*rossalleni)* housed with the necropsied snake revealed similar embryonated eggs. Based on the successful use of tetramisole for lung worm infections in snakes[1] the corn snake and Everglades rat snakes were administered intraperitoneally 10 mg/kg body weight levamisole hydochloride.[b] Two weeks following treatment fecal examination for nematode ova was negative. At this time the snakes received a second dose of levamisole and future fecal examinations proved to be negative. Levamisole hydrochloride thus appears to be an effective and safe parasiticide for lungworms in snakes.

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a Mychel-S, Rachelle Inc., Long Beach, California.
b Ripercol-L, American Cyanimid Company, Princeton, N.J.

## DISSEMINATED BACTERIAL INFECTION IN AN INFANT RHINOCEROS

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A brief survey of the literature indicates that disease processes in the rhinoceros are infrequently reported; and to date, no well described cases of enteric infection have been found. A case of probable bacterial infection, leading to the death of an infant rhinoceros, is described in this report.

CLINICAL HISTORY

On 20 January 1975 a female, southern white rhinoceros *(Ceratotherium simum simum)* was born at the San Antonio Zoo. The animal appeared normal until 16 February 1975, when diarrhea was noted. Clinical examination indicated dehydration, weakness, and severe fluid diarrhea. Treatment included oral sulfanomides, neomycin, and belladonna alkaloids[a], intramuscular injections of sodium sulfachlorpyridazine[b] and 4 cc of atropine (1 /120 gr/cc). In addition, sodium bicarbonate and 5% dextrose in lactated Ringer's solution were given intrperitoneally.

The treatment was repeated in 6 hours and the infant returned to its mother for the night, as both animals appeared to be severely stressed by the separation. Since the infant was too weak to nurse, she was again removed and taken to the Health Center for further treatment. Despite continued therapy the animal died shortly after being moved.

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a Sulkamycin-S, Norden Labs.
b Vetisulid - CIBA

NECROPSY RESULTS

The trachea and bronchioles contained reddish-tinged froth. The lungs were mottled purple-red and a considerable amount of blood exuded from cut surfaces. The heart was rounded and the ductus arteriosus appeared patent. The liver was dark red-purple. The intestinal serosa was dark red, and the wall of the gut appeared to be thickened. The mucosa of the small intestine was ulcerated and covered by a yellow-white membrane. The mucosa of the large intestine was reddened and appeared necrotic. Mesenteric lymph nodes were swollen and had pinpoint red foci throughout.

HISTOLOGIC DESCRIPTION

Diffuse pulmonary congestion was seen. In the liver, focal areas of necrosis occurred throughout the parenchyma. These necrotic areas had an inflammatory response consisting primarily of lymphoid and reticuloendothelial cells (Figs. 1 & 2). Moderate sinusoidal congestion was also present.

Severe lymphoid depletion was seen in mesenteric lymph nodes, and numerous macrophages were scattered throughout the nodes. Focal areas of necrosis and neutrophil accumulation were also present. Within the mucosa, lamina propria, and submucosa of the colon there was a diffuse infiltration of reticuloendothelial cells and lymphocytes (Fig. 3). Scattered foci of complete mucosal necrosis were seen (Fig. 4) along with necrotic debris, a pleocellular inflammatory exudate, and numerous small gram negative rod-like bacteria.

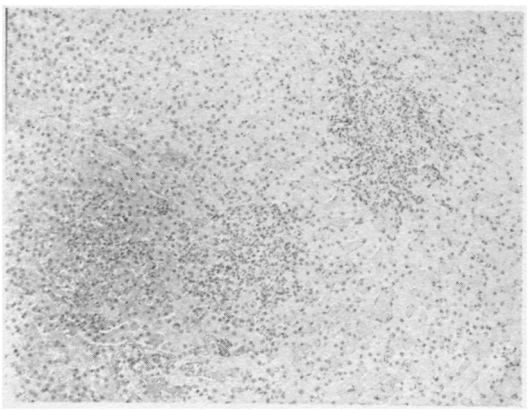

Fig. 1 Foci of reticuloendothelial (RE) cell proliferation in the liver. H&E X108.

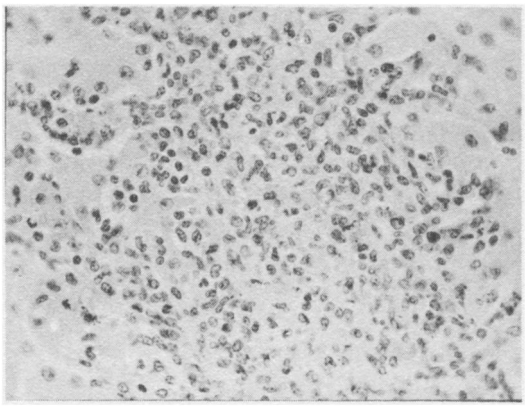

Fig. 2 Higher magnification of an area in Fig. 1. Note the pleomorphic nuclei of the RE cells. H&E X415

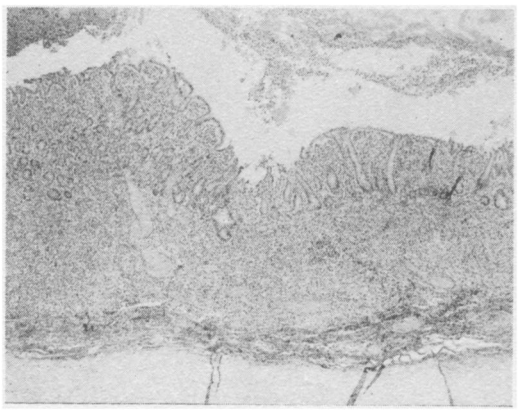

Fig. 3 Area of minimal necrosis and severe inflammatory cell infiltrate in the mucosal lamina propria and submucosa. There is obliteration of much of the muscularis mucosa. H&E X40.

Fig. 4 Area of colon with complete necrosis and severe pleocellular inflammatory infiltrate. H&E X40.

## LABORATORY RESULTS

*Klebsiella* sp. and *Pseudomonas aeruginosa* were cultured from fecal material.

## DISCUSSION

Although the isolation of both *Klebsiella* sp. and *P. aeruginosa* might lead to an immediate assumption that one or the other organism was the etiologic agent, a consideration of the morphologic lesions and the reported disease conditions associated with these organisms complicates the picture.

Smith, Jones, and Hunt[4] state that *Klebsiella pneumonae* establishes itself as a normal enteric organism postnatally, and is not associated with disease. This organism can, however, infect tissues if it gains entrance to the peritoneal cavity or the respiratory tract. *Pseudomonas aeruginosa* is often found in necrotic pneumonias, enteritis, traumatic pericarditis, and other purulent infections[1], and occasionally in septicemic conditions.

The gross and microscopic changes in the young rhinoceros are very similar to those in young horses with salmonellosis[3] - - hemorrhagic inflammation of the large intestine, acute regional lymphadenitis, and foci of Kupffer cells hyperplasia in the liver. Salmonelleae have been reported in young rhinoceroses[5], although not as the cause of fatalities. In addition, a large number of zoo animals have been found to have salmonella[2,5]. Bruner and Gillespie[1] state that outbreaks of salmonellosis are more common in young animals; and, in the young, mortality is greater than in adults.

For these circumstantial reasons, we consider that this young rhinoceros may have had an acute case of salmonellosis. The reason or reasons for failure to isolate salmonella are not apparent; and the isolation of other potential pathogens is considered of secondary importance, in that the disease pattern is not characteristic for these organisms.

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