Inclusion body disease (IBD) is a viral infection that occurs in pythons, boas, and palm vipers. The causative agent is believed to be a retrovirus. The disease has been observed in pythons since the mid 1970s and in boas since the early 1980s. The virus causes a separate set of clinical signs in pythons and boas. The disease is usually more rapid in pythons leading many reptile specialists to believe that boas may act as a carrier for the virus before becoming clinically ill. For this reason, I do not recommend mixing pythons and boas in the same collection. Regardless of the species of snake developing the disease, the outcome is usually terminal once clinical signs develop.

Inclusion body disease can effect the gastrointestinal, respiratory, and nervous systems. Clinical signs in juvenile boas rapidly culminate in flaccid paralysis and death. In adult boas the disease will progress more slowly. Early signs include chronic regurgitation leading to weight loss and pneumonia. Later as the nervous system becomes involved dysecdysis occurs due to the inability to control body movement for proper shedding. Additionally, failure for the snake to right itself when turned over will occur. In the final stages, the boa will become unable to strike, constrict, and swallow food items eventually leading to its death.

In pythons the disease develops more rapidly. Most of the early signs are the same as for boas, except for the lack of regurgitation. Pythons also tend to develop infectious stomatitis in the early stages. As the disease progresses the nervous system will become involved leading to the loss of the righting reflex, hyperreflexia (exaggerated reflex motion), disorientation, loss of motor coordination, and eventually death (see photos).

Tissue samples from snakes affected with inclusion body disease will usually show specific histological changes. Intracytoplasmic eosinophilic inclusions of various sizes are commonly found in the epithelial cells of the kidney, pancreas, and liver. They also are found within the neurons of the brain and spinal cord. It has recently been determined that the inclusions are comprised of antigenically distinct 68-kd protein band. Although the presence of these inclusions will be diagnostic of this disease, their absence does not mean the snake is free of the disease.

The inclusions of inclusion body disease closely resemble the byproduct of cell metabolism because the cells are not degenerative, there is an overall lack of any inflammatory response, and the inclusions do not contain any infectious particles. New theories suggest that inclusion body disease represents an infectious disease that represents itself morphologically as a defect in cell metabolism resulting in an accumulation of protein byproduct. Additionally, this alteration in cell metabolism ultimately results in the various disease states recognized in infected reptiles.

The exact route or routes of transmission have not been established. Because the causative agent is a virus there are currently theories regarding the transmission of the disease. These include: (1) Direct contact with infected snakes. (2) Contact with contaminated secretions, either in an aerosolized form or by the owners hands from improper hygiene while handling the snakes or cleaning their enclosures. (3) Intrauterine transmission to embryos or eggs. (4) Venereal transmission. Additionally, the snake mite, Ophionyssus natricis, has been suspected as a vector because it is frequently found in contaminated snake collections.

Currently, there is no in-house diagnostic test available for live snakes although several options are being studied. One option utilizes a complete blood count (CBC) with a differential
white blood cell count, and plasma biochemical analysis. Acutely affected snakes (less than 2 months following the onset of clinical signs), showed leukocytosis (increase in circulating white blood cells), lymphocytosis (increase in circulating lymphocytes), decreased total protein and globulin levels, and a significantly elevated SGOT (a liver enzyme) than normal snakes. The University of Florida College of Veterinary Medicine is currently working to develop an immunofluorescence assay test to help diagnose the disease. Additionally, the University of Florida Protein Sequencing Core Laboratory is working on determining the amino acid sequence of the 68-kd protein associated with the inclusion material. Occasionally intracytoplasmic inclusions will be seen in lymphocytes on a peripheral blood smear. Regardless of the results of blood testing, to determine the actual presence or absence of inclusion bodies will require biopsies of esophageal, gastric, and liver tissue.

There currently is no treatment available for the disease. Because it is always fatal and very contagious, euthanasia is strongly recommended. Although the snake may be kept “alive” with hydration and force feeding, its quality of life will be constantly decreasing while the pain of the disease will steadily increase.

The current control and possible prevention of inclusion body disease involves several recommendations. All new boas or pythons should be quarantined for a minimum of 6 months before introduction to an established collection. Mite control and strict hygiene measures must always be followed. Take precautions when visiting pet stores, expos, and other snake collections. All infected snakes should be immediately euthanized to prevent the spread of the disease. Glass cages and wooden cages sealed with polyurethane should be thoroughly cleaned with a 5% bleach solution and placed in the sun for several days before being used for other snakes. Unsealed wooden cages and other enclosures not available for thorough cleaning and sun exposure should be discarded.

References: