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DIAGNOSTIC AND TREATMENT OF A PNEUMONIA IN A MADAGASCAR BOA
(Acrantophis dumerili) CAUSED BY A COAGULASE-POSITIVE
STAPHYLOCOCCUS

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Abstract
Respiratory disease is a major cause of morbidity and mortality in Reptiles. A lung biopsy by a
percutaneous pneumoscopy has been performed in a Madagascar Boa (Acrantophis dumerili)
presented with pneumonia. A coagulase-positive Staphylococcus has been cultured. A
treatment for 15 days with amoxicillin / calvulanate (20 mg/kg, IM q24h) and enrofloxacin (10
mg/kg, IM q48h) has been conducted successfully. This paper demonstrates the importance of
endoscopic evaluation of the respiratory tract when investigating respiratory disease. It also
describes an unusual pathogen, Staphylococcus sp. as the cause of pneumonia in a Reptile.

Key words: Reptiles, Acrantophis dumerili, Pneumonia, Pneumoscopy, Staphylococcus

Introduction
Respiratory disease is a major cause of morbidity and mortality in Reptiles due in part to the
lack of a muscular diaphragm and reduced ability to cough and remove debris from the
respiratory tract (1). Although a wide variety of bacteria have been incriminated as either
primary or secondary pathogens, infections caused by Gram-negative bacteria are most
common (2). Most of the time, culture from the pathogenic organ or tissues may give the
diagnostic. Endoscopy has proven to be a useful diagnostic tool for such investigation.

Material and Methods
A captive born Madagascar boa (Acrantophis dumerili), aged 6 years, weighting 1.58 kg, had
been submitted for clinical examination. Physical and radiographic examinations were
performed. Examination of the respiratory tract was achieved by a percutaneous pneumoscopy
under anesthesia. A 2.7 mm Hopkins telescope, a 14.5Fr examination sheath, a flexible double
oval jaws spoon biopsy forceps 5Fr, a xenon light source, air insufflation, a video camera and a
monitor composed the endoscopic equipment.
The snake was anaesthetized with propofol (8 mg/kg, IV) and maintained in straight left lateral
recumbency. The heart was visualized by its beatings. It was located at 27.39% of the snout-
cloaca length. The pulmonary air approach was carried out on the right, halfway between the
heart and the presumed cranial border of the kidney (52-58 % of the snout-cloaca length in
Boïds (3)). The skin was prepared for aseptic surgery. An incision of two centimetres was
practised between the second and third row scales. The coelomic membrane was reached by
dissection of the muscles and punctured, letting appear the liver. The ribs were inclined dorsally
and the liver ventrally using spacers. The serosal surface of the non-pulmonary lung was then
visualized and penetrated. An incision of approximately one centimetre is practised allowing the
passage of the endoscope. The endoscope was directed cranially and caudally to visualize the
upper and lower respiratory system. A biopsy of the pulmonary tissue was done and sent for
culture. Closure of the surgical wounds was accomplished with simple interrupted absorbable
sutures. The muscle and the skin incisions were closed in separate layers. The skin layer is
closed with an evertting mattress pattern. The animal was after hospitalized in a terrarium
heated with 32°C.
Results

Clinical history
The animal lived in a terrarium with three other male captives. He had been submitted for clinical examination 4 months ago for stomatitis and large ulcerative lesions on his face. The animal recovered successfully with a 15 days local (cleaning and healing wounds solution and cream: malique a., benzoic a., salicylic a. - DERMAFLONND Solution and DERMAFLONND Crème, Pfizer Santé Animale, 75014 Paris France) and general (gentamicin, 2 mg/kg IM q72h) treatment.

Clinical findings
Clinical findings included a mandibular edema that was not regularly present and consistent with a tracheal lung swelling. General radiodensity of the lung was observed on thoracic radiographs, tending to hiding the heart shadow, suggesting a pneumonia. Differential diagnoses included a bacterial or fungal origin. An infection secondary to his facial lesion was the most likely differential diagnosis. An important inflammation of the air sac and the pulmonary parenchyma was observed during the endoscopic examination. A pure culture of coagulase positive Staphylococcus could have been identified from inflammatory tissues.

Treatment
A treatment of 15 days with amoxicillin / calvulanate (20 mg/kg, IM q24h) and enrofloxacin (10 mg/kg, IM q48h) was initiated. One month later the animal appeared clinically normal, no other swelling has been noted. Radiographs still demonstrated radio densities. It was however less important. The heart shadow was better visualized.

Discussion
As we can see in this case, pneumonia is not always related with an “open mouth breathing animal”. Mandibular oedema or swelling may be a sign of deep pneumonia. In such case, a non obstructive pneumonia may “force” the animal to use its “tracheal lung”. As in mammals and birds, one distinguishes, in the reptiles, two types of endoscopic examinations: coelioscopic of the pleuroperitoneal cavity (technical used here) and non invasive endoscopic examinations carried out via the natural ways (pneumoscopy by trans-tracheal way). The endoscopic examination by trans-tracheal way always requires the use of an endoscope of very small gauge, even in big size reptiles. Moreover, it presents risks of rupture of the tracheal membrane, always very fragile in these animals. The pneumoscopy technique takes advantage of anatomical features of the snake. Puncture of the lung caudal to the respiratory portion minimizes the possibility of haemorrhage. Percutaneous pneumoscopy is a safe method for evaluation of the lower airways of the ophidian patient (5). Bacterial infections are by far the most common cause of pneumonia in reptiles. The common infecting organisms in the respiratory tract in lizards and snakes are Pseudomonas aeruginosa, P. maltophilia, Salmonella Arizona, Providencia rettgeri, Aeromonas hydrophila, Morganella morganii (2). These bacteria become invasive when conditions decrease the resistance of the host, select for pathogenic organisms and/ or follow primary viral infection, such as ophidian paramyxovirus pneumonia (2). The bacterial organisms most commonly isolated in healthy snakes, which implies that they are normal airway inhabitants, are Providencia rettgeri and coagulase-negative Staphylococcus spp. (4).

It is quite uncommon to find a coagulase-positive Staphylococcus spp as the major pathogen agent of pneumonia in a reptile.

Acknowledgements
We thank our assistant Claire Réjaud, the keepers of the Menagerie du Jardin des Plantes, Gérard Dousseau, Franck Simonnet, Cyril Caffelaire and Véronique Marion.
References

STREPTOCOCCAL MENINGOENCEPHALITIS IN A LOWLAND GORILLA
(Gorilla gorilla gorilla)

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Key words: Gorilla, Gorilla gorilla gorilla, Meningoencephalitis, Streptococcus

Extended abstract
A 24-year-old female Lowland Gorilla was observed with weakness of her left arm and left leg. On the following day, she appeared weaker, but was able to eat and drink with assistance. The following morning she was scheduled for an MRI to determine the cause of her illness. She was found agonal and died. The gorilla was submitted to the Diagnostic Center for Population and Animal Health, Michigan State University, with a history of a suspected hemorrhagic stroke and subsequent death.

