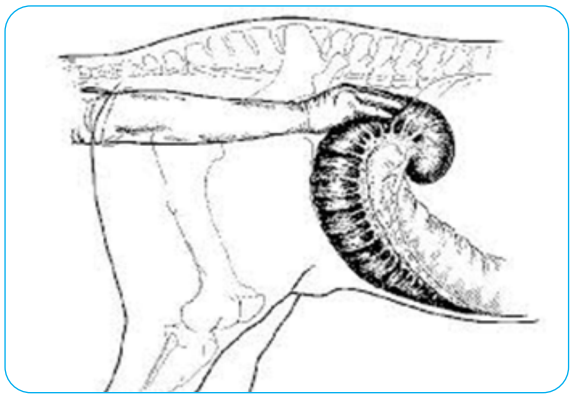
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Rectal Palpation in Cattle



Black tarry faeces due to long stagnation in ileus



Black tarry sticky foul smelling faeces in peritonitis

In this issue

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- DISTINGUISHED WOMEN VETERINARIAN

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EMPTY RECTUM IN BUIATRICS: AN ESTABLISHMENT OF POINT OF REFERENCE IN CLINICAL DIAGNOSIS

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Empty rectum in buiatrics is a term used to indicate the failure to pass faeces for 24 hours or more; Empty rectum is also used to denote scanty faeces. In most of the ruminant gastrointestinal dysfunctions, defaecation is either absent or reduced in amount with abnormal composition and are mostly pasty, foul smelling and appear overdigested because of increased transit time in alimentary tract. The relative absence of faeces is noticed in diseases causing disturbance of fore stomach disorders, abomasal and intestinal motility. Empty rectum in cattle is either due to functional obstruction or physical obstruction. Usually, complete absence of faeces is related to physical obstruction, while marked reduction is seen in case of functional obstruction, which is a major source of diagnostic tool. This article briefs about important diseases causing empty rectum in buiatrics under field conditions.

ETIOLOGY

I. PHYSICAL OBSTRUCTION

Complete absence of faeces until obstruction is relieved

- **Intraluminal obstruction (Obstruction of lumen only)**
  - Phytobezoars
  - Trichobezoars
  - Foreign bodies
  - Parasitic infestations
- **Strangulation obstruction (vascular supply involved)**
  - Intussusceptions
  - Left displacement of abomasum
  - Right displacement of abomasum and volvulus
  - Volvulus of intestine
  - Caecal dilatation and dislocation
  - Gut tie
- **Extraluminal obstruction**
  - Stenosis (traumatic duodenitis)
  - Neoplasm
  - Abscess
  - Adhesion
  - Fibrous bands
  - Hernia

II. FUNCTIONAL OBSTRUCTION

A dark and scanty faeces for 24 hours or until motility is restored. Typically faeces never had blood.

Fore stomach

- Traumatic reticulo peritonitis with reticular abscess
- Vagal indigestion
- Omasal impaction
- Abomasal impaction
- Abomasal ulcer
- Duodenal obstruction
- Paralytic ileus
- Acute diffuse peritonitis

III. MISCELLANEOUS

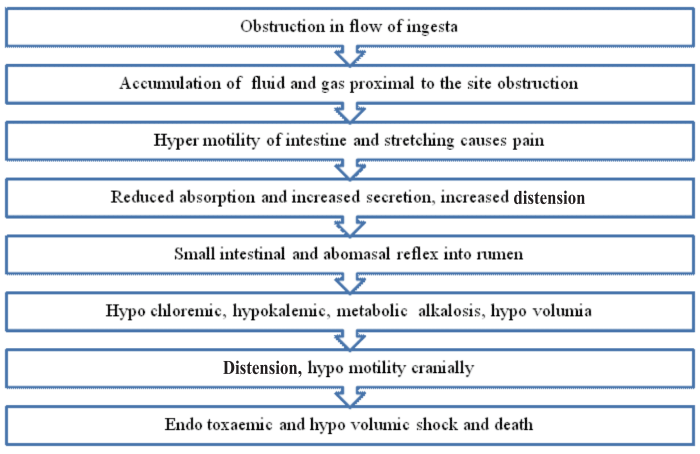
- Hypocalcemia
- Fatty liver

IV. CONGENITAL DEFECTS

- Malformation of intestine eg. atresia coli
- Atresia of large intestine

PATHOGENESIS

The general pattern of change following obstruction appears to be similar, but intensity varies with species, location and duration of obstruction.



