

# JEJUNAL HEMORRHAGE SYNDROME OF DAIRY CATTLE

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## Introduction

In the last 3 years veterinary practitioners from Iowa, Minnesota and Wisconsin have reported a peracute, segmental hemorrhagic enteritis in mature dairy cattle with increased frequency.

*Clostridium perfringens* is the most important cause of Clostridial enteric disease in domestic animals and is divided into 5 phenotypes (types A, B, C, D and E) based on the production of 4 major toxins: alpha, beta, epsilon and iota ( $\alpha$ ,  $\beta$ ,  $\epsilon$ , and  $\iota$ ). *Clostridium perfringens* type A produces  $\alpha$  toxin; type B produce  $\alpha$ ,  $\beta$ , and  $\epsilon$  toxins; type C produce  $\alpha$  and  $\beta$  toxins, type D produce  $\alpha$  and  $\epsilon$  toxins, and type E strains produce  $\alpha$  and  $\iota$  toxins. *Clostridium perfringens* type A is the most common *C. perfringens* type, the most variable in its toxigenic properties and the most confusing organism with respect to its potential pathology. Much of this confusion is due to the ability to isolate this organism from the tissues, effusions and intestinal tract of cadavers within hours of death. It grows rapidly on culture and mask the presence of other organisms.

*Clostridium perfringens* type A is implicated as the cause of enterotoxemia in lambs, tympany, hemorrhagic enteritis, abomasitis and abomasal ulceration in calves in the western United States, necrotic enteritis in domestic chickens, necrotizing enterocolitis and villous atrophy in suckling and feeder pigs, hemorrhagic gastroenteritis in dogs, and necrotic enteritis of foals.

Recent isolations of *Clostridium perfringens* type A strains from Jejunal Hemorrhage Syndrome cases (JHS) have demonstrated a variant that expresses Beta2 toxin. Of 47 cases in adult dairy cattle 25 isolates yielded the Beta2 toxin. Beta2 toxin was first found in association with type A *Clostridium perfringens* in necrotic enteritis of swine and enterocolitis of horses in Europe, and this particular toxin is known to create inflammation of the small intestine with the loss of mucosa.

Based on reports from NE Iowa, SE Minnesota and SW Wisconsin, as well as across the nation during 1999- 2001, many veterinarians and herd owners have begun to suspect that JHS is a new emerging disease syndrome. Presentation of this syndrome has been sporadic, affecting only individual dairies and individual mature cows within the dairy. Based on practitioner reports, a morbidity of 1-2% of the mature cow population would be typical with mortality of affected animals approaching 85-100% due to the peracute nature and severity of this disease. However, individual herds have experiences individual and multiple outbreak episodes.

## Diagnostics / Therapy

**Clinical Signs:** Based on cases attended by the authors and other veterinarians, the clinical signs of JHS are peracute. Frequently the producer will see no prodromal signs and find the mature cow dead. Sometimes an individual may be found down and in systemic collapse. In early onset, animals are restless, painful, and may kick at their side. Other clinical signs include:

- Sternal recumbency
- Vocalization
- Diaphoresis

- Bruxism (teeth grinding)
- Enophthalmia (sunken eyes)
- Shock, as evidenced by pale mucous membranes and poor capillary refill time.
- If the cow is standing, ballotment of the lower right abdomen can elicit a pronounced fluid slosh due to the backup of ingesta and fluid behind the occlusive lesion.
- Rectal examination may indicate signs of constipation, followed by evidence of melena or frank hemorrhages and clots within the rectal vault. Dilated intestinal loops may also be palpable.

