

# Disease of Newborn and Suckling Pigs I

## Nutritional anemia and food "hypersensitiveness"

H. C. H. Kernkamp, D.V.M., M.S.\*

**D**URING the past two decades disease in the newborn and suckling pig has demanded the attention of veterinarians in practice and in research more than at previous times. A quarter of a century and more ago the man whose baby pigs sickened and/or died, seemed to accept the situation as a circumstance peculiar to his lot. The situation is much different today. At the present time almost everyone profoundly interested in the swine industry is eager to learn the reason for sickness and death of young pigs and the methods for their prevention and control.

The losses among newborn and young pigs are far in excess of what they should be. While we are in possession of considerable knowledge of the cause, nature, and methods of treatment and control of some of the diseases of young swine, we are also cognizant of the fact that too little is known about many other of their ailments. In fact, not much fundamental information is available with respect to the normal physiology of swine of any age and it cannot be expected that great accomplishments will be forthcoming regarding the abnormal conditions until the former has been prosecuted. We are frequently coming in contact with disorder and disease in newborn and suckling pigs for which a more comprehensive understanding of the process or condition awaits physiological investigation.

Nutritional anemia is a disease of suckling pigs in which there is a reduction in hemoglobin, in red blood cells or in both

resulting from an insufficient intake or uptake of certain food or dietary constituents, chiefly iron. This disease is also referred to as *iron deficiency*.

### Nutritional Anemia

Nutritional anemia is more common than is generally supposed. For reasons mentioned later, nutritional anemia of suckling pigs is more important than many folks realize. This disease is especially common in pigs born during the winter and early spring seasons where north temperate and arctic climates prevail. These are times of the year when very young pigs are kept indoors and are thereby removed from natural habitats where they might obtain some of the essentials that prevent the development of this disease. Pigs farrowed outdoors and where they can obtain the essential iron in the soil, or those farrowed indoors but privileged to run out on natural soil or where soil is made accessible to them, are much less likely to suffer from this condition.

The iron reserve of newborn pigs apparently is not sufficient for their normal blood regeneration for more than 3 to 5 weeks after birth. Therefore it must be supplemented. Hemoglobin determinations and red cell counts of a large series (237) of young pigs living under conditions favorable to the development of anemia showed that a marked reduction in hemoglobin occurred in a very high proportion of the cases and that a reduction in red blood cells occurred in more than 50 per cent of the pigs. The examinations began on the day of birth and then once each

\*Division of Veterinary Medicine, University of Minnesota, University Farm, St. Paul, Minnesota.

week for 5 to 7 weeks. This anemia could be prevented by supplemental feeding of inorganic iron and the course of the disease, based on the degree of the anemia, could be satisfactorily managed by administering various quantities of oven-dried soil (in this case a loam soil gathered up at University Farm) or by permitting the pigs to eat it *ad libitum*.

### Symptoms

The clinical picture of nutritional anemia in suckling pigs is quite distinctive and diagnostic. In the first place one takes into consideration the circumstances under which the disease most frequently occurs. Chief among these is the situation in which the suckling pigs are confined to stalls or pens where they cannot contact soil (in this connection we have in mind soils that contain more than just traces of iron). More and more we are finding that farrowing pens and rearing pens are constructed with concrete or board floors, which, insofar as this ailment is concerned, are not the most desirable. We are not condemning this sort of construction because it has many good points with respect to maintaining good hygiene. The deficiency can be compensated by methods which will be described later.

A second important point in connection with the occurrence of nutritional anemia is the matter of the age of the affected pigs. The third to the fifth week of post-natal life is the time that the earliest physical signs are generally manifested. This means, however, that the disease was in progress for several days at the best. There is pallor of the skin of the ears and snout and in breeds with nonpigmented skin, even the entire skin is pale. The mucous membranes and sclera of the eye, and the mucous membranes of the mouth are pale. "Plumpness" and "fatness" in pigs of anemia age, even to the extent of wrinkling in the regions of the neck, shoulders, loin, and flank are characteristics of anemia more often than they are signs of vigor and health. The "plumpness" and "fatness" are frequently the result of accumulations of fluid in the subcutaneous tissues (anasarca) due to a dis-

turbance of osmotic pressure balances of the blood plasma.

With the progress of the disease, respiratory symptoms are evident. A rapid, "thumpy" breathing abdominal in type occurs. The reduced capacity of the blood to transport oxygen together with the edema of the lungs is the reason for the hyperpnea. Another symptom displayed by many cases is the voiding of a light colored and watery stool. This often takes on the characteristic of "scours." A small proportion of the cases will vomit. No thermic reaction occurs.