At necropsy, the primary lesions were noted in the lungs and in the brain. There was moderate subcutaneous edema over the caudal aspect of the left side of the thorax. All tracheobronchial lymph nodes were enlarged. Both lungs were mottled red to dark purple and were wet, heavy, and edematous. There were multiple 1 to 2 cm diameter pulmonary abscesses randomly distributed throughout the cranioventral regions of both lungs. The meninges were slightly cloudy. There was a 1-cm diameter abscess within the right pyriform lobe of the cerebrum in the area of the periamygdaloid cortex and a 0.8-cm abscess within the right ventral thalamic nucleus. The right lateral ventricle was markedly distended with purulent material causing asymmetrical expansion of the right cerebral cortex. There were multifocal hemorrhages around the dorsal and lateral aspect of the right lateral ventricle and within the right corona radiata caudal to the thalamic abscess in the right cerebral hemisphere.

Microscopic examination revealed severe, subacute, suppurative meningoencephalitis, moderate, subacute, suppurative, interstitial pneumonia with multifocal abscessation, degenerative cardiomyopathy and vegetative valvular endocarditis of the right atrioventricular valve. Other lesions included focal, suppurative, tonsillitis, suppurative lymphadenitis of the thoracic lymph nodes, and multiple intraepidermal pustules in sections of haired skin from the chest. Intralesional gram-positive bacterial cocci were observed within abscesses in the brain and lung, as well as in intraepidermal pustules.

Sections of lung, brain, and liver were submitted for bacteriology. Bacterial culture yielded mixed populations of Streptococcus and Staphylococcus. An alpha hemolytic Streptococcus was cultured out of all tissues, and grew in greatest numbers and was therefore considered the most likely primary pathogen.

Streptococcal infections causing vegetative endocarditis are not uncommon in humans and primates. The natural reservoir of streptococcal strains pathogenic for primates is unknown, as is the prevalence of this organism in primate populations. Various streptococci are considered to be part of the normal flora of skin, pharynx, upper respiratory tract, vagina, and prepuce of primates and humans. Interestingly, this gorilla had multifocal epidermal micropustules with intraleosional gram-positive cocci. The source of the infection described in this report could not be determined, but a primary cutaneous infection has to be considered. The most likely pathogenesis of this
gorilla’s illness is a primary vegetative valvular endocarditis with fracture of valvular thrombi causing showers of bacterial emboli to lodge within pulmonary capillaries and the choroid plexus in the lateral ventricle of the right cerebral hemisphere. Subsequent bacterial growth and abscess formation within the thalamic nucleus would have caused the (stroke-like) signs noted clinically. However, primary upper respiratory tract infections as well as gingivitis, otitis and sinusitis have been implicated in the development of streptococcal meningoencephalitis in primates. There was no evidence of a gingivitis, otitis or sinusitis in this case. The presence of multiple pulmonary abscesses made it difficult to determine the exact pathogenesis in this case. Since infections with alpha hemolytic streptococci have been reported in human beings, personnel working with primates, pose as a possible source of transmission and are also at risk for zoonotic primate to human transmission.

References

GENERALISED CALCINOSIS IN TWO TWO-TOED SLOTHS (Choloepus didactylus)

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Key words: calcinosis, hypervitaminosis D, Two-toed sloth, Choloepus didactylus

Extended abstract
Generalized calcinosis due to a Vitamin D intoxication was diagnosed in two adult two-toed sloths (Choloepus didactylus) from a private zoo in Switzerland. The first animal was a male about 13yr old and had been kept in the zoo for eight years. It died after nine days of clinical signs of diarrhoea, weakness and weight loss. The second animal was a 17yr old female that died without previous clinical signs. The male was severely emaciated whereas the female was in a good body condition. Necropsy revealed severe mineralization of the aorta, small to large arteries of several organs, laryngeal mucosa, gastric mucosa, pulmonary alveolar septa, and renal tubules. In the male, there was multifocal subepithelial mineralization of the cornea of both eyes. Both animals were kept in a mixed species exhibit including three two-toed sloths, two night monkeys (Aotus lemurinus) and two prehensile-tailed porcupines (Coendou prehensilis). They were housed indoors with an inverted 12hr light cycle with artificial light. The sloths were fed with various fruits and vegetables, cooked rice, cooked eggs, and dried bread supplemented with a preparation containing vitamins and trace elements (about 6 g). Additionally they received willow and hazel tree branches twice a week and had access to the primate diet (containing 1.9 IU vitamin D3 per gram) that was intended for the night monkeys. Three years before the death of the male sloth, the vitamin preparation containing 20 IU vitamin D3 and 200 mg Ca per gram (Gistocal®, AB Wilnis, The Netherlands) was replaced by a preparation containing 340 IU vitamin D3 and 250 mg Ca per gram (Totalin®, Werner Stricker AG, Zollikofen, Switzerland). Generalized metastatic mineralization occurs as a result of hypercalcemia and/or hyperphosphatemia. The cause of the calcinosis in the sloths was assumed to be due to an over supplementation with vitamin D3 during the last three years. Other factors known to cause hypercalcemia, such as ingestion of plants containing 1,25-dihydroxycholecalciferol or related compounds, intake of a cholecalciferol containing rodenticide, a paraneoplastic syndrome, a primary or secondary hyperparathyroidism and hypoadrenocorticism (2, 6) could be ruled out. No other cases of vitamin D3 toxicosis have been reported in this zoo, although all the animals kept are supplemented with the same vitamin preparation. Whether the sloths have special requirements for vitamin D3 and are more susceptible to toxicosis is unknown. There are several reports on nutritional diseases in sloths, especially during the first months of captivity (1, 3, 4), but no cases of vitamin D3/Ca-related problems have been found in the literature. An interesting finding in this case was the calcification of the cornea, as this may give a hint for generalized calcinosis on clinical examination. A similar finding has been described in cases of vitamin D toxicosis in humans (7). There is a great variation in the vitamin D requirement among different species of animals (5,8) and further investigations are necessary to determine the safe long-term dietary level of vitamin D3 needed by the sloths.
References


CHRONIC ZINC INTOXICATION IN PSITTACINES

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Abstract
After euthanasia of two red-sided eclectus parrots (Eclectus roratus polychloros) an elevated zinc (Zn) level was determined in their livers. Because of some slight alterations in the behaviour of some birds and some non-specific symptoms of illness in some others housed in more or less the same type of enclosures blood samples of all psittacines were collected. 55 % of the birds had an elevated Zn plasma level. The results are discussed.

Keywords: Red-sided eclectus parrot, Eclectus roratus polychloros, psittacines, zinc, plasma, liver.

Introduction
Proventricular dilatation syndrome (PDS) is the name of a syndrome observed in psittacines and characterized by dilatation of the proventriculus. Different aetiologies are recognized like proventricular dilatation disease (PDD), heavy metal poisoning, megabacteria, nematode or fungal infection, gastric impaction, pyloric obstruction and neoplasia (6).