**Rule-Outs:** The most frequent rule-outs for the indicated signs are Salmonellosis (*Salmonella kentucky*), abomasal ulceration and hemorrhage, and abomasal displacement, volvulus, and compromise. Rule-outs for incidence of sudden death include intestinal volvulus or intussusception, acute peritonitis, traumatic pericarditis, or abomasal volvulus. In general, *Salmonella* cases have a slightly longer survival “window” than do the peracute cases of JHS. The veterinary practitioner is encouraged to obtain a fecal sample to culture for the presence of *Salmonella* sp. prior to making a diagnosis of JHS. Due to the ubiquitous nature of *Clostridium perfringens* type A, fecal culture for this species should not be considered diagnostic. While a causal link has not been established between the presence of *Clostridium perfringens* type A and JHS a practitioner wishing to perform cultures should focus attention on sampling of the lesion site keeping in mind that overgrowth can occur early following death.

**Clinical chemistry:** Due to the peracute nature, veterinarians have little time to perform clinical chemistry analysis. A condition that could mimic JHS would be an intussusception. With this one could expect hemoconcentration with increased packed cell volume and total protein. CBC could indicate a neutropenia with strong left shift. Serum chemistry could indicate hypocalcemia, hypochloremia, metabolic alkalosis, hyponatremia, hypokalemia and hyperglycemia.

**Post-mortem findings:** Observations have demonstrated segmental lesions localized to the jejunum. These areas consist of frank hemorrhage and immediate clotting forming a functional occlusion of the lumen of the small intestine. Necrosis of the lumen may or may not be apparent. Some cases have also presented intussusceptions immediately anterior to the area of segmental hemorrhage and clotting. It is unclear which lesion presents first, or whether one contributed to the other. It is possible that the presence of an intussusception could indicate intestinal hypo- or hyper-motility resulting in the slowing of ingesta flow allowing Clostridial growth and sporulation.

**Bacterial isolations:** Isolations from suspect JHS cases presented to the Iowa State Diagnostic Laboratory have been consistent in the recovery of *Clostridium perfringens* type A in high numbers. Impression smears of the lesion sites have also yielded high numbers of gram-positive rods indicative of the presence of a Clostridial species. The isolation patterns found in these samples would be suggestive that this organism should be considered for further research as a possible pathogen in this syndrome. However, this organism is ubiquitous and can be found as a part of the gut flora of warm-blooded animals. Overgrowth following death can be rapid and may mask a potential pathogen. Additional cultures have yielded *Salmonella* species from the lesion sites in variable typings. No consistency in *Salmonella* sp. has been seen from case to

case. The presence of segmental occlusive hemorrhagic lesions may allow *Salmonella* species an opportunistic environment. Fluorescent antibody work examining the presence of BVD virus in the tissue samples has been negative to date.

**Treatment Efforts:** Due to its peracute nature, this syndrome should be considered a medical emergency. Cattle found alive with this syndrome are extremely compromised. Even considering this compromise the use of intravenous calcium has been beneficial. Additional therapy has included flunixin meglumine (Banamine<sup>®</sup>) (1.1 mg/kg IV) or isoflupredone (Predef 2X<sup>®</sup>) (20 mg IM, 1100 – 1500 lb bw) for control of pain and shock along with IV fluid therapy. Some practitioners have attempted to flush the clot from the intestinal mass using oral fluid therapy or oral administration of mineral oil, along with parenteral and oral antibiotics. Results have been variable with some individual cows expelling significant amounts of blood clots and surviving. Affected cattle frequently remain compromised and Salmonellosis may complicate the animal's recovery.

Affected cattle are extremely poor candidates for surgical intervention. They are generally so compromised that they may not even survive transportation efforts. Surgical correction has included intestinal resection and anastomosis, or alternatively, manual massage of the affected area to break down the offending clot. The post-operative prognosis should be considered extremely guarded due to metabolic compromise and the potential reoccurrence of the lesions post-surgery.

### **Field Investigation**

**History:** The Veterinary Diagnostic and Production Animal Medicine Department of the Iowa State College of Veterinary Medicine received a call from a NE Iowa veterinarian in April 1999 to investigate recurring sporadic peracute death losses in a dairy. Previous efforts centering on treatment, autogenous vaccine production and ration manipulation to halt the presentation of this syndrome had been unrewarding. Production statistics for this 140-cow Brown Swiss herd were: 21,824 lbs of milk rolling herd average, 23,488 lbs of milk 305 Day ME, 65.9 lbs of average milk lbs per milking cow, 78 lbs of management level milk, 4.24% test day fat and 3.57% test day protein.

