Sow Mortality

Murphy Farms, Rose Hill, North Carolina*
C. O. Duran, DVM, PhD, MRCVS

ABSTRACT: This article presents survey information on sow mortality rates, an analysis of the factors that increase mortality on individual farms, and a description of the most common causes of unexpected death in sows. Multiple-farm surveys of sow mortality revealed that the death rate is increasing, probably because of increased herd size, confinement housing, and changes in feeding practices. Major causes of sow mortality are torsion of and accidents involving abdominal organs, cystitis and pyelonephritis, complications from parturition, and cardiac failure. Sudden death from \textit{Clostridium novyi} infection is a major cause of mortality in some European herds, but this condition has not been described in North America.

Analysis of the causes of sow mortality as an assessment of sow health has received little attention; and investigation of sow deaths and culling is lacking, particularly compared with investigation of mortality in lactating and growing pigs.\textsuperscript{1,2} The protracted nature of mortality studies in breeding herds plus the expense of performing a postmortem examination in sows means that only farms with a high mortality rate tend to be studied in detail by veterinarians. Pork producers are often unaware of the extent of sow losses and the main causes of mortality. Investigating the most frequent causes of death by performing numerous sow necropsies is of value in herds with a high mortality rate but rarely provides useful information when sow deaths are sporadic. The widespread use of computerized records of production data has made culling and mortality data readily accessible to clinicians and researchers and has allowed improved analysis of risk factors for high mortality in swine herds.\textsuperscript{3,4}

SURVEYS OF SOW MORTALITY

The variation in sow mortality rates among different surveys reflects differences in management, housing, geography, climate, diet, genotype, and disease status. Thus individual swine farm records and necropsies of deaths should be used to evaluate the causes of increased sow mortality. Nevertheless, the value of sow mortality surveys lies in the ability to detect general trends and potential risk factors and determine targets for breeding herds. Surveys of sow mortality can provide information regarding the age or parity and the stage of the reproductive cycle of dying sows and the seasonal distribution of mortality. Most studies conclude that the parity distribution of sow mortality can vary from farm to farm, depending on the principal cause of death. A majority of deaths occur in sows below parity 4, with the average parity at the time of death ranging from 3.4 to 4.3.\textsuperscript{5-8} A large survey (1996 to 1998) conducted in North America revealed that 27% of deaths occurred in sows that had never farrowed.\textsuperscript{9}

Various reports regarding sow mortality rates over multiple herds are available.

*When this article was written, Dr. Duran was at Michigan State University.
in the literature. In 1968, the annual mortality rate among 2488 sows in 105 British herds was 3.9%.\textsuperscript{10} This rate compared favorably with a rate of 4.1% a decade later, when the average herd size had increased almost threefold.\textsuperscript{11} This mortality rate was similar to the value reported in a Danish study during the 1970s (4.7%).\textsuperscript{12} The Danish data were from small herds (average size, 66 sows). Reports of the sow mortality rate in the past 15 years suggest that sow deaths are an increasing problem in breeding herds worldwide. An investigation into the reasons for sow removal, including culling and death, in Minnesota breeding herds found an average death rate of 5.7%, with individual farms suffering losses as high as 14.3%. The U.K. National Pig Survey for the years 1992 to 1993, involving approximately 5% of all pigs in the United Kingdom, indicated an average annual sow mortality of 5.3% (average herd size, 141 sows), with the less-productive herds averaging losses of 6.7%.\textsuperscript{13} Data from over 100,000 sows in 368 British herds (average herd size, 315 sows and gilts) indicated a mortality rate of 5.7%.\textsuperscript{14} Mortality was lower in surveys carried out in breeding herds in France (3.7%) and Quebec (3.3%), probably because the average herd inventory was lower than in other studies (150 sows).\textsuperscript{15,16} A Danish survey based on rendered sow carcasses estimated a mortality rate of 5% to 6%,\textsuperscript{17} which is similar to that recently reported for larger French breeding herds (average herd inventory, 225).\textsuperscript{3} In Australian herds, a 4.9% death rate and on-farm euthanasia of 3.3% of the sows have been reported.\textsuperscript{18}

