermatophytosis. On this occasion the right foreleg showed a severe bowing of the radius in a craniomedial direction. The ulna was slightly curved. A hard swelling could be palpated in the radius above the carpus. We took X-rays, and these showed a healing fracture of the radius 8 cm above the carpus that had not affected the growth plate, and an ulnar fracture just above the growth plate. The radius was markedly curved in a cranial as well as a medial direction, causing a valgus position of the foot. We decided to operate on the dog, using the ulnectomy method, as we are familiar with this procedure in domestic dogs. Although the curving of the radius meant that it would have been preferable to do a wedge osteotomy of the radius together with the ulnectomy, applying a external fixator was not an option, as the pup would not tolerate the device or bandage, and aftercare would be difficult because he would be put back in the group immediately after surgery. To put the dog under deeper anaesthesia, he was intubated, and isoflurane and oxygen were given. An IV drip was put in, and circulation and breathing were monitored by a pulse oximeter and a capnographer.

**Operation**

We prepared the caudal part of the lower leg in a routine fashion. The ulna was exposed by a caudal approach. The M. ulnaris lateralis and the M. flexor carpi ulnaris were severed from the ulna and a 3 cm segment of the proximal ulna was removed, using a Gigli wire. We made sure that the segment was exposed extraperiostally to prevent early bridging of the ulna. The ulnar gap was filled up with purified gelatine foam (Spongostan, Ferrosan A/S, Soeborg, Denmark) to prevent bleeding at the ulnectomy site. No fat graft was harvested from another part of the body to minimise any complications resulting from another wound. We also made the resected part of the ulna slightly larger than the advised 1.5x the ulnar diameter. We closed the subcutis and the skin using polyglactin 2/0 atraumatic (Vicryl FS 1 2/0 atraumatic®, Ethicon, Amersfoort, the Netherlands). A high dose of enrofloxacin 8 mg/kg (Baytril® 2.5%, Bayer, Mijdrecht, the Netherlands) was given, together with a long-acting amoxycillin in a dosage of 20 mg/kg (AlbipenLA®, 100 mg amoxycillin anhydrate/ml, Intervet, Boxmeer, the Netherlands). Before taking the dog back to recover, 25 mcg/kg atipamezole was given (Antisedan®, 1 mg/ml, Pfizer Animal Health, Capelle a/d IJssel, the Netherlands).

**Post-operative period**

On 9 April, after a good recovery and initial improvement, the dog began to limp badly again. The leg was swollen from the elbow down and the operation wound had opened. The animal was active, eating normally and seemed otherwise healthy. The next day we anaesthetised the pup again with 0.6 medetomidine HCl and 0.4 ketamine HCl (50 mcg medetomidine HCl/kg and 3 mg ketamine HCl/kg) to examine the operation wound, which was granulating and looked clean. The leg was still swollen but did not show any abnormal movement at the resection site. The body temperature was 39.3 C. Our differential diagnosis was: inflammation and/or infection of the operation wound, post-operative swelling due to inflammation or circulation problems, and new trauma to the leg caused by other dogs in the group.
group. We decided to give the dog a series of antibiotic injections, using 6 mg/kg danofloxacin (Advocin®, 180 mg danofloxacin/ml, Pfizer, Capelle a/d Ijssel, the Netherlands) and 20 mg/kg amoxycillin anhydrate.

On 16 April, after having had 0.5 cc Advocin® and 2.5 cc Albipen LA® 3 times, given every other day, the leg showed a freshly granulating and nearly closed wound and hardly any swelling of the lower leg. The pup started to regain normal functioning, though still limping

On 6 June, 2 months after the operation, we anaesthetised the animal for another check-up, as well as to take blood samples and to vaccinate and deworm him. By that time the operation wound had closed. His lower leg still showed some bowing of the radius and seemed a bit shorter. We took X-rays, which showed a healed fracture of the radius. The radius was still a bit curved, though less than before the ulnectomy. The ulna had regrown completely at the resection site. The distal ulnar fracture had also healed.