I. PHYSICAL OBSTRUCTION

In this there is occlusion of intestinal lumen resulting in functional disturbance. In all cases of physical obstruction, common clinical signs noticed are as follows

- Abdominal pain (treading of hindlegs, stretching, kicking at abdomen, teeth grinding)
- Scant or absence of faeces
- Faeces may be blood stained
- Ruminal stasis with or without distension (according to location of obstruction)
- Abdominal distension at latter stage
- Progressive dehydration, alkalosis or acidosis (according to location of obstruction) and toxaemia leading to shock and recumbency
- Distended loop of intestine may be palpable per rectum
- Mostly treatment is surgical correction

A. LUMINAL BLOCKAGES / INTRALUMINAL OBSTRUCTION

➢ Phytobezoar / Fibre Ball

- They are undigested materials eg. nylon fibers, cotton fabric etc.,
- Sudden onset of abdominal pain, restlessness, kicking at the abdomen, lying down and getting up frequently, arching the back, anorexia, ruminal stasis, decreased faecal production, lethargy, apparent depression and increased heart rate upto 120/minute are the signs
- Distended loops of intestine and fibrous ball with hard lumps may be palpable rectally
- **Faeces will be pelleted initially or grey or yellow and foul smelling faeces or only mucus and after 3-4 days there will be absence of faeces**
- Crumbly masses may pass after mineral oil administration for several days
- Dense fibre balls require surgical treatment

➢ Trichobezoar or Hair Balls

- Similar to Phytobezoar except common in young animals because of frequent ingestion of hair
- They are lodged in small intestine or spiral colon
- Clinical signs involve the complete absence of faeces, abdominal pain, anorexia, mild abdominal distension, dehydration, fluid splashing sound in right abdomen with normal peritoneal fluid.
- Surgical removal is indicated

➢ Foreign Body obstruction

- Mostly occurs at ileum or proximal to ileo-cecal junction
- Alkalosis occurs due to small intestinal and abomasal reflux into the rumen
- Dilated loops of small intestine are usually palpable at cranial to the obstruction on rectal examination
- **Complete absence of faeces will be there**
- Surgical removal is indicated

➢ Parasitic obstruction

- Common in young calves mostly because of ascarid worms
- Naso-gastric reflux will be there in some calves

B. STRANGULATION OBSTRUCTION

➢ Intussusception

- It is the invagination of one portion of the intestine into lumen of an adjacent segment of intestine
- Mostly due to intramural or luminal mass or lesions, viral enteritis, intestinal parasitism, abrupt dietary changes, foreign bodies and/or administration of intestinal motility modifier drugs
- In cattle, most commonly occurs in jejunum but frequent in colon and ileocecal junctions.
- Intermittent colic exhibited as restlessness, kicking at the abdomen, lying down and getting up frequently, assuming abnormal posture for first 24 hours after the onset of disease
- After the initial signs, abdominal pain subsides and affected cattle become progressively lethargic and recumbent and show apparent depression.
- Faecal production may be normal for up to 12 hours after the occurrence of the intussusception but minimal after 24 hours of duration. Passage of blood and mucus from the rectum is common at this time. **Blood is often present, not as melena but as altered red blood, in the form of a thick red slurry, leaving dried flakes of it around the anus**.
- In some cases they will be **hard lumps, usually covered with mucus**.
- **Latter it turns in to melena** because of devitalized and sloughed intestine which ultimately leads to peritonitis, bowel rupture, toxic shock and finally death

- Distension of the intestine with fluid and gas proximal to the obstruction leads to abdominal pain, progressive dehydration and electrolyte depletion
- In rectal examination only 20% of the intussusceptions can be palpable
- Death usually occurs 5 to 8 days after the onset of clinical signs

