



pork industry handbook

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Lactation Failure in the Sow

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Lactation failure in swine (in the past commonly referred to as "MMA" or "mastitis, metritis, agalactia") is a disease syndrome that results in the death of baby pigs through starvation or increased susceptibility to other diseases of the newborn. It is referred to as a syndrome to indicate that there may be several causative factors and clinical diseases that are referred to collectively as lactation failure. Because of the economic impact, lactation failure is of major importance to the pork producer.

The usual swine agalactia syndrome occurs in the very early stages of lactation. If the herdsman or attendant has been trained to recognize the signs, the disease may be evident to him at farrowing. Under the title of lactation failure, a sow can have a total lack of milk production (agalactia); but more frequently, the sow has only a reduction in normal amounts of milk secreted (hypogalactia). This hypogalactia or reduced milk production may be more difficult to recognize because piglets grow at a slower rate with only occasional deaths. The number of sows or gilts affected with this disease syndrome during a farrowing period in a given herd varies from 0% to 100% with an average of 13.1%.

Signs and Symptoms

Lactation failure can be present at farrowing time or can appear within several days after parturition. Although some hypogalactic sows are clinically normal, signs observed in affected females may include rapid breathing, depressed attitude, lack of desire to eat or drink, fever, reluctance to move about, and refusal to allow nursing. When the herdsman observes uncomfortable baby pigs, he should realize this indicates a need for closer evaluation of the sow.

Mastitis, an inflammation of the mammary glands usually caused by bacterial infections, is a frequent component in this disease syndrome. Although earlier reports have suggested that abnormalities or disease were present in other organs such as the uterus, ovaries, or adrenals, it is now agreed that the mammary glands of the sow or gilt are the primary location of the problem. The degree of agalactia varies from only one gland to the entire udder. The affected mammary glands are usually enlarged, more firm, warmer, more sensitive, and often discolored when compared to the other glands although hypogalactic sows may experience little or no change. Careful palpation of the mammary glands of each sow several times within the first 12 to 36 hours after farrowing may reveal developing hypogalactia and allow for treatment early in the syndrome. In some sows, the affected glands may show typical signs of regression similar to that seen at weaning. In these instances, the glands probably are not mastitic. Palpation prior to farrowing can be helpful in detecting sows with excessively hard or edematous ("caked") glands. The presence of purulent discharge (pus) from the birth canal does not necessarily indicate that the sow or gilt has a uterine infection (metritis). Studies have revealed that over 60% of clinically normal sows have a vulvar discharge. Postmortem examinations of sows having agalactia indicate metritis is very infrequent in this disease condition.

Tissue changes within the affected sow are variable because of the different causative agents or management factors involved. Lactation failure can be a primary disease or secondary to other diseases. The majority of affected animals will reveal abnormal mammary gland tissues. The changes may be characteristic of mastitis, or a partially functional gland, without mastitic changes or be undergoing complete regression.

The area of mastitis within a gland is frequently small and has led to the comment that the degree of mastitis could not produce the clinical signs seen in the sow or the starvation in the baby pigs. The partially functional or nonfunctional gland looks and feels very similar to the gland with mastitis, making an accurate diagnosis difficult. This decrease in milk secretion is believed to be the result of some alteration in the hormonal sequence of events necessary for normal mammary gland secretion. The hormones primarily involved in mammary gland development and function (estrogens, progesterone, and prolactin), along with a number of direct and indirect synergistic hormones, are essential to full development and secretion of milk by the mammary glands. Each of these hormones must be present at the right time in the right amount to initiate and maintain lactation. Anything altering the levels of the hormones—such as, environmental stress, poor nutrition, bacterial endotoxin, or improper preventative injections—can decrease lactation. Other body tissue changes usually involve swelling, redness, and possibly hemorrhages in tissues around and in the mammary glands (including adjacent lymph glands), kidneys, synovial membranes, adrenal glands, and in some instances, the pituitary gland.

The precise role of bacterial endotoxin in the lactation failure syndrome still needs to be clarified. Endotoxin is a large lipopolysaccharide molecule released from the cell wall of bacteria such as *E. coli* after they die. This endotoxin can be readily absorbed from the mammary gland into the blood vascular system. Endotoxin can cause many changes in pigs, including fever, increased adrenal corticoid secretion, and in early lactation, a decrease in prolactin secretion.