Results
Between June 2003 and January 2004 the dog did very well from a clinical point of view. He was only occasionally seen to limp slightly, and when the time came for his final check-up, it had become quite difficult to distinguish him from the other dogs by the way he walked. On 23 January 2004, at the age of 14 months, the dog was anaesthetised for a final clinical check-up and to take new X-rays. The leg was measured and compared to the other leg to assess possible shortening. Clinical examination revealed a slightly cranially bowed radius and a thickened proximal ulna at the former resection site. The elbow joint had a normal range of motion and could be fully flexed and extended without crepitation. X-rays showed abundant but consolidated bone formation at the proximal resection site of the ulna. There was an osteophyte on the cranial radial head, indicating some arthrosis although no other radiological signs of arthrosis were seen. All growth plates were closed. The radius showed some cranial and medial bowing, though far less than at the time of the operation. This could explain the slight shortening of the leg. We compared the length of both legs, measuring the distance between the olecranon and the os carpi accessorius. The affected leg appeared to be 1 cm shorter. This could explain the slight limping in the dog’s trotting gait.

Discussion
Growth disturbances of the radius and ulna are common in domestic dogs. Longitudinal growth of the radius and ulna occurs from the proximal and distal growth plates. The distal growth plate contributes 70% to the ultimate length of the radius; the distal growth plate of the ulna produces 85% of its growth. The remaining percentages are derived from the proximal growth plates, however in the ulna this plate contributes more to the length of the olecranon than to the shaft of the ulna. In domestic dogs, premature closure of the distal ulnar physis and subsequent deformities of the lower foreleg are the most common complications of forelimb physeal injuries. The distal ulnar growth plate is conical in shape, instead of the flat shape seen in all other animals. The African wild dog shows the same unique conical distal ulnar physis. The canine distal ulnar physis cannot shear due to this conical shape, so tear forces are transformed into compressive forces and injury to the germinal cartilage. Significant retardation in the growth of the distal ulnar physis results in a shortened ulna, which then - because of interosseous ligamentous connections between the bones - acts as a bowstring, restricting longitudinal growth of the radius. Consequently there is first anterior and then medial bowing of the radius, causing lateral deviation of the foot (carpal valgus deviation). Later, external rotation of the carpus and subluxation of the elbow occur. In large dog breeds, growth plates close between 7 and 9 months.

If we compare the African wild dog to a large breed of domestic dog at the age of 4 months, the distal radial physis should have a growth potential of about 4 months. In the case of the wild dog we were lucky to catch the problem at a fairly early stage. Though there was a markedly crooked radius already, no visible damage had been done to the elbow and there was still growth potential left for the radius. Ulnectomy at the age of 4-5 months in a case of distal ulnar trauma is in our opinion a good method to treat the radius curvus syndrome in African wild dogs. It seems like the African wild dog is more closely related to the domestic
dog than you would think, considering the fact that it lacks the first digit in both forelegs and hind legs.

**Acknowledgements**
We would like to thank the African wild dog keepers at Artis Zoo-Amsterdam for their support, as well as Ron van der A of the Painted Dog Research Project and Artis photographer Ronald van Weeren for providing some photographs for the presentation. And last but not least my wife Mandy Savage for correcting my English.

**References**
MEDICAL TREATMENT OF A PARTIEL VAGINAL PROLAPSE IN AN AFRICAN ELEPHANT (*Loxodonta africana*)

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**Abstract**
This report is about a successful medical treatment of a vaginal prolapse (the size of two footballs) in an *African elephant* (*Loxodonta africana*).

**Key words:** Partial vaginal prolapse, African elephant, *Loxodonta africana*

**Introduction**
The syndrome of a vaginal prolapse is also seen in *elephants*. Schröder (2003) reports about the successful surgery of two animals in the past years.