The purebred Brown Swiss herd had reported 1-2 undiagnosed sporadic deaths per year for the last 20 years. In the 2 years preceding the investigation the incidence-rate of sudden deaths greatly increased with the owner and veterinarian reporting 30 deaths due to enteritis. The case rate increase was coincidental with expansion efforts in which the herd doubled in size from 70 to 140 head. Cow numbers were increased from within the herd and no outside cattle were purchased. A freestall barn was built to house the expansion and the tie-stall barn was converted to a parlor. TMR feeding was also adopted at this time. All animals were administered a Clostridial 7-way bacterin/toxoid (Fortress 7, Pfizer) at 10-12 months of age, with a repeat vaccination annually prior to freshening. Since the outbreak of deaths an autogenous *Clostridium perfringens* type A bacterin/toxoid was administered to each lactating individual every 60 days, and the Clostridial 7-way bacterin/toxoid was administered on a quarterly basis.

**Clinical Findings:** Prior to the farm visit the producer was requested to submit samples of the lactational TMR for Penn State shake box testing and wet chemistry analysis, realizing that a

truly representative TMR sample might be impossible to obtain. The individual components used in the TMR were also submitted. Producer was performing monthly DHI herd testing (AgSource, Verona WI). Current and previous records were obtained from AgSource. Following the pre-visit evaluation, an on-site field investigation with the herd's owners, veterinarian, nutritionists, and dairy cooperative field man was arranged.

The diet was typical of rations fed in northeast Iowa and was fed as a single-group TMR. Dry matter intake was 53 lbs/head/day when calculated across all lactations and days in milk. Table 1 presents the daily amounts of ration components presented to the herd in a TMR on an "As-Fed" basis. Table 2 displays the ration parameters as determined by computer calculation and wet chemistry analysis.

**Table 1: Daily amounts of components presented in TMR to the herd (as fed)**

<b>Feed</b>	<b>As Fed Basis lb/head/day</b>
2nd Cutting Alfalfa Haylage	18
Corn Silage	30
2nd Cutting Alfalfa Hay	6.5
High Moisture Corn	12.8
Whole Cottonseed	5.5
Linseed Meal	1.8
Wet Corn Gluten	18
Roasted Soybeans	2
Mineral Mix	1.9
Water	4
<b>Total</b>	<b>100.5</b>

**Table 2: Ration parameters as determined by computer calculation and wet chemistry analysis**

<b>Ration Determinations</b>	<b>Calculated Dry Matter Basis</b>	<b>Wet Chemistry Dry Matter Basis</b>
Moisture	49.20%	45.30%
Dry Matter	50.80%	54.70%
Crude Protein	17.18%	17.85%
ADF	20.19%	18.43%
NDF	35.95%	34.29%
Calcium	0.82%	1.08%
Phosphorous	0.53%	0.66%
Magnesium	0.28%	0.35%
Potassium	1.22%	1.56%
Ash	7.97%	7.53%

Fat	4.64%	4.91%
Protein Solubility		33.55%
TDN	74.26%	74.55%
NFC	34.58%	35.42%
NEL Mcal/cwt	77.00	77.47
NEG Mcal/cwt	53.00	52.37
NEM Mcal/cwt	81.00	80.66

Physical form of the ration was evaluated using the Penn-State shaker box. Shake test results are shown in Table 3. All TMR shake analysis determinations were performed in 3 replicates to ensure consistency. Of particular concern was the TMR 24 hr. refusals. The increase percentage of long fiber length particles was indicative that cows were preferentially consuming the high caloric, small particulate matter instead of the long fiber fraction.