The 1990 U.S. National Swine Survey estimated an 8.3% mortality rate based on 712 monitored farms,\textsuperscript{17} and a 1996 study of 28 U.S. herds (1993 to 1994) with an average herd size of 285 sows using PigCHAMP records reported a 7.4% annual sow death rate.\textsuperscript{18} Analysis of another database of North American herds between 1996 and 1998 revealed steadily increasing annual mortality rates, above 10% since 1997, with wide variation between different farms or systems in the database.\textsuperscript{9} Data from 350 farms during 1998 using breeding stock from one company revealed a mortality rate of 9%.\textsuperscript{19} Many individual farms experienced annual sow mortality rates as high as 15%. The mortality rate reported in all these recent studies surpasses previously suggested targets of 3% annual sow mortality for herds with 150 or fewer sows and up to 5% for herds with more than 200 sows.\textsuperscript{1} Thus targets for sow mortality rates apparently need to be reassessed in light of changing management practices, herd size, and genetics of breeding sows. Perhaps the action level for annual sow mortality should be raised to 8% to 10% in larger confinement breeding herds (1200 to 5000 sows) without losing sight of the fact that a sow mortality rate of 10% is an economic drain to the farm. The cost of sow replacement and opportunity loss has been estimated at $400 to $500 per sow in U.S. herds.\textsuperscript{9} High sow mortality is also a welfare problem and can affect farm worker morale.

This apparent rise in sow mortality during the past two decades has particularly been noted in herds with urinary tract infections.\textsuperscript{2,20,21} The increased incidence of ascending urinary tract infections has been linked to reduced exercise and confinement of sows and gilts to stalls. Increased mortality rates noted in some surveys may have resulted from changes in European Union welfare regulations prohibiting the transport of sick and injured animals, leading to an increased number of sows being culled on the farm and counted as deaths.\textsuperscript{7,15} Therefore, it is important when analyzing data from computerized recordkeeping systems to correctly define sow mortality, and sows culled on the farm should be compiled as a separate category.

Another factor implicated in the rising mortality rate could be the increasing number of deaths attributable to torsion and accidents involving abdominal organs.\textsuperscript{15,22,23} Earlier mortality surveys did not include these problems as a major cause of sow losses.\textsuperscript{10,12} More recent surveys in the United Kingdom, Canada, and Denmark have found torsion and accidents involving abdominal organs to be an increasingly common cause of sow death.\textsuperscript{7,21,22} Changes in management practices related to intensification of production (e.g., feeding once a day), increased sow excitement during feeding, and certain types of rations or the total volume of feed appeared to be involved in the pathogenesis of these deaths.\textsuperscript{15,22} Some reports indicated that sow mortality is elevated in individual herds having outbreaks of porcine reproductive and respiratory syndrome (PRRS),\textsuperscript{24} and abortion and sow mortality outbreaks have recently been linked to highly pathogenic viral strains isolated from herds with PRRS.\textsuperscript{25,26} In 1997, a survey of U.S. swine practitioners revealed that 138 herds fit the typical clinical presentation for this new syndrome.\textsuperscript{25} During outbreaks of the so-called sow abortion and mortality syndrome, mortality in some herds was estimated at 10% of the inventory during a 1- to 3-month period.\textsuperscript{25} Sows examined by necropsy presented with interstitial pneumonia, lymph node hyperplasia, meningoencephalitis, multifocal necrotizing hepatitis, and frequent retained near-term piglets and endometritis.\textsuperscript{26}

**RISK FACTORS**

The peripartum period is particularly propitious for unexpected sow mortality: Mortality occurred in 26% of the sows examined in one study\textsuperscript{2} and in over 40% in another\textsuperscript{3} in the period 3 days before and 3 days after...
farrowing. Other recent studies found that over half of sow deaths occurred in the farrowing house during the peripartum period and the subsequent 3 weeks. Cardiac failure, retained piglets, vaginal tears, peritonitis, and uterine prolapse (Figure 1) usually caused deaths at this time, particularly in higher-parity sows. In some herds, perforated gastric ulcers are apparently becoming a more frequently diagnosed cause of death during the lactation period.