**Material and Methods**
The case at hand is about a female *African elephant* at the age of app. 25 years called Samba, which lives in a circus together with one female African and five female Indian elephants. The food consisted of hay at their own disposal, carrots, apples, stale bread, logs and various green feed. The animal was presented because it suffered from a massive oedema in the caudal abdominal region and a vaginal prolapse in the size of two footballs. During the first checkup parts of the described prolapse were already necrotic and the animal had less appetite. It was also significant that the *elephant* needed to urinate every few minutes. Samba also refused to lie down when she was ordered to. Although she was trained she wasn’t very cooperative during the whole process and as the owner didn’t want an expensive examination (including blood tests) an antibiotic treatment with 50 ml Veracin® im. and a diuresis with 2 grams Furosemid orally was practised for 2 days. In addition the prolapsus was to be creamed with milking fat daily. Two days later Samba was already a bit better, the prolapse had become smaller and the appetite was better. As the therapy worked it was continued. Her condition further improved within the next 48 hours, only the oedema in the caudal abdominal region had hardly changed. When discussing the case with colleague Hinke on the phone she also saw the possibility of a heart disease and the corresponding therapy. The owner however didn’t agree and so the therapy with Veracin® injections, Furosemid and the milking fat was continued. All in all the *elephant* got Veracin® for 10 days and Furosemid daily for the first 4 days and then every other day. On the 10th day after the beginning of the treatment the prolapse only had the size of half a football and the oedema in the caudal abdominal region also diminished. Samba had a normal appetite and urinated normally with the intervals becoming longer and longer. She also lay down again. When the circus moved to a rather distant location for a week the prolapse was to be creamed with a sulphonamide-fish liver oil ointment while they were away. Samba also got 2 grams of Furosemid every three days. During the next examination 10 days later the prolapse had totally regressed only when urinating a little bit of mucosa was to be seen. The oedema in the caudal abdominal region was still there but had diminished. Even now the owner could not be convinced of a cardiologic diagnosis and possible therapy. As the circus finally moved on the course of the disease could not be watched.
History
As reported later the oedema in the caudal abdominal region grew which caused the animal to lie down for longer intervals. When lying the pressure of her own body led to a partial diminution of the oedema. Every now and then there was a lameness, the reason of which is unknown. This lasted for several weeks.
Six months after the outbreak of the illness the animal was unable to rise and died without further therapy.

Necropsy
The necropsy was carried out at the Institute for Pathology of the “Tierärztliche Hochschule Hannover” with the result that a pneumonia had led to the death. Changes in the female genital and cardiovascular system could not be found.

Discussion
A prolapse of the vestibule of vagina in older female Asian elephants has occurred and has been discussed several times before (Rüedi 1995). In all cases the treatment is a surgery. The same author also reports about two vaginal prolapses in free-living African elephants without giving information about the cause.
In the case of Samba a cardiovascular disease was also taken into consideration but the owner did neither agree to a corresponding diagnosis nor to a diagnostic therapy. The necropsy proved him to be right. A nutritive cause could be excluded as only this one animal fell ill. Also pregnancy could not be the cause of the oedema as the elephant had lived in a female-only group. Whether the course of the cycle favoured the prolapse – all females in the heat urinate permanently but marginally – could not be determined. It is possible though that a small prolapse occurred because of the permanent urination during the heat which became partially necrotic and then grew. Maybe this also led to a cystitis which would explain the permanent urination that lasted for nearly one month. But the owner didn’t agree to a microbiological examination of the urine either. However the broad spectrum antibiotic also works in the bladder. Because of the prolapse local circulatory disorders might have happened which caused the caudal abdominal oedema. The therapy with the broad spectrum antibiotic, the diuretic and the creaming of the prolapse with a softening ointment and later an antibiotic one led to a total disappearance of the vaginal prolapse or at least a partial diminution of the oedema.

Acknowledgment
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PARTIAL PULPECTOMY IN LEFT TUSK OF AFRICAN ELEPHANT

(Loxodonta africana)

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Abstract
Four years old male elephant “Lotek”, who arrived to Warsaw Zoo in summer 2003. Animal showed certain degree of excitement since it's loading for transportation that was mainly expressed in striking its tusks against obstructions such as walls. This lead to tusks damage, mainly the left one. After photographs and x-ray analyses as well as the direct examinations it was decided to conduct a partial pulpectomy in the left tusk and to fill a mild cavity in its right tusk.

Key words: Loxodonta africana, African elephant, tusks cavity, pulpectomy.

Introduction
The elephant showed signs of excitement, which initially were interpreted to be caused by tiring transport and change of environment. After several weeks however, it become obvious that it must be a different reason for elephant’s behaviour. Close examinations on 31st August 2003 showed open cavities, particularly of its left tusk, positioned centrally on a tip. The cavity was obscured by mud of ivory resembling colour. The left tusk cavity was about 15 to 20mm in diameter well reaching a pulp as the tusk was already rubbed off. It was clear from the x-rays there was an inflammatory process, but one could not be certain if and what bacteria were involved without microbiological examination - it was decided not to conduct any further. At the same time right tusk cavity was some 8 to 10mm in diameter and not exceeding 5mm of depth. It seemed that tusks showed low degree of calcification, thus favouring deepening of the pathological process. Damages of tusks mainly considered tips but were also localised laterally and on upper surface. Prior to decision of treatment the additional study on subject was done (1,2,3,4,5,6,7).