**Table 3: TMR Shake Test results**

	Avg NE Iowa TMR	Subject Herd TMR	Subject Herd TMR Refusals
<b>Long Fiber Length</b>	8.70%	11.10%	23.40%
<b>Medium Fiber Length</b>	34.50%	36.80%	35.30%
<b>Short Particles</b>	56.90%	52.10%	41.30%

Samples of each individual feedstuff used in the TMR were visually evaluated to determine the producer's level of feedstuff management. All feed products were well preserved and in good condition.. The product of most concern was the sample of ensiled high moisture corn. Following harvesting, high moisture corn is blown into a vertical structure, ensiled and removed via a silo unloader. Following these processes the product appeared to be extremely fine in consistency. Given the high moisture content of this product it could be expected that a high rate of ruminal fermentation should result. Conversely it could be possible for the starch like component to escape ruminal fermentation and pass directly down the digestive tract. The high moisture corn was shaken using Fisher Scientific brass sieves (#4, #8, #16 and pan). The results were 32% remaining on the #4 sieve, 37% on #8, 13% on #16 and 17% in the pan.

Since the onset of the increased incidence rate the producer kept detailed records of all cows that had died. No clear trends emerged as milk production of affected cows ranged from 50–120 pounds of milk daily and days in milk ranged from 10–455. The producer expressed an opinion that all affected individuals were aggressive eaters (high DMI). No deaths occurred in first calf heifers.

Figure 1 displays the herd management level milk compared with the occurrences of deaths. Examination of Figure 1 indicates a trend that management level milk increases were associated with an increase in death losses. Factors associated with increased production of milk could be considered a potential risk factor for JHS.