Proventricular dilatation disease is also known under different names like macaw wasting or fading syndrome, myenteric ganglioneuritis and psittacine encephalomyelitis (6). Clinical signs are related to (pro)ventricular and sometimes neurological dysfunction. Anorexia, regurgitation, weight loss can be observed and undigested seeds may be seen in the faeces. Typical histological lesions of PDD are lymphocytic and monocytic infiltration of intrinsic and extrinsic splanchnic nerves of the muscularis tunics of the alimentary tract. Other alterations are leiomyositis in organs innervated by affected nerves, a non-suppurative encephalitis, myelitis and radiculoneuritis (3, 5, 6, 7). A viral aetiology has been suggested (3, 6) and therapy is still in an experimental stage (Schoemaker N, pers.comm.).

Zinc intoxication can be the result of the ingestion of zinc containing foreign bodies like certain coins but also by the intake of zinc from a wire mesh enclosure, usually referred to as “new wire disease”, (1, 2, 7, 9, 10, 11). Zinc is present in galvanized wire. The risk of toxicosis can be reduced, but not eliminated, by scrubbing the wire with an acidic solution (vinegar) (2, 8). Other possible sources of zinc are clips used to construct enclosures, padlocks, hangers, food sticks. An excess of zinc can lead to sudden death, lethargy and weakness, neurological (ataxia, paresis, seizures) and gastrointestinal (anorexia, vomiting) symptoms, anaemia and polyuria/ polydypsia (PU/PD) (2, 5, 7, 9, 10). A more chronic course has also been described in which dullness, weight loss and intermittent excretion of greenish droppings could be observed. Gross necropsy findings were unremarkable, except for signs suggestive of impaired gastrointestinal tract motility (5). Therapy consists of the administration of chelating agents and supportive treatment (2, 9, 10).

Case Report
A more than 11-year-old female red-sided eclectus parrot was presented in a reasonable body condition with the right wing drooping. Her faeces were slightly too loose but no
parasites or other abnormalities could be detected. Haematology and serology revealed no abnormalities and polymerase chain reaction (PCR) on polyoma virus and Chlamydophila was negative. The bird had never produced fertilized eggs but had always been a good foster parent.

Radiographs were taken, also after giving barium sulphate per crop needle (at 0, 30, 75, 135 and 255 minutes). The radiographs were suggestive of proventricular dilatation syndrome and proventricular dilatation disease was suspected. The bird was euthanized. Eight days later the male cagemate started regurgitating. Radiographs showed a dilated proventriculus and this bird was also euthanized. The previous year this bird had experienced a period of abnormal use of the legs and perching.

Material and methods, results

Post mortem examination and results

At necropsy, the liver of the female was soft and enlarged with bulging edges. The kidneys were also slightly swollen and soft. The proventriculus did not look clearly dilated and was almost empty, the content of the intestines was somewhat mucoid. There were no other significant findings and bacteriology of the liver and kidney was negative.

The male was in a good condition, the liver was also soft with bulging edges. The kidneys were rather dark and slightly swollen, the proventriculus was dilated, the ventriculus empty. The intestines showed some hyperaemia but otherwise no significant findings. Bacteriology of liver, lung and kidney of this bird was also negative.

Tissue samples from liver, lung, kidney, crop, proventriculus, ventriculus, pancreas, duodenum, and jejunum were fixed in 4% phosphate-buffered formalin, embedded in paraffin, cut at 4 µm sections and stained with hematoxylin and eosin (H&E).

Histologically, the liver showed slight fibrosis with predominantly lymphocytic infiltrations in the portal areas. In the lung, subepithelial in the parabronchial areas, mononuclear infiltrations and anthracosis were evident. In addition to cystic, protein material containing tubuli, interstitial foci of lymphocytic infiltrations were present. The crop, proventriculus, the intestines, and the pancreas showed no lesions. Lymphocytic infiltrations associated with vessels were seen in the serosa of the ventriculus. The nerves were not affected.

As there was no histological evidence of PDD it was decided to determine zinc levels in the livers of the dead parrots. Frozen liver of the two birds and plasma from the dead female were sent to the University Laboratory for Farm Animals and Horses of the Faculty of Veterinary Medicine and analysed for zinc levels using flame atomic absorption spectrometry (FAAS Varian AA 250 plus, Varian Springvale Australia).

The liver of the female red-sided eclectus parrot contained 339 mg zinc /kg d.m., the liver of the male 123 mg zinc /kg d.m.. Plasma from the female was also sent to the laboratory and contained 67 µmol/ L Zn.

Additional research and results

Heparin blood samples of all psittacine birds in the collection were collected to determine the zinc levels. The results are summarised in Table 1. Birds marked with an asterix were reported to have close contact to the wire and other pieces of metal.

Discussion

In the dead parrots focal mononuclear infiltrations were present in the liver and the kidney. This finding is in accordance with the histopathological changes described in zinc intoxication (5).

Liver zinc concentrations greater than 100 mg/ kg d.m. are considered toxic and can be diagnostic for zinc toxicosis (7). Both the female and the male liver zinc concentrations were higher than 100 mg/kg d.m..
A study involving 260 blood samples showed that the average physiological zinc concentration in plasma or serum is at or below 30 µmol/L in most psittacines. Cockatoos and Eclectus parrots appear to have higher normal blood levels: 45 and 37.5 µmol/L respectively. In liver samples no difference with other genus groups could be detected (7).

The female red-sided eclectus parrot had an elevated zinc plasma level. Seven out of eight birds of the group reported to be often in close contact with the wire had an elevated zinc plasma level. The plasma of eight out of the 20 other birds contained too much zinc. Thus 55% of all the psittacines in the collection appeared to have elevated zinc levels. Birds which were reported to have the closest contact to the wire and other pieces of metal, showed a higher zinc level. The hyacinth macaws with the lowest values are housed in a complete stainless steel cage. Galvanized dishes, powder coated cages and zinc containing paint have never been used. Despite this, investigation learned that in almost every cage smaller or larger zinc sources were present. Examples are galvanized screws and structural steel, hinges, painted galvanized wire from which some paint had disappeared. A few nesting-boxes had the entrance covered with a ring of zinc, thus preventing the birds breaking down the wood by gnawing. Sometimes psittacines were placed in cages meant for other birds; these were made of galvanized wire. A case has been described in which 6-month-old cages treated with an acetic acid solution still can caused an intoxication. Zinc-tainted water in this enclosure did the same four years later (8).

Acute zinc intoxication may lead to the sudden death of an animal. At Rotterdam Zoo this has never been the case. During the last years, only occasionally non-specific symptoms like feather picking, drooping wings, abnormal use of the legs and periods of PU/PD have been

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noticed in the collection. In some cases like the couple red-sided eclectus parrots fertility was not as good as expected. In a case of orange-bellied parrots (Neophema chrysogaster) the fertility rate was 54%, 59% and 66% respectively in 3 consecutive years. After replacing the galvanized wire the fertility rate increased to 86% the next year (4).

Our conclusion is that at Rotterdam Zoo several psittacine birds have been suffering from a chronic form of zinc intoxication without obvious clinical symptoms. Therefore, the enclosures were thoroughly changed and stripped from all zinc containing materials.

