

Embryonic and Fetal Mortality in the Porcine Species

L. E. Evans, D.V.M.*

The occurrence of embryonic and fetal mortality in the porcine species has continued to challenge those pursuing its epidemiology. While sporadic and endemic field cases are continually studied, a pantoscopic approach to this problem is difficult to achieve. This failure is due partly to the fact that multiple etiological agents are involved as well as the fact that less than 50% of the abortions and stillbirth cases are properly identified with a recognized etiological agent.

Basically one of four phenomena follows the death of an embryo or fetus. If death occurs early, resorption of the embryo usually follows. Abortion may occur anytime after early pregnancy with the expulsion of a live or a dead fetus. If death occurs between 35 and 90 days, mummification is a common sequela. Fetal death after about 90 days post-conception usually terminates as a stillbirth.

Probably the least perceptible form of embryonic or fetal death is that which results in resorption of the embryo. The period from 1 to 30 days post-conception is generally when the greatest fetal mortality occurs. Roberts (1962) mentions that 20 to 80 per cent of the ova shed may die during this period. Abnormalities of the ova or the spermatozoa may result in death of the zygote after fertilization. Abnormal development of the blastocyst may lead to death at this stage of pregnancy. Genetic lethals may be a cause of death at this time also. The chemical and patho-

logical changes that accompany uterine infections may be uncomplimentary to embryonic life. Hyperthermia often results in a high embryonic mortality. Therefore, any disease condition that produces extreme pyrexia may contribute to embryonic death and resorption. Embryonic resorption may go unrecognized or manifest itself by repeat breeding problems and extended anestrus.

Abortions in the porcine species are more discernible than embryonic resorption or mummification since the fetal membranes as well as the fetuses are expelled. Frequently estrus is observed in three to five days after an abortion. The pathogenesis of abortions is ambiguous and in many cases is not known. Competent diagnosis in the laboratory is often complicated by inadequate or contaminated specimens.

Acute infections resulting in abortions or stillbirths of nearly normal fetuses are usually bacterial in origin. These fetuses frequently exhibit petechial hemorrhages throughout the body. The following bacterial agents are commonly listed as causes of abortion or stillbirth in swine:

- Streptococci
- Staphylococcus
- Escherichia coli
- Leptospira
- Brucella

The significance of low grade infections of Streptococci, Staphylococcus and Escherichia coli is not well established since these bacteria have been isolated from both normal and abnormal animals.

* Dr. Evans is an instructor in the Department of Veterinary Clinical Sciences.

Fennestad (1955) reported an isolated case of abortion at 87 days pregnancy caused by *Staphylococcus aureus*. Fennestad later identified *Staphylococcus aureus* as a cause of returning to heat, abortions, and metritis in sows. This problem was traced to coital infections originating from a boar with a bladder abscess.

Thorne and Wilson (1961) confirmed reports of *Staphylococcus* causing abortion in swine. Saunder (1958) has isolated hemolytic *Escherichia coli* as well as beta and alpha streptococci from cases of swine abortions as a cause of returning to heat, isms as the cause of swine abortions is low and probably account for sporadic cases of abortion more frequently than herd problems.

Brucellosis in the pregnant sow may cause abortion anytime in the gestation period. Sows with a higher degree of resistance may carry the pigs to term but there is a weakness and a high mortality rate among the newborn. Normally a sow will recover sufficiently to produce subsequent litters. Uterine infections usually do not persist, but in a few animals a uterine discharge continues and serves as a possible source of herd infection. A positive diagnosis is based upon isolation of the *Brucella* organisms from the aborted fetus or uterine discharge. Frequently a diagnosis of brucellosis is dependent upon demonstrating a significant increase in serum antibodies to *Brucella* organisms. The results of an individual serum test is much less reliable than a herd test.

Leptospirosis has been characterized as causing abortion and stillbirth in the pregnant sow. Infection caused by *Leptospira pomona* are associated with abortion within the last month of pregnancy or weak pigs at birth. Diagnosis is usually based upon isolation of the organism or a demonstrable rise in serum antibody titers approximately two weeks after the acute infection. Michna (1962) reported that *Leptospira canicola* can cause abortion in sows pregnant five weeks or less or the birth of weak pigs if infected at mid-pregnancy or later.

Erysipelothrix insidiosa, *Corynebacteria* spp., *Salmonella* spp., and *Pasteurella*

have been isolated from aborting sows. These abortions are probably due to septicemia.

Toxic agents often mentioned as a cause of abortion are dicoumarin, estrogens and nitrates. Estrogens are sometimes accidentally added to the feed or produced by certain molds growing in the feed. The effects of high nitrate diets in pregnant sows have not been well determined.

Viral agents may induce abortion in swine but more commonly they manifest fetal death in the form of mummified fetuses and stillbirths. As mentioned earlier mummification may follow the death of 35 to 90 day fetuses. After fetal death there is a condensation of tissue aldehydes to form phenolic agents which preserve the fetus and the fetal membranes. The culturing of mummified fetuses have in general yielded few bacterial or viral agents.

In swine several viruses are known to cause fetal death, delayed growth or fetal abnormalities. Attenuated hog cholera, vesicular exanthema, pseudorabies, and certain enteroviruses (including T.G.E.) are known to cause these changes. Swine influenza appears to be able to cause similar results in the pregnant sow.