## Management Level Milk Vs. Occurrences of JHS

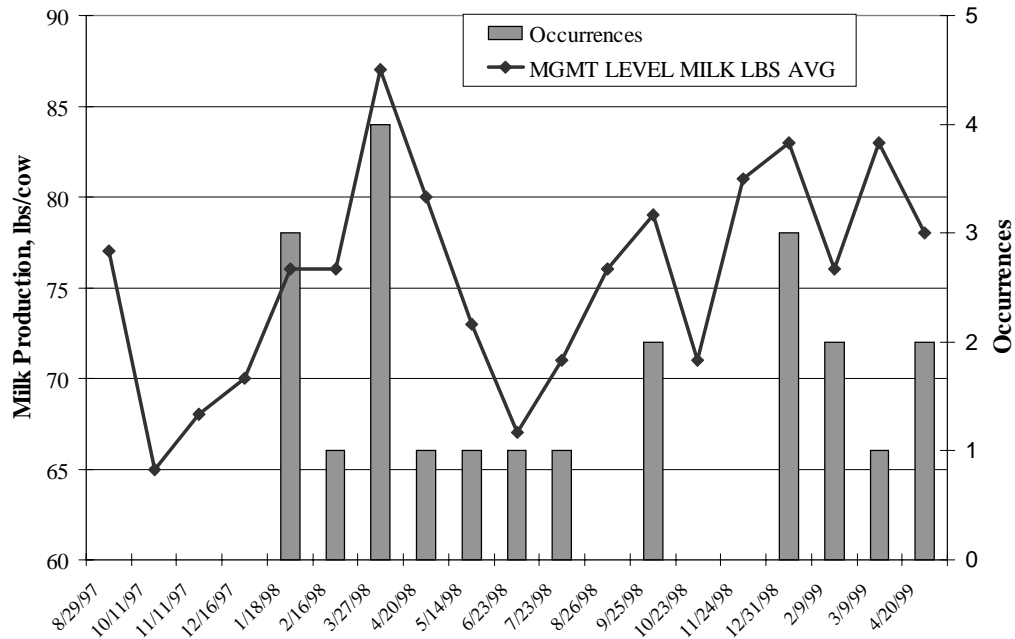


Figure 1. Herd management level milk vs. occurrences of death

High carbohydrate levels or low fiber levels may predispose lactating cows to subclinical ruminal acidosis defined by a rumen pH of  $< 5.5$ . Suspect pH readings for subclinical ruminal acidosis are 5.8 or less. During low rumen pH, ruminal volatile fatty acid proportions change; acetate levels drop, while proprionate levels increase. No drop in herd DHI butterfat levels was noted that could be associated with peaks in the deaths due to JHS. A confounding factor is that DHIA reporting is a once a month snapshot that could miss a transient whole herd acidosis event occurring during another part of the month. To achieve better sensitivity through increased sample frequency, the researchers analyzed the bulk tank pickup records for herd butterfat and milk protein percentages. Figure 2 represents the JHS events compared to bulk tank records for butterfat and milk protein. Examining the records (using statistical process controls) at the time of the JHS incidences did not indicate any characteristic whole herd butterfat drops in relation to milk protein.

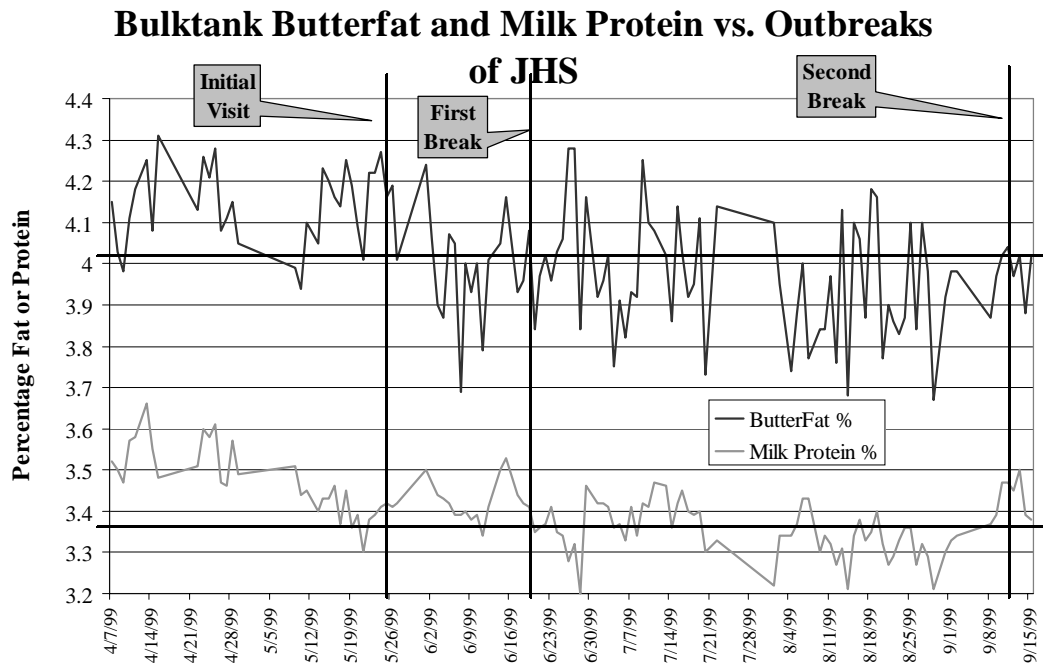


Figure 2. Daily bulk tank butterfat and protein measurements

Table 3 examines the days in milk and milk production distribution of individuals with technical DHI butterfat inversions. A broad range of milk production was noted for all lactations with technical inversions. First lactation individuals demonstrated inversions only of individuals greater than 250 days in milk. Greater than 1<sup>st</sup> lactation individuals demonstrated inversions in a range of 38 to 436 days in milk. This tends to mimic the pattern of days in milk distribution of the deaths attributed to JHS in this particular herd. While interesting, cows with technical inversions can be considered only at risk for SARA. Definitive diagnosis of this depends on demonstrating ruminal pHs of 5.5 or less.