References
CHRONIC COPPER INTOXICATION IN A CAPTIVE NILE CROCODILE
(Crocodile niloticus)

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Abstract
A thirty year old female Nile crocodile (Crocodile niloticus) died after sudden onset of anorexia, apathy, indigestion and hemolysis. Its stomach contained 14 pieces of different coins, minted 16-28 years back. All of them were made from copper alloys. Copper presence in liver and kidney was demonstrated by rubean acid staining. Copper concentrations in the liver and kidney were 1022 and 360 ppm, respectively. The animal suffered from necrotic and ulcerative gastritis and enteritis, diffuse necrosis of the liver and chronic degeneration of hepatocytes, hemoglobinuric nephrosis, interstitial pneumonia and perimysial necrosis of the muscles. The immediate cause of death was bacterial sepsis (Aeromonas hydrophila, Proteus sp. and Escherichia coli) erupted due to chronic copper intoxication and organ degeneration, leading to gradual immunosuppression.

Introduction
Copper is an essential trace element, which is an important catalyst for heme synthesis and iron absorption. Besides zinc and iron, copper is the third most abundant trace element in the body (2). Toxicity and accumulation of copper in eukaryotic cells has already been studied. Higher accumulation of proteins and lipids per cell was observed, as well as failure in completing cell division (4). Most information about copper toxicity in vertebrates concerns humans and large mammals, especially ruminants. In reptiles copper was studied as a part of environmental pollution load (3,7). Chronic copper toxicity is rare and primarily affects the liver (2). In mammals, copper from external sources builds up to 150 ppm or more in the animal’s liver, then, it is released following a stress event. Common clinical signs in mammals include anorexia, weakness, lethargy, vomiting, dehydration, jaundice, hepatomegaly, hemolytic crisis and hemoglobinuria. Specific treatment is based on D-penicillamine and ascorbic acid, and is generally unrewarding except the acute intoxication (6).

Case description
A thirty years old female Nile crocodile (Crocodile niloticus) died after sudden onset of anorexia and apathy in October 2001. Prior clinical examination revealed gas distension of the gut. The blood was sampled for plasma chemistry. The animal was treated with intramuscular clonobutin (750 mg pro toto), ascorbic acid (1000 mg pro toto) and ceftazidim (1000 mg pro toto), and 5 per cent glucose intraperitoneally. It died 3 days later.

Methods
A male and two females of the Nile crocodile (Crocodile niloticus), kept in Brno zoo, were imported from Africa presumably in 1973. That is why we believe that the animal was more than 30 years old at the time of death. Detailed records concerning their keeping up to 1985 are missing. From 1985 on the animals were kept in a glass enclosure, without any contact

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with visitors, under temperatures of 24-30 °C. One half of the enclosure was land, the other half water. No substrate was at the bottom of the water tank. Food consisted mainly of freshwater fish, rats and chicken. Once a week food was powdered with complete vitamin and mineral supplement (Vitamix for laying hens, Biofaktory CR; Plastin, Bioveta CR). The male bred successfully with both females for over 10 yrs. None of them suffered from any disease throughout their lives. The body weight of the female was about 35 kg at the onset of clinical signs. It’s reproductive organs were active.

Clinical examination of the animal was possible with mechanical restraint only, without any sedation. Blood was taken from the ventral coccygeal vein and heparinized submitted to the laboratory of the Veterinary and Pharmaceutical University in Brno. Samples for histology were fixed in 4% buffered neutral formalin and submitted to the private laboratory T & M, Brno, for histology. The same lab made for us the histochemical staining for the copper presence in the liver and kidney. The method of rubean red staining was used. The samples were stained with 4% solution of rubean red for 24 hours. Samples for toxicology were freezed and submitted to the State veterinary institute, Brno, Czech Republic.

**Results**

**Clinical pathology**

The main changes in plasma chemistry were increased level of uric acid, aspartate aminotransferase (AST/GOT) and alkaline phosphatase (ALP) (Table 1). Blood was hemolitic.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Unit</th>
<th>Value</th>
<th>Reference interval (1,5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total protein</td>
<td>g/l</td>
<td>35</td>
<td>37-85</td>
</tr>
<tr>
<td>Uric acid</td>
<td>µmol/l</td>
<td>546.2</td>
<td>80-450</td>
</tr>
<tr>
<td>ALP</td>
<td>µkat/l</td>
<td>25</td>
<td>up to 2*</td>
</tr>
<tr>
<td>AST/GOT</td>
<td>µkat/l</td>
<td>21.6</td>
<td>0.3-1.6</td>
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<tr>
<td>ALT/GPT</td>
<td>µkat/l</td>
<td>8.9</td>
<td>0.6-1.4</td>
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<td>Glucose</td>
<td>mmol/l</td>
<td>3.8</td>
<td>3.4-9.6</td>
</tr>
<tr>
<td>Potassium</td>
<td>mmol/l</td>
<td>7.8</td>
<td>4.4-5.0</td>
</tr>
<tr>
<td>Sodium</td>
<td>mmol/l</td>
<td>125</td>
<td>132.6-152.2</td>
</tr>
<tr>
<td>Calcium</td>
<td>mmol/l</td>
<td>4.2</td>
<td>2.2-3.4</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>mmol/l</td>
<td>7.1</td>
<td>0.42-1.45</td>
</tr>
</tbody>
</table>

* personal experience

**Post mortem**

At necropsy the crocodile was in good body condition having both subcutaneous and intracoelomic fat deposits. The liver was of tough consistence and had rounded edges. The stomach contained 14 coins (Table 2) and extensive mucosal erosion, covered with emerald green mucus. Anemia, hyperemia of the lung and segmental hyperemia of the intestine was found. Bacteriologic examination of the organs revealed *Aeromonas hydrophila*, *Proteus sp.* and *Escherichia coli*.

Histologic examination revealed necrotic and ulcerative gastritis and enteritis, diffuse necrosis of the liver and chronic degeneration of hepatocytes, hemoglobinuric nephrosis, siderin in the renal parenchyma, interstitial pneumonia and perimysial necrosis of the muscles. Histochemical staining of the liver and kidney for the copper presence
demonstrated copper deposits in hepatocytes, renal tubular cells, and in intima of the big vessels.
Toxicologic analysis revealed copper concentration in the liver and kidney amounting to 1022 and 360 ppm, respectively.

**Numismatic analysis**
All the coins were subjected to numismatic analysis. We checked the year of production, content, weight loss and total pure copper loss, which amounted to 3.856 g of copper.