The seasonal effect on sow losses has not been well investigated, but two studies described higher mortality during hot summer months and another detailed an epidemic of sow mortality related to very high daily maximum and minimum temperatures with high relative humidity. Conversely, the number of dead sows submitted for diagnostic investigation is often reduced in the summer. The effect of higher temperatures (above 28°C) on sow mortality probably results from heat stress leading to cardiovascular failure, particularly when moving sows, during breeding, and around farrowing.

A comparative analysis between indoor and outdoor production systems in the United Kingdom reported lower sow mortality in outdoor rearing systems, particularly in herds with higher productivity. In addition, early studies of mortality found that most deaths occurred in the winter when sows were housed, rather than when they were out on pasture. These findings suggest that increased sow mortality can be related to confinement housing but may be confounded by such other factors as higher sow inventory, feeding and management practices, and differing genotypes. One study indicated that herds housing dry sows in individual stalls had higher mortality rates compared with those using tethered systems. Various studies have reported that the risk of sow mortality increases with herd size.

Very few studies, however, have analyzed the controllable risk factors for sow mortality at the herd level. Because the mortality rate varies considerably among farms, it seems logical that certain management practices or herd characteristics would influence sow mortality. The following risk factors for high sow mortality in breeding herds were identified in a study of 102 larger breeding herds: Three feeding variables were significantly associated with high mortality—ad libitum feeding of lactating sows, once-daily feeding during gestation, and feeding of pelleted rations rather than ground meal during pregnancy. All these factors are likely to affect sow mortality rates because of increased deaths from abdominal organ torsion. The use of straw bedding appeared to provide some protection from gastrointestinal disorders, presumably because the sows had access to nondigestible fiber. In multiple-herd studies, wet feeding did not increase the risk of high sow mortality. Other management practices, including the use of artificial insemination, lack of quarantine, vaccination, multiple sources of replacement gilts, and batch farrowing, have not been associated with the risk of increased mortality. Weaning piglets at 28 days or more was associated with increased risk in a French study, contrary to study results from Danish herds. The risk of sow mortality increased with decreasing age at first farrowing and with reduced body condition score at the time of farrowing. According to one study that analyzed 3.6 million sow parities, sows with multiple stillbirths were also more likely to die during the postpartum period. Gilt multiplication farms breeding purebred sows have an increased risk of high mortality. Herd health factors correlating with higher mortality included increased prevalence of urinary tract disease, metritis, and sow lameness.

**CAUSES OF DEATH**

According to recent surveys, the most common causes of sow mortality are torsion and accidents involving abdominal organs, cystitis and pyelonephritis, and cardiac failure (Table 1); these causes account for over half of all mortality. Other less likely reasons include endometritis, uterine prolapse, various complications of farrowing, gastric ulceration of the pars oesophagea with severe hemorrhage, proliferative hemorrhagic enteropathy, and pneumonia. The criteria for making a particular diagnosis vary among surveys and may influence the incidence of conditions, such as cardiac failure, that are difficult to diagnose. Some surveys include locomotor failure as a cause of death, presumably after euthanasia on the farm. Locomotor problems are a major concern in modern large breeding herds, with euthanasia of affected sows accounting for 2% to 3% of the total wastage rate. The incidence of locomotor problems may be up to 10% or higher in...
problem herds; and the causes include downer sows, osteochondrosis, osteoarthrosis, posterior paralysis, septic arthritis, fractures, foot rot, and leg injuries.\(^{32}\)

In many instances, the cause of death cannot be determined at necropsy because of the poor condition of the carcass, presence of multiple pathologic lesions, or lack of detectable pathology. The percentage of undiagnosed cases varies from 8% to 16% among the studies.\(^{2,5,7,21,27}\)