While the physical signs described above are very valuable diagnostic criteria, nevertheless, the most specific evidence of the disease rests with the blood picture. Hemoglobin examinations on the freshly drawn blood are of greatest value. Values of 6 Gm. of hemoglobin per 100 cc. of blood are indicative of an abnormal state. Cases in which this falls to 3 Gm. per 100 cc. are to be considered poor risks. If these are to be measured on the Tallquist scale then 50-60 per cent is indicative of anemia and 20-30 per cent a case with great potentiality of death. Where a count can be made of the number of erythrocytes per cmm. one frequently finds less than 3,500,000 cells in cases of anemia. It is highly desirable to prepare a blood smear and stain it by Wright's method. An examination of a smear from a typical case of nutritional anemia will show that most of the red cells are smaller and paler staining than normal. The coloring of these cells seems to be more concentrated at the periphery leaving them quite pale in the center.

### Post-mortem Findings

The autopsy on a typical case of nutritional anemia will show certain changes that can be interpreted as quite characteristic. If one has the opportunity to sacrifice a case for post-mortem purposes, careful notice should be given to the appearance of the shed blood. In patients with anemia the blood will be very light colored, "watery," and "thin." The heart is enlarged and flabby. In some cases a pronounced exudate collects in the peri-

cardial sac. This attaches to the epi- and pericardium and when the latter is removed it leaves a surface that resembles two buttered slices of bread that have been pulled apart (so-called "bread and butter" exudate). The liver is enlarged and mottled in appearance. Usually the abdominal cavity contains a large amount of fluid. Edema of the lungs is also a lesion in a great many cases. The mucous membranes throughout the body are anemic.

### **Prevention and Treatment**

In prevention and treatment of nutritional anemia, it must be remembered that iron is an essential constituent of the blood because it is the important part of hemoglobin. It is necessary for normal blood regeneration. Iron, therefore, is required in the prevention and treatment of nutritional anemia in suckling pigs. By permitting the young pigs to ingest soil we have repeatedly been successful in the prevention of this disease. It is important that they be able to obtain the soil as early as the fourth or fifth day following their birth and it should be available to them at all times up to the fourth or fifth week of life. It is quite possible that some soils do not contain enough iron to supply the requirements of the young pigs in which case it can be fortified by adding iron to it. To 10 pounds of damp soil (not powder dry, nor muddy wet) add from 10 to 15 tablespoonfuls of a solution of ferrous sulphate (ferrous sulphate 3 ounces, water 4 liters). This amount of soil would be sufficient for 7 to 9 pigs for 3 to 4 days. It is sometimes necessary to administer treatment more direct and in this event, 1 teaspoonful of the above mentioned iron solution given once each day should suffice.

### **Intrinsic Factor**

During the past few years we have on several occasions experienced a situation which has been very interesting. From clinical appearance, blood picture findings, and post-mortem examinations, we have seen little pigs sick and dead with what we believed to be nutritional anemia

due to an iron deficiency. Furthermore, the pigs in this case had full opportunity to consume soil and there was good indication that they had been eating it. By analysis, the iron content of the soils involved is not known but it is of interest to report that one of the farms is located in the iron mining section of Minnesota. Whether or not we were dealing with a deficiency of the so-called intrinsic or gastric factor, is not known. This factor is believed to be concerned as exerting an influence on the normal hematopoiesis.

Nutritional anemia, besides being a disease *sui generis* and one which can snuff out the life of the patient unless properly managed, places the patient in a favorable condition to contract and become affected by infectious and parasitic diseases. McNutt and Packer in the 1943 fall issue of this journal describe streptococcal infections in young pigs that had a definite history of anemia just prior to the time of contracting the infection or the infectious process was superimposed upon the existing anemia. We have seen some of the most severe cases of necrotic stomatitis and gingivitis in pigs also suffering from anemia or pigs that had had anemia. Many pigs will recover from the anemia but are left in a state where they subsequently fail to grow and develop economically. They are stunted.

### **Hypersensitiveness**

There sometimes occurs in young swine a symptom-complex marked by epileptiform seizures. Because of the circumstances surrounding its occurrence and its clinical manifestations, the condition belongs in the category of the hypersensitive, anaphylactic, or allergic phenomenon. The disease is striking to behold and engenders much interest to all coming in contact with it.

The history and clinical picture of this condition is remarkably similar in all cases. The first notice of any unusual or abnormal signs is the sudden onset and violent seizures displayed by the patient. These will occur within a very short period of time (a few seconds as a rule) after

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## BOOK REVIEW

*The Practice of Veterinary Medicine.* D. H. Udall, B.S., D.Sc., Professor of Veterinary Medicine and Superintendent of the Ambulatory Clinic, Emeritus, in the New York State Veterinary College at Cornell University. 4th revised edition. 723 pages. 102 illustrations. Cloth. Published by the author, Ithaca, N. Y. Price, \$6.50.

The fourth edition of this book on veterinary medicine in 10 years indicates its popularity. Only 4 years intervened between the third edition and this new revision. Quoting from the author's preface: "A period of 4 or 5 years may be taken as a generation in the growth of medical knowledge."

Further information was necessary because of the progress which has been made in several fields, namely, the control of parasitic diseases, the employment of the sulfa drugs in certain infections, specific biological therapeutics, and disturbances resulting from faulty nutrition, all of which have received the careful attention of the author.

An examination of the references shows that facts from recent periodical literature are included to insure information of the latest research work. Much of the material is based upon the author's personal observations and experiences and the diction is in his characteristic style.