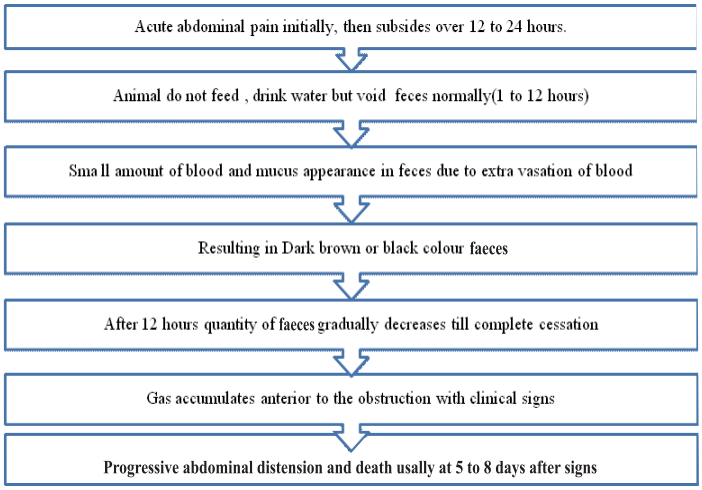




Fig 1. Foamy mucus on the rectum in intussusceptions



Fig 2. Presence of mucus in rectum



Fig 3. Black tarry mucus coated dung in intussusception



Fig 4. Intussusceptions mass during surgery

➢ Left and right displacement of abomasum

- The risk period is the transition period i.e two weeks prepartum and two to four weeks postpartum.
- It is manifested as inappetance, complete anorexia, reduced consumption of concentrates, or alternating periods of normal appetite and anorexia, ketosis, reticulo ruminal movements not clearly audible or absent
- Left displacement of abomasum (LDA) increases abomasal luminal pressure leads to perforation, diffuse peritonitis and death. Abdominal pain and pneumoperitoneum are other common sequelae
- **Faeces are often normal to softer than normal but reduced in volume**
- Surgical intervention is necessary.

➢ Right displacement of abomasum and volvulus

- Right displacement occurs at approximately 10% to 15% the frequency of LDA
- Distended abomasum may twist in a clockwise direction and cause acute obstruction with local circulatory impairment and ischemic necrosis of the abomasum

- Anorexia, absence of rumination, scant and abnormal faeces, right abdominal distension and fluid splashing sound on allotment of right para lumbar fossa.
- The faeces are usually **scant, soft and dark in color, and become blood stained or melenic in the ensuing 48 hours,** if the cow lives long enough. The soft feces must not be mistaken for diarrhea
- Rupture of the abomasum may occur in abomasal volvulus cases and cause sudden death

➢ Intestinal Volvulus

- It is the axial rotation of mesentry and attached small intestine over its own axis.
- Torsion of small intestine is rare in cattle
- Volvulus of the root of mesentry or midjejunum to distal jejunum and proximal ileum occurs where the mesentery is long. This long mesentery and associated bowel may rotate about its own axis without involving the remaining small intestine.
- Volvulus results in intestinal distension, vascular compromise, intestinal necrosis and eventually death, unless surgically corrected.
- Acute and severe abdominal pain demonstrated by kicking at the abdomen, rolling, lying down and getting up frequently and grunting will be noticed continuously even after 10 days of onset.
- On rectal examination, intestinal loops may be small, soft, mobile with possibility to follow tightly stretched mesenteric bands crossing doors-ventrally in the middle part of abdomen.
- **Empty rectum felt with tarry faeces and exudates in the rectum**

➢ Caecal Dilatation and Dislocation

- Caecal dilatation and volvulus are common in dairy cattle where as cecal torsion is not often
- Caecum gets distended, displaced and in some cases, retroflexion or torsion of the cecum and proximal portion of the colon occurs
- It leads to partial or complete obstruction of the passage of digesta
- Inappetance, acute onset of mild colic, reduced rumen motility, **decreased to absent faecal output,** distention of the right flank with mild abdominal pain are the typical signs
- Grossly distended intestinal loop extending horizontally across the abdomen just cranial to the pelvis and caudally or medially to the rumen.
- 95% of cases can be diagnosed by rectal examination
- Blind end of caecum may be palpated near the pelvic inlet as round, dome shaped structure.
- In later stage, ping sound can be produced by simultaneous percussion and auscultation in right flank which is distended.

➢ GUT TIE

- It occurs in recently castrated cattle.
- When spermatic cord is pulled and broken during castration, it may recoil throw the inguinal ring and become entangled around small intestine causing a physical obstruction.

C. EXTRALUMINAL OBSTRUCTION

➢ Compression Stenosis

- This may arise from blood clot which compresses the intestinal lumen and cause physical obstruction.