<table>
<thead>
<tr>
<th>Coin/ weight of a new coin (g)</th>
<th>Count (pcs)</th>
<th>Actual weight of all pcs (g)</th>
<th>Composition</th>
<th>Total loss of copper from all pcs (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 Kcs / 7</td>
<td>1</td>
<td>6.6</td>
<td>Cu:Ni 80:20</td>
<td>0.320</td>
</tr>
<tr>
<td>1 Kcs / 4</td>
<td>1</td>
<td>3.6</td>
<td>Cu:Al:Mn 91:8:1</td>
<td>0.364</td>
</tr>
<tr>
<td>50 Hal older / 3</td>
<td>2</td>
<td>5.2</td>
<td>Cu:Zn 90:10</td>
<td>0.720</td>
</tr>
<tr>
<td>50 Hal younger / 3,2</td>
<td>2</td>
<td>6.15</td>
<td>Cu:Ni 80:20</td>
<td>0.200</td>
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<td>20 Hal / 2,6</td>
<td>8</td>
<td>17.95</td>
<td>Cu:Zn:Ni 79:20:1</td>
<td>2.252</td>
</tr>
</tbody>
</table>

**Total loss of copper from all coins (g) 3.856**

**Discussion**
I our opinion, the cause of death of the female Nile crocodile (*Crocodylus niloticus*) was multifactorial. In entirely all organs, submitted for bacteriology, a mixed culture of *Aeromonas hydrophila*, *Escherichia coli* and/or *Proteus sp.* was found. These species belong to facultative pathogens. They are common inhabitants of water with organic remnants. We considered them to be only secondary pathogens and searched for some immunosupresive agents. The intravitral plasma chemistry (elevated liver enzymes, low total protein) and necropsy finding (hemolysis, anemia, gastric ulceration, liver degeneration, renal tubular necrosis) corresponded to the finding of coins in the stomach and to our preliminary diagnosis of chronic copper intoxication.

We made an effort to answer two main questions:
I) How long were the coins in the body?
II) How many grams of copper released from them?

Ad I) The oldest coin was minted in 1964 and was valid up to 1977. It could, therefore, be ingested from 1973 (i.e. animals came to the zoo) to 1977, which means 24-28 years ago. The youngest coin was made in 1979 and was valid up to 1992. It could be ingested from 1979 to 1985 (i.e. the documented closed enclosure), which means 16-22 years ago.

Ad II) We weighed all the coins carefully and calculated the pure copper loss. It amounted to a total 3.856 g of copper.

All information has certain limit: A) We do not know, if there were more coins in the body, which passed through by the time. B) We do not know the intensity of copper elimination and the amount of copper really excreted from the body.

In our case we assume following pathogenesis: copper from external source (coins) accumulated in the body (principally liver) for 16-28 year. Due to this chronic copper load, organs gradually degenerated and immunocompetence decreased. When the threshold organ capacity was exceeded, opportune bacteria affected the body. Due to this stress,
copper from depots was released and signs of acute copper intoxication emerged (lethargy, hemolysis, possible hepatoencephalopathy, elevated liver enzymes, hemoglobinuric nephrosis). In sheep and dogs with copper intoxication the liver concentration is higher than 150 and 1000-2000 ppm, respectively (6). Comparing this, copper concentration of 1022 ppm in the liver and 360 ppm in the kidney of the female crocodile, is high enough to be considered as an evidence of copper poisoning.

Acknowledgements
We thank prof. F. Tichy for the histologic examination of tissues, all keepers for their help and all the laboratory staff for their dependable work. We are most grateful to Jiri Pikula for his language help.

References
CLINICAL TOXICOLOGY AT THE BUDAPEST ZOO.

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Abstract
Three case reports are mentioned, all of them deals with the clinical toxicology at the Budapest Zoo. The species are involved the Parma wallaby (Macropus parma) (9), the Egyptian fruit bat (Rousettus aegypticus) (7), and the White-tailed sea eagle (Haliaeetus albicilla) (10). The aim of this paper is to give a context between keeping methods and the source of the toxicological episodes and to highlight the diagnostic difficulties the veterinarian often encounters at zoos.

Key words: Parma wallaby, Egyptian fruit bat, White-tailed sea eagle, toxicology, pesticide.

Introduction
The toxicological work has often very limited boundaries at zoos. If the clinician does not have a proper anamnesis it can be exceedingly difficult to set up proper and fast diagnosis (6). To achieve our goals we have to use laboratories. Despite of this the toxic agent is not always revealed, since the number of the potential toxins are numerous, the clinical course is often very rapid, and the costs of the laboratory analysis can be very high.

Material and Methods
The three selected cases represent three very different aspects of clinical toxicology at our institution. Two of the cases are well explored, but the one which affected the Parma wallaby stock is not totally solved yet.

The first case involved 16 Egyptian fruit bats which were a part of a larger group (91 animals). 6 animals were treated, 4 of them survived (the untreated animals dead suddenly and treatment was not possible). The carcasses were submitted for necropsy examination to the Department of Wildlife Diseases and Parasitology of the Central Veterinary Institute, Budapest. Tissue samples were obtained from the lungs, liver, spleen, kidneys, duodenum and brain and were fixed in 10% formaldehide solution. Routine histological sections were prepared and were stained with hematoxilin-eosine. Liver tissue and stomach contents were submitted for toxicology. The analysis was performed by GCMS.

The second case involved 6,3 Parma wallabies from a New Zealand originated stock (25 animals). Despite of the treatment only 2 animals survived this episode. The carcasses were submitted for necropsy examination to the Department of Wildlife Diseases and Parasitology of the Central Veterinary Institute, Budapest. Tissue samples were obtained from the lungs, liver, spleen, kidneys, heart, duodenum and brain and were fixed in 10% formaldehide solution. Routine histological sections were prepared and were stained with hematoxilin-eosine. Liver tissue and stomach contents were submitted for toxicology. The analysis was performed by GCMS.
The third case represents a White-tailed sea eagle which was a rescued bird, originated from the Great Plain area of Hungary. Blood was collected and x-ray was made. After a 4-week long treatment of the emaciated bird it made a full recovery and was released back to the wild.

Results

Case 1

Clinical history

The Egyptian fruit bat case followed a pesticide treatment at the building where the animals were kept. The company who regularly does large scale pesticide controls carried out this certain procedure without any former information towards the Veterinary Department at the Budapest Zoo. They used a pesticide containing organophosphate (chlorpyrifos). This pesticide use was rapidly followed by the death of 12 animals (including 2 fetuses), and the general weakness of 6 animals. The clinically ill animals showed muscle tremors and weakness, diarrhea and refused to take food. These were separated and treated with atropine-sulfate (0,2 mg atropine-sulfate/individual SID, Atropinum sulfuricum 0,1 %, EGIS). Additionally, fluid therapy and individual feeding was carried out. The treatment lasted for 4 days and 4 of the animals survived.

Post mortem

9 adults (4 males, 5 females, one of them was pregnant), 1 juvenile and 2 fetuses were examined. The animals were in good condition. Gross lesions included external signs of diarrhea, congestion of the liver, spleen and kidneys, empty stomach, a swollen mucosa in the small intestine with mucous contents. Histopathology of the livers showed lesions ranging from multifocal haemorrhages to mononuclear infiltration with activated macrophages. Haemorrhages and necrosis of tubular epithelium were observed in the kidneys and atrophy of lymphoid follicles in the spleen. Lungs had alveolar emphysema and congestion, but oedema was also found in two animals. Acute catarrhal enteritis with haemorrhages in the mucosa of the small intestine (duodenum) was found. We cultured Arcanobacterium haemolyticum from 7 livers (3 adults and the fetuses were negative).