The actual causes of agalactia have been narrowed from the 30-plus previously stated in the literature. Mastitis caused by *E. coli*, *Klebsiella*, or related bacteria is common in affected sows. Numerous factors can be involved as contributors of stress that allow mastitis to develop, sufficient endotoxin to be present, or an endocrine (hormonal) imbalance to occur, any one of which may decrease mammary gland function. It is not known if genetics has an important role in lactation failure, but stress-susceptible and stress-resistant sow lines have been identified, and stress-susceptible animals seem to have more lactation problems than the stress-resistant animals.

Diagnosis

Diagnosis of the lactation failure is usually not difficult. Proof of the cause, however, is usually difficult. Agalactia must be differentiated from other diseases such as transmissible gastroenteritis (TGE) or pseudorabies (Aujeszky's or PRV) to allow for effective treatment and/or control measures to be instituted. Evaluation of history, observation of clinical signs, palpation of the mammary glands, and postmortem examination of one or several pigs will usually allow for a definitive diagnosis.

The culturing of milk samples should be considered and, if done correctly, can reveal valuable information about a particular sow herd. Particular attention should be given to coliform bacteria. Although treatments should not be postponed until culture results are obtained, they can be changed after culture results are known if the initial treatment is not appropriate for causative organisms. Other clinical tests on an individual sow will not sufficiently enhance diagnostic efforts to be economically justified. When the disease involves many sows, extensive test-

ing is justified to determine preventive measures and eliminate other infectious diseases. In the future, more rapid analytical methods for determining endotoxin and endocrine concentrations may be available at affordable costs.

The attending veterinarian is in the best position to coordinate the diagnostic procedures in a particular herd, particularly if the veterinarian is involved routinely in the overall management of the swine production unit. The herdsman and the veterinarian should work together to characterize a particular situation completely and rapidly and develop a program of corrective action. An accurate diagnosis leads to effective treatment(s), the initiation of possible preventive measures, and the ability to make an accurate prognosis.

Prognosis

The prognosis for the life of the affected sow or gilt is good; however, the prognosis for the complete return or establishment of normal lactation is guarded. The affected sow usually recovers in 2 to 5 days with or without return of lactation. Although having the disease once does not mean the sow will develop agalactia on subsequent farrowings, culling the sow should be considered if it has several hard, nonfunctional mammary glands after recovery. Unless lactation is rapidly reestablished or supplemental feeding is successful, the chances for baby pig survival are low. The effects of chilling, secondary diarrhea, and other neonatal diseases must be fully considered in deciding on the probability of survival of individual piglets.

Treatment

Treatment of the affected sow must be directed toward establishing milk flow to aid in sustaining the life of the baby pig. No other single treatment is superior to oxytocin, which expresses or releases the milk within the mammary glands which can then be taken by the hungry pigs if they are strong enough to nurse. The principle involved is no different from the often recommended frequent milking of the mastitic dairy cow for "clean out" purposes. Multiple injections of oxytocin may be necessary to provide milk for the pigs. Because the biological half-life of oxytocin is 6 to 7 minutes, injections can be given every 2 to 4 hours, repeated over a 24-36 hour period, without being harmful to the sow. Approximate dosages of 5 to 10 units should be used. If the oxytocin preparation is 20 units/ml, then 0.25-0.5 ml should be injected per treatment. Injecting oxytocin in that manner would coincide, in part, with the normal suckling pattern of newborn pigs. Although oxytocin is the single superior treatment choice, there are some agalactic sows and gilts that respond poorly, or not at all. Reasons for the lack of response are related to the cause and effect of the disease within the individual group of sows. Oxytocin can only cause release of milk that has been secreted and is present in the alveoli of the mammary glands. Research results have proven that many mammary glands are not functional and no milk is present to be released.

Antibacterial drugs should be used in treating the affected sow since there is no immediate method for determining the presence of bacterial pathogens. Use of antibacterials does increase producer costs and forces withholding treated animals from slaughter. The added economic burden is justified because of the high prevalence of bacterial infections of the mammary glands.