➢ Neoplasm

- Neoplasms that pressures the intestine results in obstruction of intestine thereby reduces or stops the flow of ingesta and so causes empty rectum. e.g: fibropapilloma of cardia

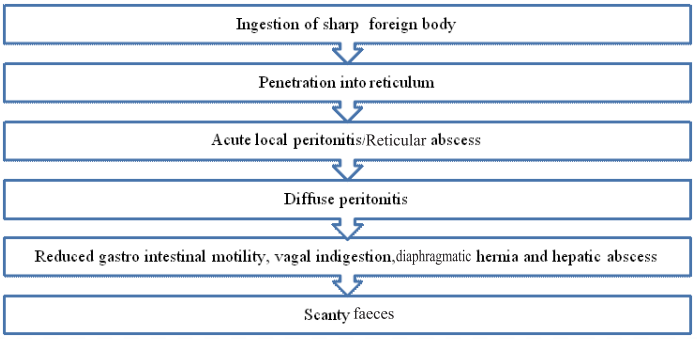
➢ Fibrous Bands

- Fibrous adhesions causes reduced gastrointestinal motility and thereby causes **scanty faeces or empty rectum**

II. FUNCTIONAL OBSTRUCTION

➢ FORESTOMACH

1. Traumatic reticulo peritonitis with Reticular abscess



- It is manifested by sudden onset of anorexia and fall in milk yield, sub acute abdominal pain , characterised by reluctance to move especially downhill
- Remain standing for long period and lie down with great care
- Habitual recumbency is characteristic in some animals
- Defecation and urination causes severe pain resulting in **constipation and scant faeces**
- **Faeces are dry, firm and covered by thin coating of mucus because of prolonged retention**
- Signs of reticular abscesses are poor body condition, a relatively full rumen but with reduced ruminal contractions or almost complete ruminal atony, persistent mild bloat and undigested particles in the faeces. Most have a clinical history of not responding to prolonged therapy with antimicrobials.
- Positive venous pulsation and brisket enema will be evident
- Rectal examination reveals smaller rumen with atony and fibrinous adhesions over rumen
- Anti microbial therapy for several days with reticular magnet and immobilisation to promote adhesion is one way of medical management in some cases
- Rumenotomy to remove foreign body, if medical treatment fails

2. Vagal indigestion

- Failure of omasal transfer/abomasal/pharyngeal trauma/reticular adhesion/pyloric stenosis/vagal nerve injury due to traumatic reticulopertonitis (TRP)
- Vagus nerve injury and reticular adhesions from traumatic reticuloperitonitis leads to failure of passage of ingesta from the reticulorumen or abomasum resulting in accumulation of fluid in the forestomach and abomasum
- Dorsal vagal nerve injury results in inhibited passage of ingesta from the reticulorumen into the omasum and abomasum, resulting in an enlarged rumen
- Injury of the pyloric branch of the ventral vagus nerve results in inhibited the flow of ingesta from the abomasums resulting in abomasal impaction
- Perireticular abscesses near the reticuloomasal orifice of cattle can cause the disease
- Inappetence for several days or complete anorexia, moderate to severe bloat, **rumen contents are pasty and frothy,**
- Disturbance in passage of ingesta due to reduced motility.
- **Feces are scant and pasty and contain undigested particles in the initial periods and latter it become scant amounts of soft pasty faeces**
- Some cases respond to fluid and balanced electrolyte therapy for 3 days combined with the oral administration of mineral oil (5–10 L) daily; if not rumenotomy can be tried.

➢ OMASAL IMPACTION

- Omasal impaction, omasal canal obstruction and omasal erosions are the common diseases of omasum
- Omasal canal obstructions usually result from ingestion of baling twine or plastic papers, polyethene, other coarse indigestible fibrous materials (particularly from seeds), TRP, reticular and extra reticular abscesses.
- It is also caused by the accumulation of soil in the omasum
- Further omasal impaction is due to localized peritonitis and adhesion between reticulum and diaphragm anterior to the omasum are most common cause of failure
- The disorder is characterized by chronic recurrent bouts of indigestion, decreased rumen motility, anorexia, an extremely firm and enlarged omasum that may be painful on palpation, **infrequent and scanty faeces** and clinical improvement following softening of the omasum.
- Four liters of mineral oil should be administered intraruminally for 3-5 days postoperatively to facilitate softening

