drainage in the low, boggy areas. Replacements from disease-free herds can be made after several months. The disadvantages of such a program are obvious, however, especially when valuable breeding animals are implicated or when it is doubtful that healthy replacements can be obtained.

A third method involves the selection within the herd of normal-appearing pigs for breeding purposes and the elimination of those that are obviously affected. Such a program could be improved by using the rhinoscope to aid in the selection and rejection of the pigs.

At the Agricultural Research Center we found that we could minimize the incidence of atrophic rhinitis by raising rhinoscopically negative weanlings to maturity in an isolation area under conditions of litter segregation and raising a second generation of pigs under the same conditions.

A fourth method offers a means of obtaining swine free of atrophic rhinitis but has practical limitations. This method consists of removing the baby pigs from the dam by hysterectomy or Caesarean section, or catching them in sterile bags or on sterile cloth as they are born and removing them 24 hours after birth. In all instances the baby pigs must be hand raised under isolated conditions. Such procedures can be used when it is necessary to obtain valuable breed strains free of the disease and when adequate facilities and sufficient help for the sanitary care and management of the baby pigs are available.

RICHARD D. SHUMAN is a veterinarian in the Bacterial and Mycotic Diseases Section of the Animal Disease and Parasite Research Branch.

JOHN S. ANDREWS is a parasitologist in the Helminth Parasite Section of the Animal Disease and Parasite Research Branch at Beltsville. He majored in parasitology at Purdue University.

F. L. EARL is a veterinarian in the Viral and Rickettsial Diseases Section of Agricultural Research Service. Following graduation from Michigan State University in 1947, Dr. Earl was employed by the State veterinarian's office in Missouri to investigate pullorum and Newcastle disease. In 1948 he joined the Department of Agriculture as station veterinarian in Beltsville.

Hog Cholera

J. P. TORREY

HOG CHOLERA is a 40-million-dollar annual expense to the 4-billion-dollar swine industry in the United States.

It occurs in every State and in parts of every country where hogs are raised, except those—Northern Ireland, Denmark, and Australia, among them—that have undertaken vigorous and continuing eradication measures. Reports are that hog cholera broke out in countries where meat from the United States was shipped for use by American Armed Forces.

We have the means to stamp out hog cholera, would we but use them.

THE FIRST AUTHENTIC REPORT of hog cholera in the United States came from Ohio in 1833. The disease spread rapidly. By 1887 it existed in 35 States. It became unusually prevalent at intervals of about 10 years between 1887 and 1926—1887, 1896, 1913, 1926.
The annual loss of hogs that died was placed at 65 million dollars. The average for a 10-year period was not less than 20 million dollars. The indirect costs doubled that figure.

The cause of hog cholera is a virus, which has a mean diameter of 27 millimicrons. (25 million millimicrons equal 1 inch.) This virus readily passes through Berkefeld and Chamberland filters. It is killed in 30 minutes by heat at 55° C.; in 10 minutes at 60° C.; and in 60 minutes in dried blood at 72° C.

The virus is destroyed by a 2-percent cresol solution in 60 minutes. A 3-percent solution of sodium hydroxide in combination with 2-percent milk of lime kills the virus in 15 minutes.

The virus lives best in an acid solution. The optimum pH for preservation of virulence is 5.0 to 5.5. The virus is quite resistant to phenol, and will survive long periods in a 0.5-percent solution. Phenol is used as a preservative when the virus is produced for immunization purposes. It has been kept in a perfect state of preservation for 7 years at —40° C. It will survive in meat products for months and will live at least 6 months in pickled, salted, and smoked meats. Putrefactive processes destroy the virus in about 5 days, except when it is in the bone marrow, where it survives for at least 15 days.

When it enters the pig's body, the virus passes to the blood stream and develops there. It is therefore a viremia, or blood infection. The blood of a pig becomes infectious within 24 hours after the virus is injected into its body. The urine and feces usually contain the virus within 48 hours. The secretions of the eyes and nose become infectious by the third day.

