Dairy cows and their gut problems

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Take home message:

It appears to be getting more difficult to send an ever increasing amount of dry matter through the small intestines of the modern dairy cow.
Recognize high feed consumption of the modern dairy cow

- Dairy cows eat **A LOT** of feed and we celebrate it.
- In order to get a large amount of feed through a cow, it has to move fast = **High rate of passage**.
- The rumen is tough and can expand to accept large quantities of feed, but the small intestines thin-walled and can plug, twist, leak and rupture.
- Most gut problems happen **after the rumen** (twisted stomach, intestinal problems).
This is the small and large intestines from a 600 lb. Holstein heifer.

It’s 120 feet long... and about the diameter of a golf ball.
Cattle compared to cats & dogs

Cow = 120 feet

Dog = 7 feet

Cat = 4 feet

If interested, human intestines are about 40 feet (no picture provided).
High feed consumption

• A mid-lactation, 1,500 lb. cow eats 4% of her body weight, it’s eating 60 lbs. of dry matter.
• Do any other livestock eat 4% of their body weight?
  – Comparable 1,200 beef cow rarely eats more than 30 lb of DM, or 2.5% of BW (37% less than a dairy cow).
  – Forestomach and abomasal impaction is seen in cows that don’t drink enough water or are fed a coarse, poorly digestible high fiber diet.
Rumen impaction

Rumen content should “flow.” Poor water consumption is a common cause in cattle. Beef cattle will impact with coarse, high ADF feeds.
### Feed : Small Intestinal Area Ratio

<table>
<thead>
<tr>
<th>Animal</th>
<th>Lbs. of feed consumed (4% BW)</th>
<th>Intestine area (cm²)</th>
<th>Ratio of lb feed consumed per cm² of intestine area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cow (1,400 lb)</td>
<td>49</td>
<td>11.5</td>
<td>4.2 lbs. / cm²</td>
</tr>
<tr>
<td>Dog (50 lb)</td>
<td>1.75</td>
<td>1.6</td>
<td>1.1 lbs. / cm²</td>
</tr>
<tr>
<td>Cat (10 lb)</td>
<td>0.35</td>
<td>0.3</td>
<td>1.2 lbs. / cm²</td>
</tr>
</tbody>
</table>

Dairy cows push 4X more feed through the same small intestinal area than a cat or dog.
Evidence that high DMI is a risk factor for non-descript gut problems

- Gut problems tends to hit high producing cows in mid lactation.
- Many farmers say “It was the best cows in the barn.”
- Less common in dry cows...which eat less
- Less common in beef cows...which eat less
- HAVING WRITEN THIS. REDUCING DMI TO ALLEVIATE A GUT PROBLEM IS NOT JUSTIFIED. REDUCING DMI CAN CREATE A SLUG FEEDING BEHAVIOR WHICH APPEARS TO BE A RISK FACTOR FOR DIGESTIVE UPSETS.
Elephant in the room = HBS

- What is HBS?
- Why don’t we know more about HBS?
- What causes HBS?
- What can be done to treat HBS?
- Why do most cows with HBS die?
- What should I do if I have HBS cows?
Definition of HBS

• Sporadic intestinal disease of milking cows also known as “bloody gut” and “jejunal hemorrhagic syndrome.”
  – Reported as far back as 1966.

• Blood clots obstruct and enlarge the intestines
  – Serum chemistry is consistent with obstruction: High glucose, high magnesium, low sodium, low potassium and low chloride.
  – Fatality rate ~85%
Hemorrhagic Bowel Syndrome (HBS)
Why we don’t know more about HBS

• We can’t reproduce the disease: When bloody intestinal contents saturated with *Clostridium sp.* are fed back to cows they don’t get sick.

• If I wanted to reproduce the disease, I would do the following.
  1. Get a high-producing dairy cow hungry
  2. Mix a ration of non-fermented corn silage, moldy cracked corn and a small pail of sand and crushed gravel.
  3. Hope she’s hungry enough to eat a slug of the feed in a short time period.
  4. Don’t offer water, don’t let her lay down, or allow her to chew cud.
HBS cases by herd size and milk production

High production = High DMI and more case of HBS.

Could small, tie stall herds have less issues because cows can’t eat all they want?
Hemorrhagic Bowel Syndrome

Figure 1. Number of HBS Cases by Year.