Materials and Methods
On 15th September 2003 4 years old African elephant “Lotek” from Warsaw Zoo was anaesthetised for dental treatment. It was supposed that total time for anaesthesia must not exceed 90 minutes. Main target was to conduct a partial pulpectomy of the left tusk, secondary – to fill the right tusk cavity and third – feet correction. Here, we concentrate on the primary one. It consisted of several stages: 1) on x-ray tusk calibration (due to establish the range of cavity depth), 2) rough cleaning of cavity, 3) detail cleaning of cavity, 4) pulpectomy, 5) working out cavity internal wall and 6) filling in the cavity. The calibration consisted of several consecutive firm tin wire loops, wrapped around task. Also one loop was introduced into the cavity until the resistance could be felt. The rough cleaning was both mechanical and with detergent water solution, then treated with Iodine-alcohol solution. To rinse off the cavity the physiological solution of Sodium Chloride (NaCl) was used during whole treatment. After drying out, the calibration x-ray was used to compare and determine depth of the cavity. At the bottom of cavity there appeared bloody-serous exudation and from that point the inflamed pulp was extracted until clear blood was seen. The walls were worked out roughly with the use of carbon drills cooled with sterile NaCl.
solution and precise cut on ivory was done with diamond drills, at the point of narrowing, some 4.5mm deeper than primary bottom of the cavity. First thin layer was the Calcium Hydroxide (Ca(OH)$_2$). It was repeated until there was no obvious underleaking seen. On top of that the 2mm layer of phosphorous cement Agatos was put. It was supposed to be partially neutralised by a part of Ca(OH)$_2$ undercoat. This particular material was chosen due to its known mechanical property. Final filling was planned with new generation material together with V generation bonding one. The 36.5% solution of Orthophosphoric Acid was utilised to etch, during 30 seconds evaluating the low degree of calcification. The ENABOND was spread equally over walls under flow of the air. Material was exposed to light for 90 seconds. Because the size of cavity was pretty extensive, each surface was exposed separately. To fill the cavity the Enamel plus HFO was used and was put in multiple layers to avoid a polymerisation cramp. It also guarantees tightness of filling. Some dozen of layers was put down and on the top of that the external coat of enamel material was done. Time limitation and position of the elephant on its right side forced to treat the right tusk more quickly. It was only cleaned and filled up similarly to the left one.

Results
The animal was closely observed for next few weeks. First effect was seen that it calmed down clearly and seized to strike its tusks for some next three weeks. The fillings were firmly in and even after two months or so, when elephant started scratching its tusk again, the left one remained unchanged. Even though left ivory was rubbed off constantly but not very deep. It was noticed that filling is harder than the ivory thus it is not rubbed out equally. Different situation was observed with the right tusk. That one became main objective of animal’s striking and scratching and by December 2003 it was cracked and separated quite visibly. Obviously this tusk started to disturb animal. At the same time it was noticed that the filling of right tusk cavity was lost. X-rays was taken twice: four and five months after treatment. First showed the spots of intensive calcification in the left tusk and no symptoms of inflammation or other disturbing symptoms. The right tusk showed that the cavity is widening and getting deeper and is joining the pulp already. However, there are spots of calcification seen as well. Second x-ray session revealed significant progress of calcification in the left tusk and further withdrawal of the pulp. Right tusk also showed progress in calcification particularly towards opening of the cavity.

Discussion
Taking the above under consideration, two possible solutions were discussed. First was to treat the right tusk as with the left one was done. Second was to leave it for self-treatment. The tusks may be compared to young tooth with unfinished tip development. The pulp of such teeth shows great vital potential and tendency to calcification while inflammatory process appears. The environment of tusks is dry, exposed to UV and well ventilated. That is why the gangrene type of bacterial complication is unlikely. Much more possible seems to be tetanus infection. The decision was to leave the animal under close observation and not to proceed with dental treatment at this stage. In order to avoid further damages of tusks, the walls of inside pens are to be covered with hardwood stamps. It is under construction now.
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STOMACH RUPTURE AND TORSIO COLI IN AN OLD MALE WHITE RHINOCEROS (Ceratotherium simum simum)

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Abstract
An old white rhinoceros (Ceratotherium simum simum) suffered from exfoliative dermatitis during his live. The last months he lost weight despite of good appetite. At the end of July he showed signs of pain and was treated with analgesics. He died from the consequences of peritonitis due to a ruptured stomach and a torsio coli. The animal showed extensive lesions of the teeth, correlated with old age.