Rarely do pigs show visible symptoms earlier than the fourth day. Infected pigs therefore can transmit the disease before any symptoms can be seen. The maximum growth of virus is reached in 6 to 8 days.

The virus moves to all parts of the body, primarily in the red blood cells. The liver and spleen contain more of the virus than other organs do.

Some viruses will cause infection in pigs when 1/5,000,000th of 1 cubic centimeter is injected. Others will not cause disease when less than 1/10,000th of 1 cubic centimeter is injected.

The incubation period—the time that elapses between the invasion of the first cells of the body and the first symptoms of the disease—is quite variable and depends on the virulence of the virus and the individual resistance of the pig. Symptoms may be observed 3 days after the injection of virus, but sometimes it may be 7 days before the pig is visibly sick.

When susceptible swine are exposed by contact or in other ways, one cannot know the exact time when infection occurs, but visible symptoms are rarely observed within less than 5 or 6 days after such exposure.

Hog cholera may be introduced into a herd in many ways. When the disease is introduced in a small amount of infected material, such as a scrap of raw pork or small amount of dirt on an attendant's shoes, one or two pigs that eat it become sick first. Several days may pass before more animals become sick.

At least 2 days pass before the urine of the first exposed pig contains virus and other pigs eating feed contaminated with the urine will become exposed. The period of incubation must elapse before these pigs show symptoms. Thus when cholera is introduced in a herd, about 7 days will elapse between the time the first sick pig was exposed and other susceptible pigs begin to show symptoms. This characteristic of the disease shows the importance of being concerned when only one pig in a herd becomes sick. Immediate isolation of the first sick pig and early diagnosis, followed by proper immunization, will prevent heavy losses in the herd if the trouble is due to hog cholera.

When hog-cholera virus is introduced into a herd in large amounts,
as in a bone brought from the neighboring farm by the farmer’s dog or litter thrown from a stock truck, the outbreak will be more explosive, and a number of animals will become sick about the same time. These cases are more difficult to handle and the loss usually is greater.

Birds, such as pigeons and sparrows, and insects have been suspected of spreading hog cholera, but experimental evidence has failed to incriminate them.

Man is most to blame for the spread of the virus. Improper handling and use of the virus used for vaccinating pigs may start an outbreak of cholera. Men who visit sales barns, stockyards, slaughterhouses, and rendering plants and do not disinfect their shoes or truck and change clothing when they return to the farm may spread the disease.

The virus is spread within a herd by contact with sick animals or material infected with excrements from a sick animal. Animals recently vaccinated with virulent virus may spread the disease, especially if they show some reaction to the vaccination. Such animals should never be mixed with susceptible swine.

The virus of hog cholera differs from most other infectious agents in its relation to the white cells of the blood. Most infections, especially bacterial infections, cause an increase of white blood cells. The function of white corpuscles is to pick up infecting or invading particles and to try to carry them from the body. Hog-cholera virus has the opposite effect on white corpuscles. It destroys the corpuscles and apparently stops the production of them. The number of white cells in the normal pig may vary between 7,000 and 30,000 per cubic centimeter. Any number below 7,000 is subnormal, and the condition is called a leucopenia.

This characteristic of hog cholera can be used as a diagnostic agent, but it cannot be relied upon if the count is higher. An animal may be infected with cholera and another disease at the same time. The cell count might then be high and a wrong diagnosis would be made.

The American strains of the virus are more virulent than the European strains. They differ also from the Canadian viruses.

The American strains seem to vary in virulence among themselves from time to time. Because there is no known serological method of differentiating the viruses, they have been considered the same virus. Heavy death losses in 1949 and 1950 in swine after vaccination with certain serials of viruses and serums indicated, however, that the viruses were different.