Toxicology

GCMS was used. All the carcasses were examined (liver tissue, stomach contents) and all of them contained chlorpyrifos.

Case 2

Clinical history

The Parma wallaby case involved nine (6 males, 3 females) animals, which were imported from New Zealand cc. 10 months beforehand. Only 2 of the affected animals survived. The clinical symptoms were general weakness, ataxia, tonico-clonic seizures. The x-ray showed tympania at the intestines and interstitial pattern in the lung. Treatment was immediately started before any laboratory results and toxoplasmosis was suspected. At the same time blood was collected and plasma was sent for Toxoplasma serological testing. Faecal samples were also taken. As the blood results revealed a very severe damage of the liver and kidney functions (ALT up to 3781 IU/l, AST up to 5318 IU/l, urea up to 31.8 mmol/l, CREA up to 184 µmol/l, phosphorus up to 5 mmol/l) the treatment involved fluid therapy, support of the liver and treatment against toxoplasmosis (natrium chloratum solution, glucose, vitamin B complex, electrolytes, aminoacids, dextrose, sulfonamides, clindamycine). Despite of the treatment 7 animals died within 1-7 days. The severity of the symptoms and the length of the survival
was not related. Due to the clinical course toxicosis was the most likely case of the deaths of the animals.

Post mortem
The animals were in weak condition. 3 adult Parma wallabies (1 male, 2 females) had oedema and haemorrhages in the subcutis. The pale, slightly enlarged liver contained a few greyish foci with blurred edges. The kidneys and spleen had no visible lesions. The lungs were oedematous, with froth in the trachea, and swollen mucosa. The heart was dilated. A thin, whitish necrotic layer on the tongue and oesophagus mucosa was visible. The mucosa of the small intestine was swollen, it contained greyish, mucous contents. The histopathology revealed acute tubular necrosis in the kidney. The liver showed centrolobular hepatocyte necrosis and hydropic degeneration with haemorrhages. Eosinophylic infiltration and oedema of the submucosa of the small intestines was found. Escherichia coli cultured from the small intestine. The lung of another adult male showed alveolar cell degeneration, necrosis and desquamation. These cells also showed erythrocytophagocytosis. Congestion of the lung, serous infiltration and intraalveolar bleeding were also obvious signs.

Toxicology
GCMS was used. Stomach contents and livers were analysed. The results were negative for organophosphates and 2,4-D, MCPA and MCPB herbicides.

Case 3
Clinical history
The White-tailed sea eagle case involved one, presumably female bird. This bird was fed on injured waterfowl nearby a hunting area. The bird lost its ability to fly and had a greenish diarrhea. The x-ray showed lead shots at the gizzard, a smaller liver, radiodense kidneys and a mild lung edema. Clinical diagnosis was based on the anamnesis and this picture, but at the same time blood was collected to detect the plasma level of the lead. Ca-EDTA (40 mg/kg, SID, Detoxamin suppositories, World Health Products) and supportive therapy was used. The bird showed fast improvement. After a 4-week long period the eagle became so anxious, and it was not able to keep it longer in a confined space. Therefore, we decided to shorten the rehabilitation period and released back to the wild (after the proper marking).

Toxicology
Gas chromatography was used. The results were negative for lead poisoning.

Discussion
The above mentioned cases well represent that clinical findings regarding toxicology are not always relevant. The Egyptian fruit bat case is fully explored and some of the animals were responded to the treatment. It is possible that those animals which were found death also showed some clinical signs beforehand, but the detection was difficult because of the large size of the group. The organophosphate poisoning is due to the irreversible inhibition of acetylcholinesterase, thus the acetylcholine activation dramatically increases on the nicotinic and muscarinic receptors and the cholinergic pathways at the central nervous system (1, 11). The Parma wallaby case only shows an evidence according to the histopathology. A thorough clinical work excluded the most common infectious agents which cause CNS signs in marsupials. We got the laboratory results during the treatment, therefore we had to adjust this to the received results. The histopathological picture of the lung is very similar those which can be found in acute paraquat or diquat toxicosis. Unfortunately, the examining laboratory did not store tissue samples for further analysis and the negative toxicological result can be explained by the metabolism of the toxic agent. The source of the toxin was most likely the food. We must mention that due to the fast course of this case it was very difficult to select the most proper tools for diagnosis. From a retrospective view some more
laboratory analysis should have been made (eg. analysis of the food). This case is not fully explored, but according to our opinion demonstrates a fine example when a zoo veterinarian must deal with clinical toxicology. One must eliminate every source of the possible toxins, though the laboratories are often slower than the course of the disease and the costs are very high (especially if the anamnesis does not give a clue about the possible toxins).

The White-tailed sea eagle case was well diagnosed by the x-rays and the consecutive treatment was effective. Heavy metal lead intoxication is quite common in wild birds of prey. Lead particles in the gizzard are exposed to stomach acids and dissolve (2, 3, 4, 5). Unfortunately, the measuring of the ALAD (delta-aminolevulenic acid dehydratase) level (8) was not possible due to the analytic procedure. This case was demonstrated since a twofold reason: the suppositories were as effective as the intramuscular route of Ca-EDTA and despite of the negative laboratory result the clinical evidence was enough for successful treatment.

Acknowledgements
We thank the Central Veterinar ian Institute Toxicology team for their efforts. Csaba Jakab from the Veterinar Faculty of Szent Istvan University gave us an invaluable help while analysing minor histopathological changes.

References
MALIGNANT LYMPHOMA IN A MANATEE

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Abstract

We will be findings were insignificant but included fleshy appearance of the kidneys. The diagnosis was based on histopathological and immunohistochemical studies. Biopsy samples from 10 organs were subjected to immunohistochemical staining using 10 commercially available antibodies: CD3/polyclonal, CD5/CD5C6, CD8/4B11, CD20/L26, CD21/1F8, CD45/2B11+PD7/26, CD45RO/UHCL1, CD79a/JCB117, CK/LP34 (DAKO, Glostrup, Denmark) and CD23/1B12 (Novo Castra, Newcastle, presenting the pathological findings in a West Indian manatee (Trichechus manatus) with malignant lymphoma. The lymphoma was infiltrating the lung, liver, kidney, mesenteric lymph nodes and eye and was identified as the cause of clinical disease in the manatee. Gross pathological UK). The cytoplasm of near to 100 % of the neoplastic cells was positive for CD3, a marker for T-cell differentiation. The neoplastic cells were negative for other applied markers. Few background cells in the lymphatic organs stained positive for CD79a and CD20, markers of B-cell differentiation. The cause of this neoplasm was not determined. This is the first report of lymphoid neoplasia in the mammal order Sirenia, though lymphoid neoplasia has been described in a wide range of other mammals.
TRAUMATIC SKIN INJURY IN A GOLDEN-HANDED TAMARIN (Saguinus midas).