**Table 3: Distribution of days in milk and milk production for cows identified as having a butterfat to milk protein ratio of <1.0.**

<b>First Lactation Cows (n = 5)</b>				
	Days in Milk	Milk lbs.	Butterfat Percent	Milk Protein %
Mean	343	43	3.20	3.78
Median	356	35	3.00	3.80
SE Mean	24.04	9.72	0.15	0.12
Minimum	256	16		
Maximum	398	67		

<b>Greater than First Lactation Cows (n = 17)</b>				
	Days in Milk	Milk lbs.	Butterfat Percent	Milk Protein %
Mean	238	67	3.02	3.57
Median	260	59	3.10	3.60
SE Mean	26.52	6.45	0.14	0.06
Minimum	38	16		
Maximum	436	118		

**Diagnostic testing:** Prior to the field investigation the veterinary practitioner harvested post-mortem tissues and submitted them on three different occasions to diagnostic laboratories at South Dakota State University, University of Wisconsin and Iowa State University (ISU). Isolations at all three institutions yielded high numbers of *Clostridium perfringens* type A on culture. Isolations from other dairies experiencing this syndrome that had been submitted to the ISU Diagnostic Laboratory have also yielded *Clostridium perfringens* type A.

**Management/Prospective Investigation:** With the potential routes of pathogenesis determined, steps were taken to monitor future outbreaks.

- **Establishment of a rolling 10 day bank of TMR samples**
- **Keep a log of daily feed intake** –producer elected not to maintain this .
- **Submit the next case or fatality** to the ISU Veterinary Teaching Hospital for treatment and/or evaluation.
- **Submit samples from all ensiled feeds** for Clostridial culture as per the isolation procedure and for mycotoxin analysis.
- **Submit several herd fecal samples** to examine for the presence of digestive tract parasites leading to small intestine motility aberrations.

**Clostridium culture and isolation:** A standardized protocol was developed at the Iowa State University Veterinary Diagnostic Laboratory for isolation of *Clostridium perfringens* type A from feed, haylage, silage and feces. Toxin analysis and typing were done by Dr. Glenn Songer at The University of Arizona.

**Initial laboratory results:** All ensiled materials had no detectable amounts of aflatoxin, ochratoxin, vomitoxin, zearalenone and T-2 toxin. Fecal flotations on mixed samples from the herd were negative for parasite eggs. Efforts to isolate *Clostridium* sp. from the corn silage and high moisture corn were negative. An alfalfa haylage sample was obtained on the day of the field investigation from the vertical silo (old crop), which was the last of that particular batch. A new crop sample from the same structure was obtained for culture 45 days following ensiling both old and new crop yielded *Clostridium perfringens* type A.

Due to the number of positive isolations from alfalfa haylage samples, the investigation team decided to sample haylage from other dairies in the area. Six samples were obtained from a variety of structures including, bunkers, upright silos and plastic bags. Four of the six samples returned positive isolations of *Clostridium perfringens* type A with one isolation being positive following genotyping for production of Beta2 toxin. Sampled dairies had no previous or current history of JHD.

**Further disease outbreaks:** When the producer finished the old crop alfalfa haylage, the nutritionist recalculated the ration to include more long-stem hay to counter the loss of the haylage. No deaths were reported during the 3.5 weeks that the herd was on the modified ration. As alfalfa haylage became available the producer reverted back to the original ration with haylage. Within 1.5 weeks of the ration change, 4 cattle were affected overnight on June 21 1999. Two of the affected individuals were found dead and the two that survived were transported to the Iowa State University Veterinary Teaching Hospital. Both were dead on arrival. Post-mortems were performed immediately. Post-mortem revealed segmental hemorrhaging, clotting, subsequent intestinal blockage, and intestinal intussusceptions in both cows. Cultures were performed on the isolated tissues revealing large numbers of colonies of *Clostridium perfringens* type A by direct isolation method. No Beta2 toxin expression was found in the *Clostridium perfringens* type A isolations. No *Salmonella* species were isolated.