Figure 5. Percent of Operations that Reported Having at Least One HBS Case During the Previous 5 Years, by Season.
My interpretation of the seasonal differences is that (1) inadequate corn silage fermentation is a major driver in the fall and winter and (2) fresh feeds in the spring/summer may be a minor driver.

<table>
<thead>
<tr>
<th>Season</th>
<th>Summer 24%</th>
<th>Fall 27%</th>
<th>Winter 38%</th>
<th>Spring 11%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-ferm</td>
<td>Fresh feed +/- fermentation</td>
<td>Fresh, and unfermented</td>
<td>Some unfermented</td>
<td>No fresh feed, all fermented</td>
</tr>
<tr>
<td>Fresh feed</td>
<td>Fermented</td>
<td>Not fermented</td>
<td>+/- fermented</td>
<td>Fermented</td>
</tr>
<tr>
<td>Corn silage</td>
<td>Fermented</td>
<td>Not fermented</td>
<td>+/- fermented</td>
<td>Fermented</td>
</tr>
</tbody>
</table>
What causes HBS?

• Cause? Nobody really knows. It’s still a syndrome.

• The following have been postulated
  – Molds and mycotoxins
  – *Clostridium perfringens* type A
  – Other bacteria (*E. coli*, etc.)
  – Management:
    • Bunker, bunk, sorting, slug feeding, inconsistent mixing, etc.
  – Excessive ash (dirt, soil, gravel, sand, rocks, etc.)
**Clostridium perfringens?** Similarities, but many differences.

<table>
<thead>
<tr>
<th></th>
<th>Ovine over-eating disease</th>
<th>HBS in cattle</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cause</strong></td>
<td><em>Clostridium perfringens</em> C and D infection and overgrowth in the starch-saturated intestines</td>
<td>Multifactoral. <em>Clostridium perfringens</em> type A is often grown, but is it the cause?</td>
</tr>
<tr>
<td><strong>Death mechanism</strong></td>
<td><strong>Toxin!</strong> Lesions and death are caused by the potent, lethal toxins</td>
<td><strong>Plugged intestines?</strong> There are no toxic lesions. Emphysema, necrosis, etc.</td>
</tr>
<tr>
<td><strong>Role of Vaccination</strong></td>
<td>C and D vaccines do a wonderful job of stimulating the production of toxin-binding antibodies.</td>
<td>Type A vaccines might bind alpha toxin, but the cow still dies of a plugged gut.</td>
</tr>
<tr>
<td><strong>Value of Vaccination</strong></td>
<td>Essential vaccine for small ruminants. Not using a C and D vaccine is foolish.</td>
<td>In my experiences, type A vaccine has not solved our HBS problem.</td>
</tr>
</tbody>
</table>
2015 Investigation

• 15 cows diagnosed with HBS by farm personnel were examined and posted.
  – 8 cows had excessive amounts of dirt
  – 2 cows had hardware disease
  – 2 cows had an LDA
  – 1 cow had an intestinal intussusception
  – 1 cow had an RDA
  – 1 cow had “Hemorrhagic Bowel Syndrome”

• Cows passing any amount of blood in their feces are over-diagnosed as HBS.
This cow clearly has a gut problem, but is a mesenteric torsion and not HBS.
This farm has “HBS”, but the blood clots are filled with non-fermented corn cob.
The “corn cob” cows have luminal hemorrhages, but no tissue lesions.
“Corn cob” HBS

• Corn was planted in July due to wet field conditions.
• The corn never matured and was chopped late fall while frozen.
• The frozen corn silage did not fermented.
• The kernel processor on the chopper was not used because the kernels were immature, soft and didn’t need to be cracked.
• Large pieces of frozen, non-fermented corn cob were fed through the winter and spring resulting in an increase number of “HBS” cases.
The plugged intestines set off a cascade of problem which only can be fixed if the obstruction is corrected.

Some vets have surgically opened cows and broken down the obstruction, but the prognosis remains poor.

What should we treat them with?