After extensive investigation, C. N. Dale and other scientists of the Department of Agriculture learned that the viruses differed from regular viruses in their antigenic properties—ability to produce immunizing properties—in a serum. The immunizing properties produced by a regular virus would not protect against the irregular viruses unless the amount of serum was greatly increased. These viruses are called variant viruses.

Since this discovery, variant viruses have been isolated from sick pigs that were not vaccinated with serum and virus. Variant viruses may have been involved in post-vaccination losses for a long time, but the cause of the losses may not have been determined.

The differences between variant viruses and regular viruses are: Variant viruses have low antigenic properties and therefore will not make good hyperimmune serum or vaccine. Hyperimmune serum made from regular viruses will not protect pigs against variant viruses in the same dosage as is used for regular viruses. The minimum lethal dose of variant viruses is greater than the dose of regular viruses. The variant characteristics are not stable, and after several passages in pigs the characteristics are the same as regular viruses. The variant characteristics are retained when the virus
is injected into susceptible pigs simultaneously with subprotective doses of immune serum.

Hog-cholera virus has been propagated in swine tissues implanted on the chorio-allantoic membrane of chick embryos.

It has been cultivated more successfully in tissue cultures that employ various swine tissues, such as lymph node, spleen, kidney, testicle, and choroid plexus. More recent methods used only spleen as a culture medium.

The symptoms of hog cholera differ in different hogs and in different herds, depending on the strength of the virus and the ability of the animal to resist the disease attack. Because of the variations, the disease is said to exist in two forms. In the acute, or severe type, the hogs sicken and die quickly. In the chronic, or less severe type, the hogs may be sick for weeks or months before they succumb or (in a few cases) recover.

At first the pig's temperature quickly rises to 105° to 107° F. The temperature continues high for a few days. Then it gradually drops. The temperature may go to subnormal if the pig lingers for a time.

A few pigs (or possibly only one) sicken at first. They refuse to come for feed with the herd but remain in the nest. They may appear to be cold and shivery, and their backs may be arched when they are driven from the bed. The sick hogs become gaunt or tucked up in the flank as the disease progresses. They have a weak, stilted, staggering gait; the weakness is most noticeable in the hind legs. Constipation is commonly present in the early days, but it may be followed by a diarrhea.

A red or purplish discoloration of the skin on the belly, ears, and inner surface of the legs may happen in some cases, but not all. Because the same discoloration may be found in other diseases, it cannot be a diagnostic symptom.

The eyes in the early stages may have a watery discharge. Later this becomes thicker and gums the eyelids shut. The eyes may be congested.

Postmortem examination of sick pigs is important in making a diagnosis, but one cannot rely on it entirely. All the lesions described for hog cholera rarely are found in one animal. Only a few organs may be involved, or there may be no visible changes at all. The absence of lesions is more often encountered in the early stages of the disease; therefore, it is important to make more than one postmortem examination in a herd.

Lesions are often in the spleen, kidney, lymph glands or nodes, and urinary bladder, but they are by no means constant findings.

The spleen may be enlarged and congested, or it may have irregular raised areas of a dark color, called hemorrhagic infarcts, along the edges.

The kidney may be congested, or it may have small, red spots, called petechiae, in the surface just under the outside covering or capsule. The spots may be as small as a pinpoint or as large as a pinhead. They may be few or numerous, like the specks on a turkey egg.

Lymph glands or nodes are normally gray, but they become enlarged and bright red or dark when they are infected with cholera. The redness may be on the outside surface, while the inside has a normal color. Sometimes the entire gland may be dark red.

The glands of the neck (cervical glands), of the lower jaw (submaxillary glands), beneath the kidney (renal glands), along the small intestines (mesenteric glands), and between the hind legs (inguinal glands) may have hemorrhages. One or more of the glands may be involved at a time.

The urinary bladder may be congested or may have hemorrhages on either the inner or outer surface, or both. The hemorrhages may be very small or quite large. Hemorrhages may occur in the stomach, small intestines, and large intestine.
DETECTION of hog cholera in the early stages often is difficult because postmortem findings may be negative or may be indistinguishable from a number of septicémie diseases.