**Torsion and Accidents Involving Abdominal Organs**

A majority of abdominal accidents involve gastric dilation followed by torsion, which may be accompanied by splenic torsion in approximately two thirds of cases. Other less frequent abdominal accidents include intestinal accidents (e.g., rupture, volvulus) and splenic and liver lobe torsion. Most gastric dilation deaths occur a few hours after feeding, and the sows are presented for necropsy with a grossly distended abdomen. Higher mortality has been reported in herds with sows housed in confinement stalls with slatted floors and once-daily feeding during gestation.\(^{22,33,34}\) Postmortem examination reveals a heavily distended stomach filled with fluid, partly digested food, and gas, often with a

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**TABLE 1**

**Main Causes of Death in Sows from 12 Studies of Sow Mortality**

<table>
<thead>
<tr>
<th>Year</th>
<th>Country</th>
<th>No. of Herds (study duration)</th>
<th>No. of Sows Necropsied</th>
<th>Causes of Death (% of sows necropsied)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1967(^{28}) England</td>
<td>1 (1 yr)</td>
<td>36</td>
<td>Cystitis/pyelonephritis (21.7%)</td>
<td>Peritonitis (8.7%)</td>
</tr>
<tr>
<td>1968(^{10}) England</td>
<td>106 (1 yr)</td>
<td>81</td>
<td>Complications of parturition(^a) (16%)</td>
<td>Cystitis/pyelonephritis (14.8%)</td>
</tr>
<tr>
<td>1975(^{12}) Denmark</td>
<td>9 (4 yr)</td>
<td>109</td>
<td>Locomotor problems (25.5%)</td>
<td>Cardiac failure (14.9%)</td>
</tr>
<tr>
<td>1980(^{29}) England</td>
<td>1 (2 yr)</td>
<td>69</td>
<td>Torsion of abdominal organs(^b) (21.7%)</td>
<td>Cystitis/pyelonephritis (8.7%)</td>
</tr>
<tr>
<td>1984(^2) France</td>
<td>52 (1 yr)</td>
<td>88</td>
<td>Cystitis/pyelonephritis (42%)</td>
<td>Metritis (17%)</td>
</tr>
<tr>
<td>1984(^{21}) Scotland</td>
<td>6 (1 yr)</td>
<td>102</td>
<td>Cystitis/pyelonephritis (29.3%)</td>
<td>Torsion of abdominal organs(^b) (14.7%)</td>
</tr>
<tr>
<td>1991(^{27}) Canada</td>
<td>NA (7 yr)</td>
<td>426</td>
<td>Cardiac failure (22.1%)</td>
<td>Torsion of abdominal organs(^b) (18.3%)</td>
</tr>
<tr>
<td>1991(^5) Canada</td>
<td>24 (1 yr)</td>
<td>116</td>
<td>Cardiac failure (31.4%)</td>
<td>Torsion of abdominal organs(^b) (15.3%)</td>
</tr>
<tr>
<td>1994(^{23}) Canada</td>
<td>23 (1 yr)</td>
<td>38</td>
<td>Torsion of abdominal organs(^b) (47.4%)</td>
<td>Gastric ulcer (18.4%)</td>
</tr>
<tr>
<td>1996(^{15}) Denmark</td>
<td>NA (2 yr)</td>
<td>598</td>
<td>Locomotor problems (21.6%)</td>
<td>Torsion of abdominal organs (19.4%)</td>
</tr>
<tr>
<td>1996(^{30}) England</td>
<td>4 (5 yr)</td>
<td>170</td>
<td>Clostridial hepatopathy(^c) (34.7%)</td>
<td>Cystitis/pyelonephritis (22.9%)</td>
</tr>
<tr>
<td>1998(^7) England</td>
<td>3 (3 yr)</td>
<td>102</td>
<td>Torsion of abdominal organs(^b) (17.6%)</td>
<td>Clostridial hepatopathy(^c) (16.7%)</td>
</tr>
</tbody>
</table>

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\(^{a}\)Include uterine prolapse, retained piglets, vaginal tears, and acute illness around farrowing.

\(^{b}\)Includes torsion and other accidents involving abdominal organs.

\(^{c}\)Refers to sudden death caused by *Clostridium novyi* infection.