Since it is now clear that bacterial endotoxins can suppress prolactin secretion and lactation, reduction of coliform bacterial populations and correction of prolactin deficiencies are indicated. A major difficulty is that drugs, such as phenothiazine tranquilizers, which release prolactin in other species, are not effective in the lactating sow. Thyrotropin-releasing hormone (TRH) does stimulate prolactin release in lactating sows, but it is not very effective when used to counteract the effect of endotoxin on prolactin secretion. Similarly, estrogen injections have not been proven effective in increasing prolactin secretion. Although tranquilizers apparently are not effective in stimulating prolactin release, they may be clinically useful in quieting excitable sows and improving the normal suckling response.

Because endotoxin also causes large increases in prostaglandin secretion, administration of antiprostaglandin drugs such as flunixin meglumine may be helpful. Data are not available to confirm that this treatment would restore prolactin secretion, but reduction of prostaglandin release should improve the general status of the sow. Corticosteroids also are used frequently and may have supportive benefit. It must be remembered that bacterial endotoxin invariably stimulates a very pronounced rise in corticosteroid secretion. In such cases, additional corticosteroids may have limited benefit.

The use of vaginal or uterine infusions, douches, or pessaries to combat suspected uterine infections may stimulate a neurohormonal reflex action that could result in the release of oxytocin from the posterior pituitary gland. However, this action is not different from injecting oxytocin and, in the opinion of the authors, should not be done because of the possibility of contaminating the reproductive tract with pathogenic bacteria.

Treatment programs should usually include supplementary dietary support for the pigs because they have small energy storage capabilities which make their food intake critical for sustained life. Supplemental heat (85 to 90°F) for the baby pigs during the first few days is energy-conserving to them and will aid in survival as a part of routine treatment or prevention. Use of a hover can aid in holding this heat in the piglet area.

Prevention

Prevention should center around herd health management and nutrition. The veterinarian should be thoroughly familiar with the total management practices of a particular farm before attempting to offer suggestions for prevention of lactation failure in sows. The veterinarian who is consulted only when a sow is ill and is not

familiar with the total breeding and farrowing program is handicapped in offering constructive suggestions.

Immunization procedures, if feasible, must be done in advance of anticipated problems such as bacterial mastitis. The bacterial cultures from the milk of infected sows can be used to prepare a bacterin. Those herds, where bacterial mastitis occurs frequently, may benefit from immunization if other management practices will not control the problem.

Efforts to reduce stress throughout gestation, especially near parturition, are important preventive measures. An example would be sow acclimatization to the farrowing facility. Use of a new or remodeled farrowing house for the first time may initiate lactational failure in some sows.

Gestational feeding of sows and gilts has an influence on incidence of agalactia. The underfed sow apparently cannot maintain blood glucose levels as well as the adequately fed sow; therefore, resistance may be lower. Supplementation of the sow ration with vitamin E and selenium may be helpful in geographic regions known to be deficient in selenium. Pork Industry Handbook fact sheet PIH-23, concerning gestation and lactation rations, should be consulted. Laxative materials such as wheat bran or beet pulp can be included in the diet if constipation is a problem. Additives to reduce mammary gland edema might be considered in some herds. The mixture of 12 parts potassium nitrate NF, 4 parts methenamine USP, and 1 part dicalcium phosphate by weight, given at the rate of 1 ounce twice daily 1 week prior to and 1 week following farrowing may help reduce such edema.

Management attention to controllable environmental factors in the farrowing house is critical. High humidity and temperature around the sow or gilt should be avoided. Use of sprinklers or other cooling devices can be of help during the summer. Provisions should be made for heat lamps or heat pads for the baby pigs; but keep heat lamps and pads away from the sow at all times. Maintenance of clean, dry floors during late gestation and parturition can reduce bacterial populations.

Preventive research efforts related to various drugs and hormones (fed and/or injected) prior to farrowing have occasionally produced excellent clinical results. However, critical evaluations and continued study prove that what works in one herd may have no value in another.

Lactation failure in sows is a complex disease syndrome requiring much consideration and effort to produce acceptable results in prevention and/or treatment within the industry. Research efforts are continuing, and newer knowledge will be reported when available.



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