Gastric Ulcers

The first description of gastric ulcers was written in 1897, but the condition remained as a rare or undiagnosed problem until the 1950’s and 60’s. Outbreaks of gastric ulcers coincided with the introduction of modern feeding and husbandry practices such as the use of grain-based processed feeds and confinement rearing. Today gastric ulceration is a common problem almost everywhere in the world, and a major cause of mortality and economic loss, as well as a welfare concern.

Clinical Signs

The clinical signs of a gastric ulcer tend to reflect the degree of blood loss. Very commonly there is no warning that the pig is sick, until a pale pig in good body condition is found dead. When blood loss is less severe, pigs become anemic and may appear pale. Often pigs that have lost blood will be weak and reluctant to stand. They will have a rapid heart rate and may have labored breathing. The feces will be black due to the presence of digested blood, but diarrhea is not associated with this condition and this can be a useful finding to help rule-out other diseases such as porcine proliferative enteropathy. Some pigs will show signs of abdominal pain and arch their backs or grind their teeth but these findings are not common. Ulceration occurs rapidly within hours and can heal quickly. Scarring may occur in chronic cases and if severe can block the opening to the stomach. Vomiting immediately after eating or during eating is a sign of an esophageal stricture that might have been caused by a chronic gastric ulcer.

Pathogenesis of Disease and Lesions

Lesions are restricted to the area of the stomach that surrounds the esophageal opening. The surface of this part of the stomach (the pars esophagea) is identical to the lining of the esophagus, and should be smooth and white in appearance (Figure 1). Unlike the rest of the stomach, the pars esophagea has no mucus covering for protection. In a healthy pig with access to food, the stomach is
The stomach is divided into two parts. The first section, where the feed first enters has a neutral pH and the second part of the stomach surrounding the exit to the intestines, has an acid pH. It is in the second part where enzymes and hydrochloric acid are released so that the stomach can breakdown the large feed particles and begin the digestive process. It appears that under certain circumstances the acid and digestive enzymes may move into the first section and cause irritation to the lining of the pars esophagea. If given time, the body will respond to the irritating insult by increasing the thickness of the surface layer, forming a callous (parakeratosis; Figure 2). However if the irritating juices continue to attack the surface they may work their way between the cracks and cause damage leading to deeper and deeper erosions. If a large blood vessel is damaged, the pig might lose extensive amounts of blood. The erosions can be very extensive such that the entire surface of the pars esophagea is removed. The edge of the damaged pars esophagea and the surrounding normal stomach lining may be raised, creating a crater-like appearance (Figure 3).

The exact cause of gastric ulceration in pigs is not known but it is generally accepted that there are multiple contributing factors. A combination of bile, gastric acid and digestive enzymes can rapidly cause tissue damage to the surface layer of the pars esophagea. Under normal circumstances the pig's stomach contains a quantity of undigested feed and this helps to maintain a gradient between the neutral esophageal portion and the acidic distal region. If the stomach becomes empty of solid material and fluid-filled, then the surface of the pars esophagea will be bathed in the acidic material of the distal stomach. There may be factors that might make this corrosive material more harmful. For instance it has been shown that histamine promotes gastric acid secretion and histamine is often produced in response to infection. Some diseases might cause pigs to stop eating and also cause histamine release. In humans ulcers in the duodenal part of the stomach are caused by chronic irritation of bacteria called "Helicobacter pylori." Pigs also are commonly infected with similar bacteria in the distal part of the stomach. If this bacteria contributes to ulceration in pigs it is by an indirect route because the bacteria are not located in the pars esophageal area. It has been hypothesized that the bacteria might stimulate higher levels of acidity.

It is clear that factors that contribute to increased fluidity are important contributors to ulceration. Finely ground feed is possibly the most important factor because stomach emptying time is increased in response to fine particle size. Pelleting feed and the use of a hammer mill to grind the grain contribute to the fine-
ness and are also linked to ulcer formation. Rapid stomach emptying time is more important if feed intake is disrupted. Pigs may stop eating for a variety of reasons including illness, hot weather, and contaminants in the feed, or the feed might become unavailable due to mechanical or human error. In all cases the risk of gastric ulceration is increased.