NA = not available.
180° or 360° clockwise torsion of the stomach and sometimes the spleen (Figure 2). Rupture of the twisted spleen with subsequent hemorrhage has also been described. Reports suggest that the cause of these deaths was extreme agitation of the sows at feeding time. Sows can be observed plunging at the food trough, rearing up, salivating profusely, and chewing at the stall bars while waiting to be fed, then rapidly ingesting a large volume of feed. Factors implicated in increased gastric torsion include disruption of the feeding routine, once-daily feeding, and presentation of feed in a pelleted form. One report suggested a genetic predisposition for gastric dilation and torsion, similar to that seen in deep-chested canine breeds. Examination of breeding records in this herd revealed that all sows with gastric torsion were the progeny of one Landrace boar; new cases ceased after the boar was removed from the herd. Epidemiologic data evidently suggest that thinner, hungrier sows at peak production after the third pregnancy are predisposed to torsion and other accidents involving the abdominal organs. Gastric torsion with distention has been diagnosed in sows during all stages of production and even in gilts held in quarantine. Liver lobe torsion and torsion of the spleen without gastric involvement have been reported less frequently. Splenic torsion probably occurs with relative ease because of the loose, long gastrosplenic ligament found in pigs. Intestinal volvulus with obstruction is more common in growing pigs and breeding sows kept outdoors. Sporadically, sows with torsion of the mesentery around a heterotopic (ectopic) bone have been described. The mesentery can twist around an ectopic bone that acts as a fulcrum, causing a 360° torsion. Ectopic bone formation is believed to result from trauma, hematomas, hemorrhage, or scar tissue formation with subsequent ossification. The bone may also cause adhesions to the intestine and obstruction or may be an incidental finding at necropsy. Intestinal rupture has been described in sows fed whey after excessive intestinal fermentation caused overdistention and rupture of the ileum or the large intestine followed by acute peritonitis.

**Cystitis and Pyelonephritis**

Most surveys have found cystitis and pyelonephritis to be principal causes of sow mortality, with the incidence varying between 7% and 40% of all deaths. The disease has become well characterized in recent years as an ascending bacterial infection that begins with hemorrhagic ulcerated cystitis, alteration of the ureterovesical junction leading to ureteric dilation, and ulcerated necrotizing ureteritis with hemorrhages plus severe pyelonephritis (Figure 3). Results from cases of cystitis/pyelonephritis had a mean aqueous humor urea concentration of 52.3 mmol/L compared with 9.9 mmol/L for sows that died from other causes—a statistically significant difference. These findings were echoed by a large Danish study, although concentrations of urea higher than baseline were also found in dead sows with intoxication or central circulatory failure without macroscopic renal lesions.

Analysis of the aqueous humor taken from dead sows up to 24 hours after death is well correlated with the antemortem serum concentration of urea. Aqueous humor can be collected by aspirating gently with a 16-gauge needle inserted under the cornea, avoiding contamination with blood or cellular debris. The causative agents most frequently isolated in cases of cystitis and pyelonephritis are *Eubacterium* (formerly *Corynebacterium*) *suis*, *Escherichia coli*, and *Proteus* species; but other bacteria (e.g., *Klebsiella* species and *Enterococcus faecalis*) have also been isolated. Recently, the nomenclature for *Eubacterium suis* was reclassified as *Actinobaculum suis*. Various predisposing factors are...
believed to allow bacterial colonization and subsequent ascending infection: damage to the urethra during mating, reduced water intake, infrequent urination with incomplete emptying of the bladder, and high urinary pH. Cystitis/pyelonephritis was more prevalent in older sows in some surveys but has been reported in maiden gilts and younger sows (average parity, 2.4 litters) in two other studies. Obesity, lameness, and lack of exercise have been implicated as predisposing factors for a urinary tract infection in sows. Most cases occur during the postservice period, and frequently clinical signs (e.g., hemorrhagic/mucopurulent vulval discharge, pain, anorexia) are noted before death. Boars have been implicated as a source of infection to sows, and A. suis has been isolated from the preputial diverticulum of boars and the environment of breeding pens. Recognition that adequate supply and intake of water for sows are valuable as preventive measures has reduced the incidence of urinary tract disorders in many herds, but cystitis/pyelonephritis continues to be a major cause of sow death worldwide.