The 10-day TMR sample bank was evaluated. There was evidence that on days 4 and 2 prior to the event, long stem fiber levels dropped to approximately 6%. Either the hay was being processed too fine, or there was a change in the total amount that was placed in the ration. Combined with sorting by the cows, the ration could have entered an area of carbohydrate risk. Additionally, the timing of this outbreak could suggest that the resumption of haylage feeding presented an increased risk due to Clostridial contamination.

On September 13, 1999 the producer again experienced a disease break with 2 animals being affected. The producer implemented immediate oral treatment intervention using 2 gallons mineral oil and 75 million units of Procaine Penicillin G. The cow was also administered 6 gm of Amp-Equine (ampicillin sodium) IV. Both animals were transported to ISU on the 3<sup>rd</sup> day following the break. One cow was dead on arrival, and due to the extremely poor prognosis, the other was euthanized. Post-mortem results noted segmental hemorrhage, clotting and blockage. *Clostridium perfringens* type A was again isolated on direct culture, along with a Group B *Salmonella* sp. in both animals.

Prior to the disease break the producer had run out of corn silage. He refilled the bunker and sealed it. One week later he opened the new corn silage bunker and within 2 days the outbreak occurred.

### **Discussion:**

For *Clostridium perfringens* to cause disease there has to be three elements:

1. *Clostridium perfringens* must be **present** in the intestinal tract.
2. There must be an **abundance of nutrients**, especially carbohydrates for organism growth and sporulation.
3. There must be at least a **partial slowdown or stoppage** of intestinal tract movement brought about by ingesting a particular large amount of feed, allowing the toxins of *Clostridium perfringens* to accumulate and be absorbed in the gut.

Based on the previous isolations of *Clostridium perfringens* type A by South Dakota State University, University of Wisconsin and Iowa State University, the sporadic and peracute presentation of the syndrome, the presentation of a segmental hemorrhagic enteritis and the association with ration changes, the investigators elected to more fully investigate the possibility that *Clostridium perfringens* type A could be involved with this syndrome.

The investigators identified possible mechanisms of disease based on known Clostridial enteritis presentations. In humans the presence of *Clostridium perfringens* type A causes food poisoning through the activity of *Cl. perfringens* enterotoxin. In this instance the risk factor is the presence of the causative organism itself. A second possible mechanism is represented by lamb enterotoxemia disease (overeating disease) caused by *Clostridium perfringens* type D growth and sporulation. The presence of the bacteria in the lumen of the small intestine is not enough to cause disease on its own, and it can only be replicated by direct intestinal injection of the live organism with Dextrin. Once present the ingestion of lush, rapidly growing pasture or cereal crops, heavy grain feeding in feedlots, *Clostridium perfringens* type D proliferates rapidly, sporulates and produces high amounts of toxins, thus causing clinical expression of the disease syndrome. The major risk factor is high amounts of fermentable carbohydrates. The third model of disease considered was a disruption of intestinal motility leading to ingesta stasis, thus leading to Clostridial overgrowth and sporulation.

### **Items for your investigation:**

**Individual animals:** If and when animals show symptoms of JHS, it should be deemed a medical emergency with attempts to keep the bowels flowing if the animal is still alive. In the case of sudden death with JHS symptoms, postmortems to confirm intestinal hemorrhaging/clotting are important. Tissue samples must be obtained rapidly postmortem as *Clostridium perfringens* type A will proliferate postmortem and may mask other problems. Consideration of genotyping for presence of Beta2 toxin might be warranted.

**Sources of *Clostridium perfringens* type A:** Clostridial contamination of alfalfa haylage is not unique. Alfalfa haylage samples from 6 dairies in NE Iowa yielded *Clostridium perfringens* type A isolations in 4 locations, with one sample positive for Beta2 toxin production. Clostridium can be found in many feed samples, especially haylages, and in very high numbers if ensiled too wet (<35% DM). Consideration could be given to running a fermentation analysis or a fermented

forage culture. However, in many situations, wet haylage may decrease palatability and intake, and slow down DMI. Also, studies where *Clostridium perfringens* type A have been directly infused into areas of the gut have not been able to create JHS.