Key words: Rhinoceros, Ceratotherium simum simum, stomach rupture, peritonitis, dental disease.

Clinical history
A captive born, male, white rhinoceros (Ceratotherium simum simum) imported from South Africa (Pretoria National Zoo Gardens), and exhibited in the Antwerp Zoo since 1974, developed skin lesions in 1993. Malassezia pachydermatitis and Candida parapsilosis turned out to be responsible for an exfoliative dermatitis, which was successfully treated with natamycin. Another female white rhino living together with this animal never showed any symptoms of this exfoliative dermatitis (1).

The male animal with the name Balthazar moved to Safari Beekse Bergen, Hilvarenbeek, The Netherlands, in November 1995. There he lived together with ten other white rhinoceroses. Only he developed periodically signs of this specific exfoliative dermatitis, which every time was successfully treated using topical cod oil with enilconazole. It was at that time postulated that this animal could be immune-depressed in some sort of way. In the years following he fathered three young. One male offspring also developed skin lesions and was treated in several ways, including different antibiotics. This young animal finally died 6 months old due to an endocarditis, probably caused by haematogenic transport of bacteria out of the extensive skin lesions. The other two offspring’s, both female, never showed any skin problems.

Besides the skin problems Balthazar was a healthy normal white rhinoceros. Tetanus vaccinations and occasional slight hoof problems with treatment are the only remarks in his medical report until the summer of 2001. Fecal examinations were always negative and reproduction behavior occurred. In August 2001 he developed a unilateral nasal discharge. The discharge was clear but sometimes contained some blood. Early September the animal was treated orally for five days with trimetoprim-sulfamethoxazole. Bacteriological examination of the discharge revealed beta-hemolytic streptococci, which were not susceptible for this antibiotic.

Blood examination showed no irregularities. The animal was treated with 4 grams of cefitiofur once a day i.m. by dartgun for 24 days. On day 14 the animal showed no longer signs of
In October and November the same year 2001 the nasal discharge occurred again and the animal was treated with 4 grams ceftiofur respectively for 5 and 14 days. Until July 2002 the animal was fine. At that time there was only clear discharge reported for a few days. In September however the discharge occurred again and contained blood. Beta-haemolytic streptococci, sensitive for ceftiofur were cultured. The animal was again treated for nine days with ceftiofur and recovered.

June 2003 the animal was reported to loose weight. Faecal- and blood examination revealed no abnormalities. In the next two months his weight loss progressed. Despite good appetite and apparently enough food-intake the animal was deteriorating and at the end of July it showed pain. No nasal discharge was seen at the time. The animal was treated with 6 grams of ceftiofur and 50 cc of finadyne for the two consecutive days before he died.