Cardiac Failure
Cardiac failure has been reported as the most common cause of sow mortality in Canadian herds and in approximately 12% of sows examined in a Scottish survey of sow mortality. Death usually follows events (e.g., mating, transport, fighting, parturition) that increase the demands on the cardiovascular system. Other environmental and management factors that may predispose sows to cardiac failure are high ambient temperature, halothane-positive genetic stock, lack of cardiovascular fitness, and obesity. In fact, more than 60% of deaths caused by cardiac failure occurred during the peripartum period, and this time was even more dangerous for sows when combined with heat stress. Age apparently is not directly correlated to increased risk for cardiac failure; but sows that died from cardiac failure were heavier, fatter, and had a smaller heart-weight: body-weight ratio than did those dying of other causes. Cardiac failure can only be definitively diagnosed after excluding other possible causes of death and if a suitable clinical history of a stressful event immediately before sudden death is available. This may explain the lower numbers of deaths attributed to cardiac failure in several mortality studies. Lesions indicative of cardiac failure are transudate in pericardial, thoracic, and abdominal cavities; dilation of cardiac chambers; pulmonary edema; and passive congestion of internal organs. 

Clostridium novyi Infection
Sudden death in sows after Clostridium novyi proliferation (clostridial hepatopathy) is not listed in many surveys of sow mortality and has not been described in studies in North America. However, various cases of C. novyi sudden death have been reported in intensive swine-breeding units in Europe. C. novyi is an obligate anaerobe and causative agent responsible for gas gangrene in humans and infectious necrotic hepatitis (Black disease) in sheep, cattle, goats, and horses. Presumptive diagnosis of this disease in sows is based on the absence of any other detectable cause of death, a gas-filled necrotic liver, and rapid decomposition and tympany of the carcass (Figure 4). The diagnosis is confirmed in tissue smears using the C. novyi fluorescent antibody test (Figure 5). Seventeen clostridial hepatopathy cases were described in one study of three intensive breeding herds. Sporadic reports in the early literature remarked on the similarity between the gross pathology of sows with C.
Infection and the findings in anthrax cases. All sows with clostridial hepatopathy had generalized subcutaneous edema and gas infiltration with copious, foul-smelling, bloody fluid in the pericardial, pleural, and abdominal cavities. The liver was enlarged, friable, and dark, and the parenchyma uniformly infiltrated with gas bubbles, thereby presenting a spongy appearance on the cut surface. The *C. novyi* sudden death cases presented a remarkable degeneration of the liver, regardless of the time elapsed between death and examination of the carcass. Both *C. novyi* types A and B have been isolated from the liver and spleen of clostridial hepatopathy cases, although bacterial culture is rarely attempted because of the strict anaerobic and cultural requirements of this organism.

It is essential to perform the postmortem examination and collect samples as soon as possible after death. Demonstration of *C. novyi* in the liver of sheep or sows dead for more than 24 hours does not alone constitute sufficient evidence of infectious necrotic hepatitis or clostridial hepatopathy. Difficulties determining whether necropy findings constitute postmortem degeneration, particularly in the summer, probably has caused underreporting of this cause of sow mortality. In temperate weather, liver smears from sows culled on the farm and left unopened for 48 hours before necropy gave a positive result to the fluorescent antibody test, but sows left unopened for 24 hours did not. Examination of healthy slaughterhouse and cull sows revealed dormant *C. novyi* spores in the liver.