As early as 1950 Young recognized a connection between influenza virus and pregnancy abnormalities in swine. Young reported that a human (Weiss) influenza virus was a factor in the abortions of 21 of 29 gilts. Young et al. (1955) demonstrated that attenuated hog cholera vaccines could produce fetal death with resorption or mummification. The vaccine may also cause fetal abnormalities if given between the fifteenth and twenty-fifth day. The most common abnormality was edema of the fetus. Inoculation before 10 days gestation resulted in embryonic resorption. Inoculation with attenuated hog cholera virus after 15 days gestation resulted in 38% of the fetuses to show abnormal development and 43% to be in various stages of mummification.

Most recently Dunne et al. (1956) isolated enteroviruses which were called SMEDI. These belong to the picorna group

of viruses. The inoculation of susceptible gilts approximately 5 days pregnant with SMEDI viruses resulted in embryonic and fetal death. The result of this embryonic or fetal death was complete or partial resorption. When embryonic death occurred remnants of the placental membranes could be recognized. Mummification and many times delayed parturition followed viral inoculation after the first trimester. The embryonic or fetal death was simultaneous or a progressive infection passing from fetus to fetus. Late infections usually resulted in stillbirths and weak pigs. Virus isolation was best accomplished in the live or recently dead fetus. Perivascular cuffing, edema, and endothelial degeneration was the pathology noted in the live or recently dead fetus.

Abnormalities in nidation or pregnancy accidents such as torsion of the umbilical cord cause sporadic cases of mummification or stillbirths.

The causes of stillbirth in the porcine species would include many of the viral agents mentioned previously. Dystocias and farrowing accidents likewise account for sporadic cases. Many of the nutritional deficiencies known to interfere with reproduction, cause stillborn or weak pigs. Vitamin A, calcium, iodine and animal protein deficiencies are most commonly referred to in this respect.

Many of the above mentioned diseases and conditions need further investigation. Other agents that have been or are currently being studied are aspergillosis, toxoplasmosis and mycoplasmosis. All these have been described as causing reproductive problems in domestic species. Mycoplasma has been isolated from genital infections in the bovine species and in the human. At present Mycoplasma has not been described as an agent in embryonic or fetal death in swine.

Embryonic and fetal mortality in swine has a multiplicity of etiological agents. In a given herd one or several agents may be

responsible for reproduction failures. Due to this complexity, careful consideration must be given to preventive medicine programs.

Vaccination and testing programs are an important part of preventive medicine. Isolation and acclimation of new breeding stock should be encouraged. Careful scrutiny of management and nutrition are also important.

Since many reproductive failures are approached in retrospect, an accurate diagnosis is not always possible. A knowledge of possible causes may aid in the prevention of further trouble.

REFERENCES

- Dunne, H. W.: Abortions and Stillbirth In Diseases of Swine Iowa State University Press, Ames (1964): 646.
- Dunne, H. W., Gobble, J. L., Hokanson, J. F., Kradel, D. C., and Bubash, G. R.: Porcine Reproduction Failures Associated with a Newly Identified "SMEOIN Group of Picorna Viruses. A.V.R. 26, (1965): 1284.
- Dunne, H. W., and Hokanson, J. F.: A Modern Approach to Abortion and Stillbirth Problems in Swine. Proc. Book A.V.M.A., (1963): 54.
- Fennestel, K. L., and Moller, T.: Staphylococcus aureus as a Cause of Reproductive Failure and so-called Actinomycosis in Swine. Nord. Vet. Med. 7 (1955): 929.
- Hartman, H. A., Tourtellotte, M. E., Nielsen, S. W., and Plastringe, W. N.: Experimental Bovine Mycoplasmosis. Res. Vet. Sec 5, (1964) 303.
- Hirth, R. S., Plastringe, W. N., and Tourtellotte, M. E., Nielsen, S. W.: Genital Mycoplasmosis in Cattle and Man. JAVMA 148 (1966).
- Michna, S. W.: Abortions in the Sow Due to Infection by Leptospira canicola Vet. Rec., 74, (1962): 917.
- Morrisette, M. C., McDonald, L. E., Whatley, J. A., and Morrison, R. D.: Effects of Progesterone on Embryonic Mortality of Swine. A.J.V.R., 24 (1963): 317.
- Roberts, S. J.: Fetal Mummification in Swine. JAVMA 140, (1962): 691
- Spies, H. G., Zimmerman, D. R., Self, H. L., and Casida, L. E.: The Effects of Endogenous Progesterone on Formation and Maintenance of the Corpra Lutea And on Early Embryo Survival in Pregnant Swine. J. An. Sci. 18, (1959): 163
- Thorne, H., and Nilsson, P. O.: Staphylococcus aureus as the Cause of Abortion in Swine. Acta. Scand. Z, (1961): 311
- Young, G. A., Kitchell, R. L., Luedke, A. J., and Saul (1950): 141
- Young, G. A.: Influence of Virus Infection, Vaccination or Both on Embryonic and Fetal Development. Proc. Book. A.V.M.A. (1955): 377
- Young, G. A., Kitchell, R. L., Luedke, A. J., and Saul, J. H.: The Effects of Viral and Other Infections of the Dam on Fetal Development in Swine. 1. Modified Live Hog Cholera Viruses Immunological, Virological and Gross. Pathological Studies. J.A.V.M.A. 126, (1955): 165.