**Carbohydrate consumption:** The most interesting piece of evidence that JHS might be associated with feeding practices is figure 1, which showed an association of increased death rates with increased management level milk. Maximal milk production is a product of dry matter intake and carbohydrate consumption, both of which could be considered as possible risk factors for JHS in this particular herd. Observations from the herd owner stating that affected animals were the most aggressive eaters to feedlot cases associated with alteration in dry matter intakes (where feed engorgement following lower consumption during weather changes) may implicate the role of rapid rumen turnover and increased rates of nutrient passage to the lower GIT. Finally, the role of ration changes can not be overlooked as many of the herd outbreaks occurred within 1-2 weeks following a feed change. If not already a routine practice in the herd (and certainly during an outbreak), the following analyses should be completed: 1) evaluation of the ration on paper and NIR/or wet chemistry analysis of the ration with specific interests on NEL, NFC, fat and fiber fractions, as well as effective fiber; and 2) particle size analysis of individual components and the mixed ration, including freshly delivered feed and refusals to assess proper mixing, uniform bunk delivery, and sorting issues. Consider keeping a rolling collection of ration samples for analysis.

**Disruption of gut motility:** We have evidence of gut motility disruption and the presentation of JHS as presented by the two cows submitted from the June 1999 outbreak. The presence of intussusceptions in both animals from one dairy on one day was highly unusual and points to the possibility that some form of intestinal motility aberration was possible. This abnormal intestinal motility could possibly take the form of either hypo or hypermotility. In both animals the intussusception was located directly anterior to the JHS site in the jejunum. An additional consideration concerning the presence of intussusceptions was presented in a retrospective analysis of intussusception in cattle: 336 cases (1964-1993). Analysis of small intestinal intussusception indicated the Brown Swiss breed had an adjusted odds ratio of 4.18, which was significantly higher than those of the reference group. The Holstein, Jersey, and other dairy breeds had adjusted odds ratios of 1.00, 0.65 and 0.48 respectively. This would indicate the Brown Swiss are particularly predisposed to intestinal motility aberrations, and may have increased risk factors with respect to presentation of both intussusceptions and JHS. While there was no evidence to indicate to the investigators which lesion presented first, there is information to suggest that enteritis is associated with intussusception. Conversely, the increased risk factors for intussusception in Brown Swiss cattle it would suggest that the breed itself is at greater risk. The most important point is that it appears likely that some form of intestinal motility aberration is associated with clinical JHS, and it would ask the question if alpha toxin could disrupt neuromuscular transmission.

**Vaccination:** Current commercial and autogenous vaccines and toxoids offer little help for JHS. An aggressive Clostridial vaccination program was pursued by the herd owner in our investigation for control of JHS. This included the quarterly administration of a commercial 7-way bacterin toxoid and an autogenous *Clostridium perfringens* type A bacterin toxoid every 60 days. There was no apparent remission in the case incidence rate with the use of these vaccines.

Conversely, the herd became sensitized to the commercial 7-way product and reduced dry matter intake and dropped production dramatically following administration. Administration of *C. perfringens* C and D toxoid may have some effect if infection is mixed, or due to presence of *C. perfringens* type A in the toxoid.

**Conclusions:**

We were not able to identify a cause and effect relationship between *Clostridium perfringens* type A and the presence of JHS. The data though does suggest new avenues for further investigation. This includes a possible association between increased levels of milk production and increased risk of JHS, the onset of JHS with increased soluble carbohydrate levels, low effective fiber levels, and factors accentuating faster rates of carbohydrate passage to the lower GIT, the presence of disease following the re-introduction of *Clostridium perfringens* type A positive alfalfa haylage, the association of JHS with feed changes and alterations of DMI, and the possible role that intestinal motility aberrations play in the pathogenesis of this syndrome. The wide range of investigational items would suggest JHS is a true syndrome whose presentation may not be solely dependent on the presence of a causative organism, but on the combination of a range of conditions.

**References:**

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