➢ ABOMASUM

➢ Abomasal impaction

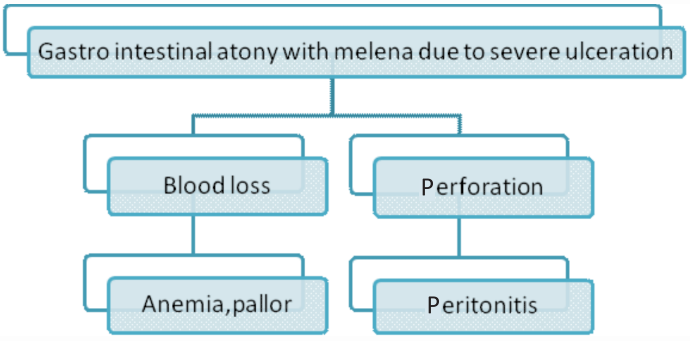
- It may be dietary or secondary to vagal nerve injury
- It is result of feeding poor-quality, coarse roughage as the sole feeding
- Impaction with sand have occurred in up to 10% of cattle with abomasal impaction.
- Stand mixed into silage while it is being packed
- Abomasal distension may occur because of vagal nerve injury with normal diets after correction of abomasal

volvulus, secondary to traumatic reticuloperitonitis, or secondary to the development of adhesions between the abomasum and the rumen and/or the abdomen

- The distended abomasum is filled with a firm mass of fibrous ingesta
- Indigestible material creates a mechanical outflow obstruction
- Reduced feed intake and a reduced volume of firmer than normal feces are the initial signs. Ruminal contractions are of normal or increased frequency initially and latter motility often is absent
- The consistency of the **ruminal ingesta may be more fluid**
- **Scant faeces with mucus may cling to the clinician's gloves after rectal examination**
- Medical management includes correction of fluid and electrolyte abnormalities by aggressive fluid therapy, easily digestible feeds, and oral administration of laxatives such as mineral oil (4 L daily). Prokinetic drugs like erythromycin (10 mg/kg bid), bethanechol (0.07 mg/kg SC tid) and metoclopramide can be tried
- Severely affected cattle will die in 3 to 6 days after the onset of signs. Rupture of the abomasum occurs in some cases, and death is imminent in few hours

Abomasal ulcer

- Abomasal ulcers are divided in to four types – 1) Non bleeding non perforating ulcers 2) Major bleeding with perforating ulcers 3) Perforating with local peritonitis 4) Perforating with diffuse peritonitis
- Microorganisms including *Clostridium perfringens* type A or D, *Escherichia coli,* Sarcina-like spp., Campylobacter, *Streptococci,* and fungi, abrasion of the abomasal mucosa by roughage, geo sediments, or trichobezoars have been associated with abomasal ulceration in ruminants.
- History of recent parturition , stress, use of anti inflammatory drugs and mineral deficiency (mainly copper) also play a role.
- Type 1 signs are mild abdominal pain, partial anorexia, decreased ruminal motility, and mild ruminal tympany. There is usually no febrile response. **Manure may be normal or reduced in amount.** Traumatic reticuloperitonitis or indigestion may be suspected with these signs.
- Type 2 signs are anemia and hemorrhagic shock. These animals have **dark blood clots in their manure or tarry, black faeces with the characteristic smell of partly digested blood.** The mucous membranes may be pale. The rumen may have a fluid consistency, and if the animal is able to stand, abdominal pain sometimes is evident.
- Type 3 – Localized peritonitis. This condition is most like TRP in presenting signs. The animal may be moderately febrile and partly or totally anorectic with abdominal pain, usually localized to the right ventral quadrant
- Type 4 - Major leakage leads to acute diffuse peritonitis. The course of the disease usually is rapid, with signs of septic shock with total anorexia and ruminal stasis developing within 24 hours of the onset.



INTESTINE

Duodenal obstruction

- Duodenal outflow problems occur as a result of obstruction or dysfunction
- Duodenal obstruction occurs as a result of trichobezoars, foreign bodies, duodenal stricture following ulcer, obstruction by displacement of viscera (e.g., gallbladder, uterus) and extraluminal compression caused by liver abscess due to TRP, omental abscess, or lymphosarcoma
- Dysfunction is due to peracute duodenitis, duodenal ulcers with or without perforation, clostridial duodenitis and electrolyte abnormalities
- Affected animals initially have abdominal pain and then severe bloat caused by fluid distension of all forestomachs, acute collapse and dehydration,

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ANIMAL HEALTH

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decreased appetite, weight loss, **decreased faecal production,** lethargy, and apparent depression and finally death