**Post mortem**
At necropsy, based on fat stores and muscle reserves, the animal was in a moderate body condition. Multiple superficial lesions of the skin were present. Locally on the right leg a wrath-like lesion was seen. On the right thigh, at the site of the trochanter tertius a vesicular lesion with connective tissue and proliferation of bursal epithelium was present. Inspection of the oral cavity revealed irregularly eroded teeth covered with large amounts of plaque, chronic gingivitis, periodontitis, numerous loose teeth, and exfoliation of a molar. Food impaction between the teeth was evident. Petechial hemorrhage was present in the heart muscle, chronic filamentous pericarditis on the right auricle, and irregular atrio-ventricular valves. The lungs showed chronic filamentous, adhesive pleuritis, and focally alveolar emphysema. The bronchial lymphnodes were enlarged. The abdominal cavity was filled with approximately 100 liter of turbid yellow and red fluid with food elements. The omentum was hyperemic and covered with fibrin depositions. In the esophageal zone of the stomach a 7-cm long rupture was evident, furthermore multiple, large and irregular ulcers were present. The stomach contained a lot of food and sand. The small intestines were overfilled with watery contents. The contents of the ascending colon were poorly digested, very dry, and contained much sand. The left dorsal and ventral colon was rotated 180° along the long axis. The liver and the kidneys were enlarged and pale. Tissue samples from the heart, lung, liver, stomach, intestine, pancreas, adrenal glands, and skin lesions, were fixed in 4% phosphate-buffered formalin, embedded in paraffin, cut at 4 µm sections and stained with hematoxylin and eosin (H&E), and Perls’ Prussian blue reaction for ferric Iron. Aerobic bacterial culture of the liver and colon contents was performed using standard techniques. Histologically, the myocardium was hemorrhagic. The lung showed alveolar emphysema. The liver was hyperemic, and kupffer cells and to a lesser extent hepatocytes were filled with iron pigments. There was focal ballooning and necrosis of the hepatocytes. The esophageal zone of the stomach showed ulceration of the epithelium, with locally many bacteria, and little inflammatory reaction. Elsewhere, more prominent mixed granulocytic and lymfo-plasmacytic inflammatory reactions, with necrosis, and angiogenesis with fibrosis in the submucosa were present. The intestinal serosa was covered with a thick fibrinous exsudate with bacteria and plant material. The muscle layer showed superficial necrosis. The pancreas had multiple hyperplasias of exocrine pancreas tissue. The adrenals showed multiple hyperplasias of medullar cells. The kidney showed interstitial fibrosis with calcium depositions. The superficial lesions of the skin showed aggregations of bacteria in the superficial keratin layer. In some areas the superficial keratin layer had disappeared. There was no inflammatory reaction.
The process on the trochanter tertius was a proliferation of connective tissue with numerous vessels. No bacteria were cultured from the liver. A mixed population of bacteria was cultured from the colon.

**Discussion**

The animal died of the consequences of the rupture of the stomach and subsequent peritonitis due to food substances in the abdominal cavity. Equidae, rhinoceroses and proboscides in nature often die at old age because of teeth problems. The fact that they can’t eat or can’t digest properly anymore induces emaciation and finally death. This animal had severe changes in the mouth. Varying from gingivitis, loose teeth, and alveolitis and heavy plaque formation. We think that the animal tried to eat enormous amounts of food, in order to cope with his energy needs, without being able to properly chew the food.

Sand and impaction of the colon with maldigested food provoked torsion of the left dorsal and ventral colon. Stomach impaction with non-chewed material and sand, subsequently evoked a rupture of the stomach. Resulting in a massive peritonitis. Colic signs weren’t that obvious, but the animal must have died with bitter pain.

In the clinical case described above we think that the animal finally died as a consequence of the described lesions of the teeth correlated with old age. Due to altered food intake, it finally resulted in the stomach rupture and the torsio coli.

**References**

TWO BIRTHS OF ASIAN ELEPHANTS (*Elephas maximus*) IN A NATURAL GROUP – OBSERVATIONS AND EXPERIENCES

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**Abstract**
In the year 2003 the first two births of Asian Elephants (*Elephas maximus*) in a natural group in Germany occurred in Hagenbecks Tierpark. The births took place in a new birth-stable which was designed based on the experiences during earlier births in Hamburg and in other European zoos.

At 14th May 0,1 calf was born by “Yashoda” a 24 years old cow. It was her third pregnancy. At the 22nd June 1,0 calf was stillborn by the primiparous cow “Lai Sinh” (13 years old). This birth process needed veterinary assistance which is described. The group showed very positive behaviour especially in the first case.

Advantages and possible problems of births in a group are discussed.

**Key words:** Asian Elephants, *Elephas maximus*, birth, natural group, birth assistance

**Introduction**
During the years 1992 - 2001 the elephant-breeding-program in Hagenbecks Tierpark, Hamburg, resulted in 8 births. In all these cases the female was separated from the group and chained. Motivated by this success and by the experiences in some other zoos (5,12) we decided to build a special birth stable in our elephant house. For this purpose we combined two boxes and a part of the keepers area. The stable measures about 120 sqm.

Several authors (1, 2, 3, 5, 11) reporting on elephant births describe an aggression of the elephant-cow against her baby immediately after birth. In these cases the cows kicked their babies. The calves glided several meters on the ground and some of them hit obstacles and got injured.