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Abstract
A male golden-handed tamarin (Saguinus midas midas) suffered from a traumatic skin injury of the left shank. The general health status of the patient remained unimpaired. Having performed a thorough review and having attended to the wound, we opted for systemic application of antibiotics and antiflogistics; no bandage was applied to the wound at this stage. With respect to persistent autotraumatization of the wound, an Elizabethan collar was used, the wound was covered with dry-to-dry bandage, and the patient was administered neuroleptics (haloperidol). Besides that, the animal was separated from the pregnant female with whom he had shared a cubicle. After 6 days, when the wound started granulating, a wet-to-dry bandage was applied. The wound was treated with a small amount of natrium hyaluronate and betadine gel, advantage being taken of the viscoelastic properties of hyaluronate and the antiseptic properties of the iodine to be released. Paraffin gauze dressing was used to provide a non-adhesive wound cover. In the beginning, the healing process was complicated by continuous traumatization of the wound on the part of both the patient and the female in the cubicle they shared. A bone sequester was eliminated in the final stage of the healing process. Full consolidation of the wound occurred within 52 days from the initial treatment. The scar covered with fur within 28 days from the full consolidation of the wound.

Keywords: golden-handed tamarin, Saguinus midas, skin injury, trauma, natrium hyaluronate, Elizabethan collar.

Introduction
Traumatic lesions are the second most common clinical problem in nonhuman primates after diseases of the alimentary tract (Bielitzki, 1998). In nonhuman primates, traumatic lesions are addressed in a similar way as in dogs and cats (Bielitzki, 1998). Although the principles of wound consolidation are the same, the approach, especially to everyday care and patient immobilization, is different (Fowler, 1995; Horne, 2001; Hubbard, 2001). The patient must often undergo repeated anaesthesia. Where the animal is kept as a pet, collaboration with the owner may be difficult; for this reason the therapeutic care and drug application should be thoroughly explained to him.

Material and Methods
Clinical history
The owner reported he had noticed a small hurt 10 days ago. The wound seemed likely to be caused by a wire protruding out of cage equipment. The wound became substantially bigger. The tamarin keeps licking it, but the limb bears full weight. Both food intake and activity have remained unchanged. The patient has been kept in the cage for three years, together with a female, who is pregnant at the moment (the exact due date is unknown). To date, 4 youngs
from 2 litters have been reared. The diet consists of insects, fruits, vegetables, and commercial food for marmosets and tamarins. The animals have constant access to drinking water. The cage is situated in a flat inhabited by the owner; the dimensions of the cage are 1.5 x 0.7 x 1.2 m.

An adult male golden-handed tamarin (S. midas midas) weighing 0.61 kg, aged 4 years, had been submitted for clinical examination. Due to the aggressivity and stress shown by the animal, an examination of the cardiovascular and respiratory systems and rectal temperature taking were followed by anaesthesia by isoflurane (Isoflurane, Rhodia) and oxygen mixture. A clinical examination identified a wound on the left hind limb. Sized 3.5 x 1.2 cm, the wound (Fig. 1) was located on the craniomedial side of the shank. The lesion affected the cutis and subcutis; muscles were but partly affected. The wound was contaminated with animal hair only; the hairs surrounding the wound were shorter due to licking. Skin elasticity was intact. The intestine contained mashy chyme. The remaining organ systems were found without pathological changes. With respect to the size and nature of the wound, blood from v. jugularis was taken for haematological and biochemical testing (without any abnormal values).

The primary treatment of the wound (on day one) included a general sterile physiological saline rinse under pressure. Having removed all contaminants thoroughly, 0.1% poviniodide saline was applied. The lesional tissue was swabbed dry and tinct. nigra was applied. The skin adjacent to the wound was treated with antipruriginous drugs (Dimentideni maleas, Finistil gel, Novartis Consumer Health SA, Nyon, Switzerland). No bandage was applied. The drugs administered included parentheral antibiotics (amoxicillin-clavulonate, Synulox RTU inj. Ad us vet., Pfizer, Italy, 15 mg/kg s.c. sid), antiflogistics/analgesics (flunixin-meglumine, Finadyne RP inj. Ad. Us vet., Schering-Plough, Switzerland, 1 mg/kg i.m. sid), and vitamin C (20 mg/kg s.c.). Drug application on the respective days, rectal temperatures, and patient weight are summed up in Table 1. The owner was recommended to separate the patient from his female mate. Due to the pregnancy of the female, the owner did not assent to the proposal, fearing the female might be exposed to undesirable stress before giving birth. Due to continuing autotraumatization, the patient was made to wear an Elizabethan collar on day three and the wound was dressed with a dry-to-dry bandage. The wound was covered with several layers of sterile gauze and secured with a self-adherent wrap (Cobane™, 3M, Germany). The tamarin ripped off the Elizabethan collar and subsequently also the bandage on day four, ending up with a widened wound. The wound was treated in the same way as on day three. The patient ripped off the Elizabethan collar on day five again and due to automutilation, the size of the wound increased to 4.8 x 3 cm (Fig. 2). Deep muscle layers were affected this time and the tibia got exposed. The owner reported intensive licking by the patient as well as the female. The wound was cleaned and antipruriginous drugs were applied to the adjacent skin. Neuroleptics (haloperidolum, Haloperidol-Richter gtt., 1 gtt. p.o. bid) were administered orally and a dry-to-dry bandage applied again. With respect to the potential injury to bone tissue, a combination of enrofloxacin (Baytril 2.5%, Bayer AG, SRN, 5 mg/kg i.m. sid) and potentiated sulphonamides (Borgal 7.5 % inj. ad us. vet., Intervet, Netherlands, 25 mg/kg s.c. sid) was administered. This combination of antibiotics was administered for 10 days. The patient was separated from his female mate.
Thanks to the greater sedation of the patient, the dose of haloperidol could be lowered to 1 gtt/day p.o. on day six. To deal with originating granulation, tinct. nigra was applied. The wound was covered with paraffin gauze dressing infused with high-molecular-weight natrium hyaluronate combined with Betadine (Hyodine, Contipro, CZ). Several layers of sterile gauze were used to provide absorbing material. A third layer was a self-adherent wrap.

The wound was treated in a similar way throughout the next period (tinct. nigra, Hyodine®, wet-to-dry bandage). The Elizabethan collar was replaced every 2-4 days depending on the degree of contamination with food. Antibiotics and tranquilizers were discontinued on day 15. On day 25, the male returned to the cage to share it with the female again – the bandage as well as the Elizabethan collar were tolerated well at this stage.

The wound was healing finely with the exception of a small area on the craniomedial side of the limb, where a bone sequester was formed (Fig. 3), to be removed on day 30 following the first veterinary appointment. On day 52 after the start of therapy a scar covering the wound was formed; the wound covered with fur again on day 80.

