Jejunal Hemorrhage Syndrome of Dairy Cattle

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Introduction

In the last three 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 (α, β, ε, and ι). *Clostridium perfringens* type A produces α toxin; type B produce α, β, and ε toxins; type C produce α and β toxins; type D produce α and ε toxins; and type E strains produce α and ι 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 masks the presence of other organisms (Kirkpatrick et al., 2001).

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 with a genotype that expresses beta2 toxin. A Pennsylvania study of 47 cases in adult dairy cattle (25 isolates) yielded the beta2 toxin (Kirkpatrick et al., 2001). 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. Despite the low morbidity some individual herds have experienced multiple individual and repeated 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 (early indicative) 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 (sweats)
- Bruxism (teeth grinding)
- Enopthalmia (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 (bloody stools) 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 as early as possible, keeping in mind that overgrowth can occur early following death.

Post-mortem findings: Observations have demonstrated segmental lesions localized to the jejunum. These areas consist of frank hemorrhage and highly organized 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 meglamine (Banamine®; 1.1 mg/kg IV) or isoflupredone (Predef 2X®; 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 anastamosis ; 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 on 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 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 shaker 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. The 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 calculation and wet chemistry analysis.

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 were the TMR 24 hr refusals. The increase in 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.

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 was 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 would result. Conversely, it would 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.

High carbohydrate levels or low fiber levels may predispose lactating cows to subclinical ruminal

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

Feed	As Fed Basis lb/head/day
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
Total	100.5

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. This results in a drop in milk butterfat levels. 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 investigators analyzed the bulk tank pickup records for herd butterfat and milk protein percentages. 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.

The investigators decided to examine the herd records to identify possible patterns associated with individuals manifesting technical DHI butterfat inversions. Table 4 examines the days in milk and milk production distribution of individuals with these inversions. A broad range of milk production was noted for all lactations with inversions. First lactation individuals demonstrated inversions only in individuals greater than 250 days in milk. Greater than 1st 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.

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.

- **Establish 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 ISU Veterinary Diagnostic Laboratory for isolation of *Clostridium perfringens* type A from feed, haylage, silage, and feces. Dr. Glenn Songer at The University of Arizona performed toxin analysis and typing.

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.

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

Ration Determinations	Calculated Dry Matter Basis	Wet Chemistry Dry Matter Basis
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%
NE_L Mcal/cwt	77.00	77.47
NE_G Mcal/cwt	53.00	52.37
NE_M Mcal/cwt	81.00	80.66

Table 3. TMR Shake Test results.

	Average NE Iowa TMR	Subject Herd TMR	Subject Herd TMR Refusals
Long Fiber Length – Top Pan (% of material)	8.70%	11.10%	23.40%
Medium Fiber Length – Middle Pan (% of material)	34.50%	36.80%	35.30%
Short Fiber Length – Bottom Pan (% of material)	56.90%	52.10%	41.30%

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, four 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 ISU 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 two animals being affected. The producer implemented immediate oral treatment intervention using two gallons of 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 3rd 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.

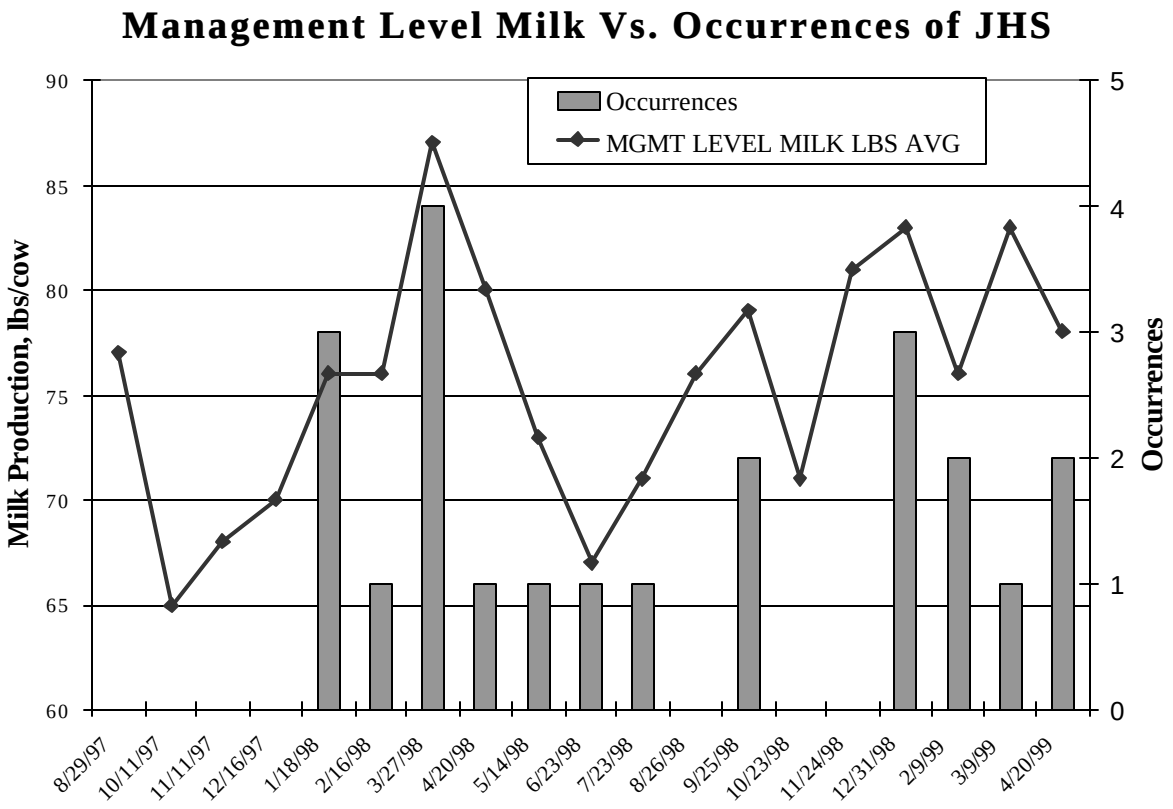
Discussion

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 ISU; 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 review the

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



possibility that *C. perfringens* type A could be involved with this syndrome.