**Epidemiology**

All ages of pigs can be affected with the highest prevalence of mortality due to gastric ulceration usually among grower-finisher hogs, but sows can also have similar high levels. Numerous studies have been conducted at slaughter and typically 80 or 90% of hogs show some type of lesion in the pars esophageal region. The thickening of the surface (parakeratosis) is very common and generally considered to be insignificant as far as its effect on the pigs health. However, erosions that sometimes involve the entire pars esophagea are found in up to 20 to 25% of stomachs at slaughter and these lesions are significant. On farms with good disease control, gastric ulcers are commonly the most important cause of mortality. Endemic mortality rates due to gastric ulceration have been reported to be 1 to 2%, with sporadic outbreaks causing much higher rates. Outbreaks of ulcer deaths are frequently associated with a concurrent outbreak of respiratory or systemic disease or a disruption in feed availability. The cost of gastric ulceration is usually estimated on the basis of mortality, but this likely underestimates the true production cost. Some pigs that lose significant amounts of blood (but survive), remain anemic and grow slowly. Pigs that develop scar tissue at the opening of the esophagus into the stomach may have difficulty swallowing feed and therefore grow slowly. There is no doubt that if anemia is severe or scar tissue prevents proper feed intake, these pigs will be culled or greatly devalued. In addition, there is also evidence that gastric ulceration may lead to a less dramatic growth reduction. This is difficult to document. Most studies that have investigated this relationship have tried to correlate lesions at slaughter with growth rate but lesions occur and heal rapidly and therefore what is seen at slaughter may not reflect what happened weeks or months earlier. Also confounding this relationship is the fact that factors like finely ground pelleted feeds and lean-fast growing genetics which are associated with gastric ulcers are also associated with fast growth rate. In summary the effect of mild to moderate gastric lesions on growth rate is not well documented but may have an economic impact that is not currently recognized. Likewise the welfare aspect of gastric ulceration has not been thoroughly examined but one must assume that at least in the case of extensive erosive lesions there is pain and discomfort.

**Diagnosis**

The diagnosis is generally confirmed by post mortem examination. Frequently in the case of a sudden death and a very pale carcass, the stomach will contain a large quantity of clotted blood, but even in the absence of blood in the stomach the pars esophagea should be visualized and the intestinal content examined for evidence of black, tarry, digested blood. Generally the post mortem examination will clearly demonstrate the cause of death was a gastric ulcer, but the ulcer might be secondary to other health issues like pneumonia. The underlying disease condition might be the more important finding. Diagnosis of ulceration in the live animal poses a more difficult problem. Several common diseases can cause bleeding into the intestine, but generally these conditions are associated with diarrhea while gastric ulceration is not. A combination of clinical signs and history as well as post mortem findings are sufficient to diagnose an ulcer problem in a herd.

**Treatment and Prevention**

Pigs that are observed to be pale and weak should be segregated from pen mates to prevent bullying and severely anemic pigs should be euthanized. Individual animal treatment with pharmaceuticals is prohibitively expensive therefore rarely used, but could be employed in the case of a valuable breeding animal. The most practical approach to help a pale weak pig to regain health is to encourage the affected animal to eat. Mass medication with sodium bicarbonate or buffering agents have been used but with mixed results at best.

If a specific risk factor can be identified as the probable cause of an outbreak, then steps to correct the problem should be taken. For example, if hot weather is causing pigs to not eat, methods to cool the pigs should be explored. If the underlying problem appears to be respiratory disease, vaccination or medication might indirectly improve the prevalence of ulcers.
In most circumstances, the problem is due to a complex interaction of nutrition, environment and management, and therefore preventing ulceration requires a constant vigilance and concerted effort by the herds- men, feed providers and veterinarians. The major difficulty in preventing gastric ulceration is that the most obvious solutions such as increasing feed particle size are associated with reduced production performance so the pork producer needs to strive to maintain a balance between production efficiency and the prevalence of ulcers. Therefore, the swine industry must endeavor to produce a pellet that contains grain particles of a uniform and appropriate size that has been prepared at a proper temperature. High temperatures during pelleting and the presence of “fines” lead to ulcers. Roller mills appear to produce a feed that is less likely to cause ulcers compared to hammer mills. Feeding practices need to be carefully monitored to prevent disruptions in feed delivery, and water needs to be readily available.

At present there are no easy solutions to the problem of gastric ulcers. This is a problem that has increased in importance as other health problems are solved and as the swine industry is pressured into being more and more cost efficient. It is an economically important problem that requires the industry’s attention.