Our conclusion from these reports and from our own experiences was to build a stable without obstacles and other details that endanger the newborns health. There are no vertical pillars. The wall-to-ground connection is angular (about 45°) to keep the calf from bouncing directly onto the wall. For the safety of the keepers (the elephants are kept under hands-on conditions) we built emergency exits in three corners of the stable.

Before the 2003 births the elephant group in Hamburg consisted of 1.11 animals. Most individuals of this herd were not related to each other. The adult ones originate from the wild or from working camps. Nearly none of them arrived to Hamburg from the same location or in the same year. Nevertheless some of them developed a close friendship.

Because our new birth stable is not large enough for all our elephant cows we had to select some of them that stay together during birth. Based on the daily observations of our keepers we decided to keep a group of four cows together during birth. These four cows had spent a lot of time together each day and had a very strong friendship. Two of them are related. The group consisted of “Yashoda” (24 years, 3rd pregnancy), “Lai Sinh” (13 years, 1st pregnancy), “Shandra” (37 years, high rancing cow) and “Corny” (7 years, daughter of “Yashoda”). “Yashoda” was supposed to give birth earlier and therefore we hoped she could teach “Lai Sinh” how to handle a birth. About three months before the first possible birth date the group was introduced in the new stable.
All preparations had been finished right in time. The hole staff was very excited to see how the elephants would deal with the new conditions.

**Material and Methods**

1. **Yashoda:**
Visible labour started at 1.00 a.m. on 14th May 2003. The typical symptoms like restlessness, changing of positions, pressing with one hind leg against the wall or kneeling down occurred. The three other cows paid a lot of attention to “Yashoda”. Especially “Shandra” and “Corny” stood close to her or touched her. They smelt at her anogenital region. There was a steady and draughty birth process.

At 2.50 a.m. the swelling of the perineum was visible for the first time. And at 5.53 a.m. the calf was born in anterior position between the legs of „Yashoda“. The cow turned aside immediately, bowed her head down to the baby and growled. With one front leg she stepped carefully on the calf to destroy the amniotic membranes. Afterwards “Yashoda” kicked the baby to encourage it to stand up. All elephants started a loud trumpet immediately after birth. “Shandra” protected the newborn against the two young and curious elephant cows. Several times “Shandra” and “Yashoda” circled around the calf. Only ten minutes after birth the baby rose up for the first time.

2. **Lai Sinh:**
During the following weeks “Lai Sinh” spent a lot of time with the newborn “Kandy”. She showed a very positive behaviour and made us hope that she would react alike with her own baby expected only some weeks later.

Around noon at 21st June 2003 visible labour started. In the beginning it looked like a normal processing birth. At 2.00 p.m. the perineal swelling was visible. During the next hours „Lai Sinh” showed powerful labour. “Shandra” and “Yashoda” initiated a lot of close contacts to her. One of them was close her all of the time. We assume that they tried to calm her down. This interest stopped at about 11.00 p.m. almost completely. Until 1.45 a.m. at the 22nd June “Lai Sinh” showed powerful and regular labour. The swelling of the perineal area grew slowly. During the following 3½ hours the visible labour slowed down. “Lai Sinh” rested and fed hay.

At 5.25 a.m. labour started again but was very soft and powerless. At 5.58 a.m. 200 mg Denaverine-HCL (Sensiblex®) were injected i.m. to support the primiparous cow during the birth process. Because there was no visible process until 6.40 a.m. the elephant was chained and a rectal examination was done. The cow showed a very nervous behaviour during this process. Only a very short examination was possible, but two legs of the same length were palpable. The ends of the legs layed more or less in one line with the hind end of the bony pelvic ring.

After these results 490 µg Carbetocin (Depotocin®) were injected via blowpipe. 15 minutes after injection the labour strengthened but could not produce the expected progress during the following 2½ hours. Even one further injection of a labour supporting drug (100 I.U. Oxytocin i.m.) 20 minutes after the application of 400 mg Denaverine (Sensiblex®) i.m. did only result in a short and very powerless increase of labour. “Lai Sinh” showed growing exhaustion. She layed down on her side several times, particularly for more than five minutes. During the following three hours there was nearly no labour visible.