Possible mechanisms of action: 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 *C. 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 *C. 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 along with Dextrin (a sugar source). 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 lamb enterotoxemia. 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.

Clostridium perfringens type A as a direct contaminant: In this particular herd *Clostridium perfringens* type A was directly isolated from repeated haylage samples taken from a vertical structure. An outbreak occurred within 10 days of the re-inclusion of the alfalfa haylage into the diet. As a result of the isolations the investigators hypothesized about the possibility of the vertical structure being contaminated with *Clostridium*

perfringens type A spores. The owner agreed to put up haylage in a plastic bag, which would be relatively free of contamination. This product was sampled for culture and isolation and proved to be very difficult to isolate *C. perfringens* type A. The producer reported no incidences of JHS while on this product.

If contamination is suspected, the producer or nutritionist could consider performing a fermentation analysis to examine the product for high levels of butyric acid (a fermentation by-product of Clostridial species). High levels could be considered a risk factor to indicate Clostridial contamination. Additionally, butyric acid is a precursor to beta-hydroxybutyrate and has been used as a research model to induce clinical ketosis in dairy cattle. High levels of butyric acid may decrease product palatability and dry matter intakes, and may contribute to higher than normal levels of clinical ketosis. In addition to fermentation analysis a direct culture of the product may be performed to characterize contamination levels.

Clostridial contamination of alfalfa haylage though is not an event unique to this dairy. Alfalfa haylage samples from six dairies in NE Iowa yielded *Clostridium perfringens* type A isolations in four locations, with one sample positive for beta2 toxin production with no incidences of JHS noted at any of these locations. A study by Ivany et al. (2001) failed to demonstrate clinical JHS following direct infusion of *Clostridium perfringens type A* alone into the jejunal lumen. The author concluded that the state of the bacteria (spore vs. vegetative), level of feed intake, pH of the gastrointestinal tract, stress levels, stage of lactation, and bacterial concentration are all factors that may be related to presentation of 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. Dry matter intake is of particular interest in that all cases of JHS were confined to 2nd lactation plus individuals. When compared to a first lactation individual the only difference becomes dry matter intake. Further supporting evidence for carbohydrates as a risk factor is the September, 1999 outbreak following the addition of corn silage to the ration that had only been fermented for 10 days. During this time period other dairies in this region reported similar outbreaks of JHS when feeding corn silage that had gone through a limited fermentation process.

The herd owner observed that the affected animals were the most aggressive eaters. Feed engorgement could lead to subacute rumenal acidosis or escape of unfermented carbohydrates to the intestinal lumen allowing lumenal vegetative growth of *Clostridium perfringens*. The investigators were unable to establish a pattern of subacute rumenal acidosis in this case. This diagnostic route is further complicated by the transient nature of subacute rumenal acidosis as related to feeding practices or intakes following changes in weather conditions.

Finally, the role of ration changes cannot 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 near infrared analysis/or wet chemistry analysis of the ration with specific interests on NE_L, 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.

Table 4. Distribution of days in milk and milk production for cows identified as having a butterfat to milk protein ratio of <1.0.

First Lactation Cows (n = 5)				
	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		

Greater than First Lactation Cows (n = 17)				
	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		

The Holstein, Jersey, and other dairy breeds had adjusted odds ratios of 1.00, 0.65 and 0.48 respectively. This would indicate 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 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. The herd owner pursued an aggressive Clostridial vaccination program 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 production dropped dramatically following administration. Administration of *C. perfringens* C & D toxoid may have some benefit if the infection is mixed. An alternative line of reasoning is that it is very difficult to produce a C & D toxoid without some contamination from C. perfringens type A. Due to this contamination there may be some alpha toxoid created in the production of this vaccine.

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 and low effective fiber levels, 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.

Take Home Messages

Clostridium perfringens type A has been implicated in the cause of enteric disease in domestic animals. Current research indicates that a variant *C. perfringens* type A producing a beta2 toxin may be involved. Any culture work performed on case samples should include genotyping for the beta2 variant.

- 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 particularly large amount of feed, allowing the toxins of *Clostridium perfringens* to accumulate and be absorbed in the gut.
- Don't make assumptions about the cause of death of individual cases. It is worthwhile having a postmortem examination performed on suspicious cases. Potential rule-outs include enteritis caused by Salmonella species or acute, bleeding abomasal ulcers.
- JHS may be a multi-factorial syndrome, allowing presentation under various circumstances. Diagnostic procedures should include a work-up of the feeding management on affected facilities.

References

Kirkpatrick, M.A., L.L. Timms, K.W. Kersting, and J.M. Kinyon. 2001. Case report – jejunal hemorrhage syndrome of dairy cattle. The Bovine Practitioner. 35 (2):104.

Ivany, J., and D.E. Anderson. 2001. Determination of the role of Clostridium perfringens type A in intraluminal intestinal hemorrhage syndrome in dairy cows. The Bovine Practitioner 35(2):145.

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