- Surgical therapy is recommended

➢ Paralytic ileus

- Diagnosed with history of recent calving or change in diet
- Occurs due to acute peritonitis or over handling of tissues during surgery, prolonged and severe distension of intestines as in intestinal obstruction or enteritis resulting in functional obstruction
- It is further caused by acid-base imbalance, dehydration, electrolyte imbalances such as hypocalcemia and hypokalemia, and toxemia.
- Accumulation of fluid and gas results in varying degrees of abdominal pain, dehydration and marked reduction in amount of faeces.
- Distended intestines, caecum and colon are palpated in the rectal examination
- Compression of intestines during examination easily flattens the affected bowel
- **Rectum is empty but it may contain sticky mucus without any blood**
- Administration of fluids, calcium and NSAIDs will help to manage these patients
- Xylazine is contraindicated because of its depressant effect on gastric and intestinal motility

➢ Acute Diffuse Peritonitis

- It is one of the main reason for functional obstruction of the intestine
- Because of paralytic ileus a complete absence of defecation, often with no faeces
- The main etiologies are TRP, abomasal ulcer, rumenitis, intussusceptions, caecal torsion and volvulus and spread from other sites like spleen, liver and umbilical vessels
- The transit time of ingesta through the alimentary tract is increased
- In the early stages there may be a transient period of increased frequency of passage of small volumes of soft faeces, latter faeces may be completely absent for periods of up to 3 days
- **Faeces are characteristically scant, dark, and like small faecal balls accompanied by thick, jelly-like mucus. The faeces may alternatively have a thick, sludge-like consistency, be tenacious and difficult to remove from glove, and have a foul smell**



Fig 5. Black tarry sticky foul smelling faeces in peritonitis

IV. MISCELLANEOUS

➢ Hypocalcemia

- Anorexia, rumen hypotonic or atonic , scant or absence of faeces for 12-24 hours, temperature normal or subnormal, heart rate increased, possibly arrhythmia.
- Good response to calcium therapy

➢ Fatty liver

- History of recent calving or abdominal displacement with ketonuria and ketonemia.
- Poor response to treatment.
- Glucose, insulin, anabolic steroids can be administered

V. CONGENITAL DEFECTS

➢ Atresia Coli

- Absence of faeces since birth, abdominal distension, dehydration and progressive depression are the most common clinical signs.
- Affected animal develops abdominal pain in 12-24 hours after colostrum consumption with gradual bilateral abdominal distension.

➢ Atresia of Large Intestine

- Complete occlusion of the intestinal lumen due to abnormal development of the intestinal wall
- Common in calves and develops in colon

DISTINGUISHED WOMEN VETERINARIAN Dr. JANICE MILLER



Today there are more women students in many veterinary colleges than men. In the 1960s, however, there were not a lot of women in veterinary colleges and many colleges did not even accept women not only in India but even in developed countries like USA. Dr.Janice Miller is a pioneer women veterinarian who took up veterinary science as her career in the 1960s. She was the only girl student when she joined the Veterinary College of Kansas State University, USA.

She completed her bachelor's, DVM and Master's degrees from Kansas State University in 1960, 1962 and 1963, respectively. She did her Ph.D. at the University of Wisconsin and completed in 1969.

While in graduate school, Janice Miller found the virus that causes bovine leukemia, and that discovery set the tone for her whole veterinary career.

Dr. Miller has been involved with biomedical research for over 35 years. During her career as a federal research scientist, she focused on several problems of importance to the livestock industry, especially diseases associated with retroviruses, herpesviruses, mycobacteria, and prions. In addition to addressing the etiology and pathogenesis of infections with these agents, Dr. Miller developed diagnostic tests to detect them.

During her career, Dr. Miller has authored or co-authored a total of 134 research or professional publications and presented over 90 invited professional presentations to colleagues and fellow scientists.

She has been the recipient of several professional awards and recognitions including the ARS Distinguished Scientist in 1988, the USDA Distinguished Service Award in 1990, and the American Feed Industry Award from the AVMA in 1999. Dr. Miller was elected to the National Academy of Science in 1999 and is known nationally and internationally for her discovery of the bovine leukemia virus, an accomplishment which was featured on the cover of Cancer Research in February of 1984.

She was awarded with the first AVMA Lifetime Excellence in Research Award for her achievement in veterinary medical research in 2006. The award recognized her achievement in veterinary medical research. There is no doubt that Dr. Miller is a role model veterinarian for all women veterinarians.

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