Although the latest edition contains almost 3 times as many pages as the first edition it has been kept comparatively free of material that is of no special interest to the busy practitioner, such as historical resumes and detailed accounts of research and experimental work. Its purpose remains, as it was primarily intended, to be a source of working knowledge.

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It is reported that Russia had 50,000 men in veterinary schools at the outbreak of the war.

## NUTRITIONAL ANEMIA

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the pig ingests food. From our experience, the particular food concerned consists in whole or in part of cow's milk but we have been informed by some folks that the symptom-complex has been observed in pigs that did not receive it. While we suspect that the particular causative factor is a food protein, we do not possess specific knowledge with respect to this point.

### Entrance of Foreign Protein

According to Rich (*Physiological Reviews* 1941, 21: 70 -), "When a foreign protein, whether bacterial or non-bacterial in origin, enters the tissues by whatever route, there ordinarily occurs in the body certain alterations which affect the reactivity of the tissues toward subsequent contact with the protein. When these alterations have occurred, the body is said to have become 'hypersensitive' to the foreign protein . . ." Accepting for the present that the proteins in cow's milk are the specific sensitizing factors, the next point is, when and how did the sensitization occur? We are not presuming to know the answer but it seems to the writer that the substance (proteins of the milk) entered the tissues from the gastro-intestinal tract. Pigs, after they are a week of age, will ingest foodstuff other than their mother's milk if they have the opportunity. Skim milk is a common foodstuff of swine and very often one finds that sows nursing pigs are fed skim milk as a part of their diet. After the protein is ingested it must reach the body tissues unaltered in order for it to effect the reactivity and thus create the hypersensitive state. This could occur in the stomach where some injury to the gastric mucosa existed. In support of the fact that injuries sometimes do exist in the gastric mucosa, we have seen ulcerative and erosion-like lesions in the stomachs of pigs that were less than 4 weeks of age. In other words, some of the factors considered necessary for the setup of a state of hypersensitivity occur in young swine.

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The clinical picture is characteristic and diagnostic. It will be ascertained that an apparently normal pig will walk up to the food trough and partake of a mouthful or two of the food and shortly and suddenly thereafter it will display the seizure. This is marked by drawing the head backward, elevating the snout with the nostrils distended and the mouth open. The eyeballs are rolled backward, the legs become stiffened, and the animal falls to the ground. The thorax is expanded and the respirations are shallow and jerky. A distressing squeal is emitted. Profound coma ensues. This paroxysm lasts for only a few minutes but the patient usually remains recumbent for 5 to 15 minutes showing much evidence of exhaustion. At the expiration of this phase it will rise to its feet, advance to the trough again, and partake of the food without showing the least signs of discomfort. As a rule, a repetition of the seizure will occur at the next feeding period, especially if the interval between feedings is at least 3 or 4 hours. This will occur many times in some pigs and finally subside. Some pigs succumb, but the number that die from this condition is small. The age of the pigs involved ranges between 4 and 10 weeks.

### **Treatment**

From the standpoint of the treatment and management of the epileptiform complex, one keeps in mind that he is dealing with a sporadic problem. Careful inquiry and inspection of the foodstuffs the affected pigs are receiving is imperative. It is also important to learn what foodstuff the mother of the patients received prior to the time the first case was noticed. This may extend into the period of gestation. We would especially call attention to cow's milk, because as stated above, our experience points to this foodstuff as the chief causative factor. This being the case, then it becomes apparent that the cow's milk should be the first foodstuff to withhold from the diet. When satisfactorily demonstrated that the disease continues after the cow's milk is removed from the diet, then it is necessary to hold back some

other protein food. By trial and error in the process of elimination one should be able to establish with considerable certainty the responsible food factor or factors.

### **Immediate Treatment**

Where immediate treatment of a suffering patient is the objective, then an intramuscular injection of adrenalin is advised. The dose of adrenalin for a 30 to 35 pound pig is 0.1 cc. of a 1:1000 solution. This will relax the muscles of the bronchioles and resuscitate a heart that fails to beat. The injection can be made directly into the heart muscle in case of heart failure but the dose must be decreased 30 per cent and the rate of injection reduced.

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## **HEREDITARY RESISTANCE**

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females with unusually viable offspring are being produced suggests that further progress in increased viability is possible by using proved blood lines repeatedly.

Admittedly, there are difficulties hard to overcome. In building a new line of hardy stock entire families must be eliminated, so it may be beyond the means of an ordinary breeder. Transmission of the viability in repeated generations of the same blood line is necessary before one can be positive that he has a family that will survive an infection of any particular disease. Also, when this is established, there is no indication that the family will be resistant to any other disease since genetic resistance is probably specific for each disease. Further, the resistance is difficult to fix. It may often be lost in the first cross, and it is difficult to maintain a line of the improved stock.

Because of the variations in disease resistance within a species or breed, it seems entirely reasonable that the more resistant types may be selected naturally to some extent, provided exposure to infection is nearly universal. But such mass selection is much less effective than quan-