– There are no good options.
## Blood chemistry of cows with gut problems

<table>
<thead>
<tr>
<th></th>
<th>Cow 1</th>
<th>Cow 2</th>
<th>Cow 3</th>
<th>Cow 4</th>
<th>Cow 5</th>
<th>Mean</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Glucose</strong></td>
<td>230</td>
<td>112</td>
<td>129</td>
<td>72</td>
<td>249</td>
<td>158</td>
<td>44-75</td>
</tr>
<tr>
<td><strong>AST</strong></td>
<td>91</td>
<td>133</td>
<td>102</td>
<td>84</td>
<td>87</td>
<td>99</td>
<td>48-204</td>
</tr>
<tr>
<td><strong>SDH</strong></td>
<td>73</td>
<td>41.9</td>
<td>70.3</td>
<td>24.5</td>
<td>52</td>
<td>6.6-37.8</td>
<td></td>
</tr>
<tr>
<td><strong>Total Bilirubin</strong></td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.0-2.2</td>
</tr>
<tr>
<td><strong>Cholesterol</strong></td>
<td>272</td>
<td>117</td>
<td>219</td>
<td>127</td>
<td>184</td>
<td>112</td>
<td>112-331</td>
</tr>
<tr>
<td><strong>Total protein</strong></td>
<td>7.3</td>
<td>6.9</td>
<td>6.5</td>
<td>5.1</td>
<td>8.7</td>
<td>7</td>
<td>5.6-7.8</td>
</tr>
<tr>
<td><strong>Albumin</strong></td>
<td>3.8</td>
<td>3.5</td>
<td>3.9</td>
<td>2.9</td>
<td>4.5</td>
<td>4</td>
<td>3.1-4.3</td>
</tr>
<tr>
<td><strong>Urea</strong></td>
<td>29</td>
<td>44</td>
<td>27</td>
<td>58</td>
<td>32</td>
<td>38</td>
<td>8-22</td>
</tr>
<tr>
<td><strong>Creatinine</strong></td>
<td>1</td>
<td>0.7</td>
<td>0.5</td>
<td>1.1</td>
<td>0.8</td>
<td>1</td>
<td>0.3-0.8</td>
</tr>
<tr>
<td><strong>Phosphorus</strong></td>
<td>5.9</td>
<td>10.7</td>
<td>6.7</td>
<td>6.1</td>
<td>7.6</td>
<td>7</td>
<td>4.1-8.3</td>
</tr>
<tr>
<td><strong>Calcium</strong></td>
<td>7.6</td>
<td>7.9</td>
<td>5.3</td>
<td>9.1</td>
<td>7.2</td>
<td>7</td>
<td>7.9-10.5</td>
</tr>
<tr>
<td><strong>Sodium</strong></td>
<td>135</td>
<td>130</td>
<td>134</td>
<td>146</td>
<td>125</td>
<td>134</td>
<td>135-145</td>
</tr>
<tr>
<td><strong>Potassium</strong></td>
<td>2.4</td>
<td>3.4</td>
<td>3.6</td>
<td>3.8</td>
<td>7.2</td>
<td>4</td>
<td>3.7-5.6</td>
</tr>
<tr>
<td><strong>Chloride</strong></td>
<td>89</td>
<td>79</td>
<td>90</td>
<td>96</td>
<td>79</td>
<td>87</td>
<td>100-109</td>
</tr>
<tr>
<td><strong>Bicarb</strong></td>
<td>27</td>
<td>27</td>
<td>29</td>
<td>40</td>
<td>31</td>
<td>31</td>
<td>22-29</td>
</tr>
<tr>
<td><strong>CK</strong></td>
<td>780</td>
<td>2609</td>
<td>382</td>
<td>864</td>
<td>290</td>
<td>985</td>
<td>50-271</td>
</tr>
<tr>
<td><strong>GGT</strong></td>
<td>20</td>
<td>20</td>
<td>29</td>
<td>27</td>
<td>25</td>
<td>24</td>
<td>4-41</td>
</tr>
<tr>
<td><strong>Anion Gap</strong></td>
<td>21</td>
<td>27</td>
<td>19</td>
<td>14</td>
<td>20</td>
<td>20</td>
<td>10-25</td>
</tr>
<tr>
<td><strong>Globulin</strong></td>
<td>3.5</td>
<td>3.4</td>
<td>2.6</td>
<td>2.2</td>
<td>4.2</td>
<td>3</td>
<td>2-4</td>
</tr>
</tbody>
</table>
Clinical ramifications of HBS.

How do they feel? Blood changes...