The pathogenesis of *C. novyi* sudden death in sows has not been elucidated. In sheep with infectious necrotic hepatitis, previous damage to the liver parenchyma, usually by migrating liver flukes, is required for proliferation of *C. novyi*. Most descriptions of the disease in sows have not found lesions of parasitic or larval migration in the liver and indeed have not described necrotic or hemorrhagic foci on the liver. Furthermore, many cases originate in herds free of intestinal parasites. Interestingly, *C. novyi* was identified in the liver of three gastric torsion cases and from the damaged abdominal wall of another sow, suggesting that the intestinal tract may be the source of *C. novyi*. Some studies have reported that in clostridial hepatopathy cases, other concomitant low-grade infectious processes (e.g., metritis, cystitis) may predispose sows to proliferation of the organism in the liver and production of the potent exotoxins responsible for the severe necrotizing and edematous damage. Older sows (mean parity, 5.6 litters) in good body condition and during gestation were more likely to die suddenly from *C. novyi* infection. Mortality attributable to *C. novyi* in a 700-sow herd occurred during lactation and did not seem to be correlated with the incidence of gastric ulcers or composition of the diet or water quality. In many outbreaks, control of mortality attributed to *C. novyi* has been cost-effective by including zinc bacitracin (200 to 260 ppm) in the sow feed during periods of increased risk. Vaccination of the sows at risk with a multivalent clostridial vaccine does not seem to be an effective control measure.

**CONCLUSION**

Increased sow mortality rates have paralleled increases in swine productivity and larger average herd size during the past 20 years. The different causes of unexpected sow death should be considered to apply the necessary control measures, which must be decided on a farm-by-farm basis. Investigation into elevated mortality in a herd must include examining herd records, determining the impact of death on production, performing multiple necropsies over time, and determining potential risk factors in the herd. As farms become larger and veterinary consultants travel farther to visit herds, mortality problems are likely to become more difficult to resolve. Veterinarians and laboratory diagnosticians need to be aware of the most common causes of death in sows, including *C. novyi* infection, although it has not been reported in North American herds.

**REFERENCES**

1. Which of the following percentages would be the accepted target for annual sow mortality rate in a 500-sow breeding herd?
   a. 2%  
   b. 5%  
   c. 10%  
   d. 48%

2. Sow mortality surveys conducted in various countries during the past 20 years revealed that
   a. the health of modern sows has improved steadily.
   b. sow mortality is an increasing problem in confinement operations worldwide.
   c. the causes of sow death have not changed over time.
   d. sow mortality is decreasing as breeding herds increase in inventory.

3. Which of the following feeding practices have been identified as risk factors for increased sow mortality?
   a. feeding to appetite during the lactation period
   b. wet feeding sows during lactation
   c. feeding rations prepared with by-products
   d. feeding sows twice daily during pregnancy

4. Which herd health factor correlates most with increased sow mortality?
   a. use of quarantine facilities for incoming gilts
   b. use of vaccines during pregnancy
   c. high incidence of urinary tract infections
   d. none of the above

5. The most common cause of death in sows involving abdominal organs is
   a. liver torsion.
   b. lymphosarcoma.
   c. intestinal volvulus.
   d. gastric dilation and torsion.

6. Cardiac failure is most likely to occur during
   a. gilt development.
   b. the peripartum period.
   c. midgestation.
   d. weaning.

7. A producer reports that a sow is off feed, seems agitated, and has a bloody vulval discharge. When you arrive at the farm, the sow has died. You suspect a urinary tract infection. In addition to characteristic findings at necropsy, which test will confirm the diagnosis?
   a. determination of the urea concentration of the aqueous humor
   b. bacterial culture of urine from the bladder
   c. urinalysis
   d. none of the above

8. After diagnosing cystitis and pyelonephritis in a herd with a 10% annual sow mortality rate, you convince the producer that control measures need to be implemented. What is the most successful preventive measure you could advise?
   a. cull older, heavier sows
   b. improve supply and availability of water to sows
   c. medicate the entire herd with in-feed tetracycline (10 g/lb)
   d. supply sows with drinking water containing a urine acidifier

9. Clostridial hepatopathy can be presumptively diagnosed when no other cause of death can be identified for a bloated edematous and gas-infiltrated carcass. What testing can improve diagnosis of this disease?
   a. polymerase chain reaction of liver tissue
   b. anaerobic culture
   c. histopathology of affected tissue
   d. C. novyi–specific fluorescent antibody test of liver smears

10. When a sow dies from pyelonephritis, the postmortem aqueous humor urea concentration
    a. cannot be accurately measured.
    b. is higher than that in sows dying free of kidney disease.
    c. is lower than that in healthy sows.
    d. does not statistically differ from that in healthy sows.