Neither blood sampling for checking Calcium-levels nor placing an intravenous catheter was possible because of the nervous behaviour of the elephant cow. Nevertheless a Calcium support seemed to be a possible treatment to restart the interrupted labour. Because of the nervous behaviour a subcutaneous drop (Calcium-electrolyte-solution) was started. The drop was fixed at the top of a long pole and one of the keepers moved around keeping the pole with all movements of “Lai Sinh”. The drop lasted about 25 minutes. About 15 minutes later 735 µg Carbetocin (Depotocin®) were injected. Only a few minutes later powerful pressing-labour started again and after about 30 minutes the amniotic sac appeared. But there were no contours of the legs visible in the perineal swelling at this time. The external bladder formed by the amniotic sac raised its volume during each pressing.

Soon the tops of the legs were visible through the skin of the perineum. Some minutes later a moderate loss of amniotic fluid through a small cut in the membranes was observed. Under
permanent strong labour the male calf was born in anterior position after 26.5 hours of birth duration at 2.37 p.m. No signs of life were visible. “Lai Sinh” kicked the baby with a front leg and tried to remove the fetal membranes.

Neither during the last hours of birth process nor immediately after delivery “Shandra” and “Yashoda” showed any interest to the occurrence. Only after “Lai Sinh” started excited trumping and growling the other cows went to her and tried successfully to calm her down. The bodyweight of the dead male calf was 108 kg.

**Discussion and Conclusions**

The described births in a natural group had been the first in Germany that occurred in a natural group. In conclusion the collected experiences had been very positive. From the zoo-veterinarians point of view the birth handling in an elephant cow staying in a natural group is a little more difficult than under chained conditions. Supposition for necessary examinations (rectal examination, blood sampling) and treatments (e.g. intravenous infusion) is a high standard in keeper to elephant relation. Only an elephant in hands on conditions can be handled during such an exciting process. The possibility of chaining for short interventions must be available in the birth stable. The combination of well trained elephant cows giving birth in natural groups seems to be a successful way of optimal birth management. The possibility of veterinary access during birth is reported as a very important tool for successful elephant breeding (8). The methods used for birth assistance in the case of “Lai Sinh” are similar to that described in other reports (4, 6, 9). With the use of labour producing drugs the fetus could be brought out the natural way. But nevertheless all preparations for performing an episiotomia have to be done early in advance (7, 8, 10).

The aggression of the mother against her calf immediately after delivery has been described in several cases (1, 2, 3). The cows behaviour towards the baby after the birth has to be inspected and compared with births under chained conditions:

1. The baby gets born between the four legs of its mother. The cow is not able to have a look at it. In birth-situation without chains she will step back carefully and can have look at the newborn. Under chained conditions she even wants to see – and protect – her baby. The cow tries to kick the baby forward to reach this goal.

2. Next behaviour is to remove the fetal membranes by stepping on them. This looks very natural and not aggressive in free moving cows. In chained situation the observer notices a second stepping at the calf which often looks extremely dangerous because of involved chains.

3. After that the cow bows down her large head producing loud contact-sounds (growling and trumpeting). This can be easily misinterpreted as an attack with the head.

4. Now the elephant cow kicks the baby to encourage it to stand up. In the free running group this behaviour can very easy be identified as usual mother child interaction. In many cases with chained cows the keepers have “saved” the calf after all “aggression” described above before this point is reached.

Summarised most of the “aggressive” behaviour shown by chained elephant cows is equivalent to the behaviour shown by other mammals after birth. This is obvious after observing elephant births without chains in a group. These results resemble those from other authors (5, 12).

Comparing the births of “Yashoda” and “Lai Sinh” results in an amazing difference regarding the interest of the other cows in the birth process. During the whole time of “Yashodas” birth the other elephants kept close contact to her. Immediately after birth they were very excited and showed highly interest in the calf. In contrast to this the interest in “Lai Sinhs” birth was similar during the first 11 hours but than stopped suddenly and went down to nearly no interest for the rest of the process. There was even no interest in the newborn immediately after delivery. There are no reports describing the behaviour of a group in case of a stillborn calf. Maybe the other cows knew that the baby was dead (infrasound?). It needs more birth observations of births with living and with stillborn calves to find comparable cases.

The main problem in breeding elephants in zoos is that most of the cows have no birth experiences and have no chance to learn it before they have to give birth to their first own calf. Births in the group as happened in Hamburg last year can help filling this lack of
behavioural training. Further primiparous cows can be supported by experienced aunts during birth process and during the first time after birth.

References

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