**High blood urea:**
- Nausea, vomiting (if cows could vomit), fatigue, muscle cramps, visual disturbances, increased thirst, mental changes, shallow breathing, low body temperature

**Low blood potassium (K):**
- **Heart** palpitations, muscle weakness and cramping, paralysis, constipation, nausea, abdominal cramping, increased thirst, mental changes (delirium & hallucinations)

**High blood glucose:**
- Glucose is high because of shock.
- **What is shock?** Shock is a life-treatening condition when the body is not getting enough blood flow due to poor blood flow. In HBS cows, cardiac output is slowed because high blood urea and high potassium is low altering **heart function**.
HBS an intestinal problem, but the rumen plays an important roll

- The rumen is a **reducing** environment.
  - The rumen takes big things and makes them smaller
  - **Protein** is reduced to ammonia and urea to build microbial protein.
  - **Fiber** is both physically broken down and fiber digesting bacteria generate VFAs.
  - **Cud chewing reduces** particle size and stimulates saliva production
If the rumen fails to reduce....

• Increased amounts of starches reach the small intestines.
  – The small intestines is not expecting starches.
  – Starches are fuel for Clostridium spp. and other non-desirable gas-producing bacteria.

• Increased large particles that may not have be “chewed up” have to potential to create an obstruction.

• Increased number of molds and mycotoxins can reach the small intestines.
  – The reducing environment of the rumen detoxifies some mycotoxins.
  – Ruminants can tolerate mycotoxins better than monogastrics like pigs, horses, chickens, etc.
  – Example: Beef cows with slow rates of passage appear to tolerate more mycotoxins because they stay in the rumen longer.
Causes of marginal rumen reduction

Ever increasing DMI = More feed = faster rate = incomplete reduction?
  - Not enough hours in a day?
  - Maybe cows need to chew cud faster?
  - Would direct-fed microbials (DFM) help?

**Slug feeding:** The rumen is full and some feed *spills* into the small intestines before being reducing.

**Non-rumen-friendly feeds:** Molds, mycotoxins and unfermented feeds stunt rumen reduction.
“Gravelitis:” Corn Silage bag on a gravel way to much contamination
Duodenum obstructed with gravel.
Dairy heifer fed silage from a gravel-based bunker, March 2016
Another heifer in the pen.
March 2016
Grazing beef cow that died from flukes. “Normal” sand accumulation in a grazing, mineral deficient beef cow.

In my experiences, a handful of sand is expected, a bucket-full is not.
Sand and gravel can irritate the gut...but also consider teeth damage.
Cud chewing & particle size reduction depends on functional teeth.

Upper molars of a cow
Digestive upset interventions

• Decrease the ash content of the feed. Top soil will work though the GI tract, but gravel and sand appear to accumulate.

• Before pushing feed up to cows, push the edge of the feed pile and the dirt from the tires to the center of the barn for disposal.

• Consider increasing the amount of weigh back refusals. Let cows sort undesirable feed and get rid of it. Don’t force cows to eat cobs, stalks, dirt, Ag Bag plastic and chunks of mold.

• Make sure feed is available when cows return from milking.

• Increase NDF from forage to stabilize the rumen. Fiber will displace some starches and may act like a net preventing large particles from slugging the intestines.

• Consider increasing soluble protein (RDP). Proteins soften GI contents and increases rate of passage. Monitor MUNs when attempting. Should range between 10 and 14.

• Consider a direct-fed microbial (DFM) when switching forages.
HBS interventions – Sick cows

• If possible, move sick cows to green grass or pasture. Green grass is a good laxative.

• Oral fluids including mineral oil and psyllium.

• If IV, don’t use glucose. Hypertonic saline is good for replacement of serum chloride.

• If abdominal exploratory, document omasal/abomasal impaction and sand and gravel accumulation.

• Post dead cows to document non-HBS pathology.

• Document sand and gravel accumulation. Attempt to dissolve any blood clots looking for coarse, sharp substances like cob, trash, etc.
Summary

• Pushing ever increasing amounts of feed through a cow requires even more consistency.
  – Limit things that stunt rumen reduction
    • Molds, mycotoxins, unfermented feeds
    • Consistent fiber to stabilize the rumen mat
    • Direct Fed Microbials (DFM) during feed transitions?
  – Avoid slug feeding
    • Monitor and document empty bunk syndrome
    • Feed should be available to the first cow back from the parlor.
  – Limit consumption of sand, gravel, dirt, soil
• Don’t expect HBS to be solved by an injection.