Results and discussion
Traumatic skin lesions are relatively frequent in nonhuman primates (Bielitzki, 1998; Hubbard, 2001). While with wounds situated on the proximal parts of limbs, the process of healing and the wound treatment protocol are similar as with body and neck lesions, lesions situated in the distal parts of the limbs are a distinct group of injuries requiring special care. Generally, even small lesions may pose a serious threat to the future functionality of the limb, or even threaten the patient’s life. These injuries generally affect the skin and bacterial infection of traumatic injury is routine. For this reason, these injuries should be left to heal by secondary healing. Brancker (1985) believes that even primary suture on limb skin may stimulate primates to take the suture out. Considering the size, localization, and nature of the wound and the time elapsed since the occurrence of the injury, we left the wound to heal by secondary healing (Fossum, 1997; Necas et al., 2000). The first-choice antibiotics used in therapy of skin injuries are beta-lactams (Fossum, 1997; Kirk et al., 1995). This is why we chose amoxicillin-clavulonate for
the initial treatment and used a combination of enrofloxacin and potentiated sulphonamides after the bone was exposed.

Sterile physiological saline or antiseptic 0.05% chlorhexidine or 0.1-1% povidoniodid salines are used for thorough wound lavage (Fossum, 1997). Kirk et al. (1995) prefer chlorhexidine to povidoniodid. Keeble (1999) reports rinsing traumatic injuries in wild animals with povidoniodid only. In contrast to that, Butinar and Pecar (2003) opt for lavage with sterile physiological saline or Ringerlactate at the initial stage. The aim of this kind of lavage is primarily to reduce the bacterial concentration and to remove smaller foreign matter mechanically.

Reviewing and cleaning wounds in these semi-wild animals requires anaesthesia. For repeated short anaesthesia a mixture of isoflurane and oxygen is used (Fowler, 1995; Thurmon et al., 1996; Horne, 2001). The analgesic and antiflogistic used was flunixin-meglumine at 1 mg/kg sid administered for 5 days. If the drug is administered for longer periods, it may have a negative impact on the kidneys (Thurmon et al., 1996; Flecknell, 2001).

It is believed that after a thorough lavage and cleaning, a wound should be dressed. Due to the nature of primates, keeping the bandage (wound) clean and preventing undesirable traumatization is however onerous, for which reason the Elizabethan collar must be applied in some cases (ohnson-Delaney, 1994; Bielitzki, 1998). Contaminated wounds are recommended to be dressed with adherent or absorbant bandages in the beginning for several days, to be replaced with wet-to-dry or non-adherent bandages after granulation tissue starts forming. Due to the relatively unimportant exsudation, we chose a dry-to-dry bandage. On day six after the primary treatment, granulation tissue started forming. Formation of this tissue can be supported by using tinc. nigra or viscous materials (Necas et al., 2000; Butinar and Pecar, 2003). At the Avian and Exotic Animal Clinic (University of Veterinary and Pharmaceutical Sciences, Brno, Czech Republic), we have had positive experience using a combination of natrium hyaluronate and iodine (Hiodine – natrium hyaluronate + potassic iodine + iodine, Contipro, CZ) to support granulation. High-molecular-weight natrium hyaluronate is routinely used in treatment of joint (Slatter, 1993; Fossum, 1997) and eye (Al-Farzai, 2003; Kottmann et al., 2003) conditions. Its viscoelastic properties are taken advantage of in human medicine in therapy of poor-healing infected wounds in combination with zinc (Curiosin gel, Chemical Works of Gedeon Richter Ltd., Budapest, Hungaria) or sulphonamides (Ialugen, plus IBSA, Lugano, Switzerland), too. Paraffin gauze dressing was used to provide a non-adhesive cover of the wound.

Initially, the healing was complicated by permanent traumatization of the wound by the patient as well as his female mate sharing the cubicle with him. For this reason, we opted for patient sedation with orally administered haloperidol (Vodicka, 2002). Haloperidol ranks among derivatives of butyrophenone, a drug with dopamine receptors inhibiting action. Activization of these receptors enhances motor activity and behaviour stereotypes in experimental animals (Katzung, 1992). At this point, the tamarin was separated from his female mate. Due to the pregnancy of the female, the step was taken following an agreement with the owner only when it was inevitable any more. As tamarin males assist in the care of their youngs and should two youngs be born, raising them successfully might be jeopardized. Another complication was the formation of a bone sequester preventing healing. We have had no experience of hypergranulation in association with the use of natrium hyaluronate.

To sum up, we may say that dressing the wound thoroughly, the choice of an appropriate dressing material, and, with large skin injuries, using systemic antibiotics are very important. The limiting factors include preventing automutilation, follow-up therapy, and good cooperation with the owner.
Acknowledgements
I thank to prof. Zdenek Knotek, DVM, PhD and Roman Vodicka, DVM for their consultations and to the technical staff of the Avian and Exotic Animal Clinic, University of Veterinary and Pharmaceutical Sciences, Brno, for their skillful assistance.

References

Table 1: Weight, rectal temperature and therapeutics use in our therapy

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<td>610 g</td>
<td>37,2 ºC</td>
<td>Fenistil gel® sterile saline 0,1% povidoniodine tinct. nigra</td>
<td>Amox-clavulonate Flunixin-meglumin</td>
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<tr>
<td>2</td>
<td>600 g</td>
<td>37,5 ºC</td>
<td>Fenistil gel® sterile saline 0,1% povidoniodine tinct. nigra</td>
<td>Amox-clavulonate Flunixin-meglumin vit. C</td>
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<tr>
<td>3</td>
<td>600 g</td>
<td>37,6 °C</td>
<td>Fenistil gel® sterile saline 0,1% povidoniodine tinct. nigra</td>
<td>Amox-clavulonate Flunixin-meglumin</td>
<td>Dry to dry – bandage Elizabeth collar</td>
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<tr>
<td>4</td>
<td>600 g</td>
<td>37,4 °C</td>
<td>Fenistil gel® sterile saline 0,1% povidoniodine tinct. nigra</td>
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<tr>
<td>5</td>
<td>590 g</td>
<td>38,5 °C</td>
<td>Fenistil gel® tinct. nigra Hyiodine®</td>
<td>Enrofloxacin T-Sulfa vit. C Haloperidol 1 gtt sid.</td>
<td>Dry to dry – bandage Elizabeth collar size - 4,8 * 3 cm separation from female</td>
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<td>6</td>
<td>600 g</td>
<td>38,9 °C</td>
<td>Fenistil gel® tinct. nigra Hyiodine®</td>
<td>Enrofloxacin T-Sulfa vit. C Haloperidol 1 gtt sid.</td>
<td>Wet to dry – bandage Elizabeth collar sedation</td>
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<td>7</td>
<td>590 g</td>
<td>38,5 °C</td>
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<td>- II -</td>
<td>Wet to dry – bandage Elizabeth collar little sedation</td>
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<td>8 - 14</td>
<td>580 g</td>
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<td>15</td>
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<tr>
<td>16-29</td>
<td>560 - 580 g</td>
<td>37,9 – 38,6 °C</td>
<td>Hyiodine®</td>
<td>-</td>
<td>Wet to dry – bandage Elizabeth collar</td>
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<td>30</td>
<td>580 g</td>
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<td>80</td>
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**Legend to figures:**
- Figure 1: Skin injury – first day
- Figure 2: Autotraumatization – fifth day
- Figure 3: Wound after 21 days – granulation tissue and epithelization, white tissue over the bone sequestr