A positive diagnosis can be made only by inoculating filtered blood from one or more sick pigs into healthy, cholera-susceptible pigs, to some of which hyperimmune serum is given at the same time. The pigs receiving the serum should remain well, and the others should get sick if cholera virus is present. That obviously takes time and is not practical in the field.

One has to rely on a careful study of the history, clinical symptoms, autopsy findings, and experience in making a diagnosis. A complete accounting of the farmer’s movements, the happenings around the farm, and the management of the herd during the previous 2 weeks often gives some idea as to how the infection might have been introduced into the herd.

No dependable treatment is available for hogs visibly sick with cholera. Prevention, therefore, is of prime importance.

Proper sanitation and other preventive measures often will check the spread of virus.

The swine raiser should know how the virus is carried. Hog cholera does not occur in a herd unless the specific virus is introduced. The most certain way to do that is to bring an infected hog into a herd. Any hog added to the herd must be considered a potential source of danger until it has been isolated long enough to allow cholera to develop if the hog had been exposed to virus.

Because hogs affected with cholera discharge the virus from their bodies in the urine, feces, and secretions of the nose and eyes, the manure, bedding, and dirt in their pens are contaminated. The virus may enter the hog’s system in food and drink and possibly through wounds or abrasions of the skin.

Infected hogs sometimes are shipped to market in order to get rid of them. Public stockyards, unloading chutes, railroad cars, and susceptible hogs may become contaminated or exposed in that way.

Cholera may be carried to a farm in infected litter, manure, or other material from public stockyards or cars. Infected material may adhere to other animals, wagon wheels, tires, and to the shoes of men who have entered such places. That also applies to farms where cholera exists—it may be carried from an infected farm to healthy herds when, for example, farmers exchange labor and implements or deliver grain and stock to market.

Cholera may be spread by stock buyers and sellers of stock remedies who go from farm to farm, and by animals bought at public sales.

Feeding garbage containing scraps of uncooked pork and bones also is dangerous. They may spread cholera and other swine diseases, particularly vesicular exanthema and trichinosis. The only safe garbage is cooked garbage.

A good ounce of prevention is to disinfect all things that might carry the virus. A 2-percent solution of ordinary lye is recommended. In places where lye cannot be used, a cresol compound disinfectant will do.

IMMUNIZATION is another means of prevention. Because the virus is highly contagious and can spread fast, some method had to be devised for protecting the hog. For that, hyperimmune serum was developed, but the men who discovered it realized that cholera could never be stamped out as long as a virulent virus was being spread. So the investigators continued to search for a better immunizing agent. They produced a killed-virus vaccine, but it, too, had limitations.

Other research workers tried to perfect an immunizing agent and developed five types of products for immunizing hogs against cholera:

- Anti-hog-cholera serum of swine origin;
Hog Cholera

A fully virulent virus of pig origin used in conjunction with anti-hog-cholera serum;
A killed or inactivated virus vaccine made from swine tissues, inactivated with crystal violet or eucalyptol;
Modified virus vaccine passed through a nonspecific host, like the rabbit;
A modified tissue-cultured virus vaccine, grown upon tissue cells from the pig.

After it was determined definitely that a filterable virus was the cause of cholera, the investigations were directed toward finding a way to protect hogs from infection by the virus.

The first attempts at preventive measures were to develop a vaccine by attenuating or destroying the disease-producing properties of the blood in liquid or dried form and by using a mixture of virulent blood and the blood from immune hogs. All those experiments were fruitless.

Efforts then were turned to making a protective serum. These experiments were started in 1903, when the first hog was hyperimmunized. This hog had been used in experiments on hog cholera by Marion Dorset, at the Federal Experiment Station, Bethesda, Md., and was identified as number 844. It had recovered from an attack of hog cholera. It was given an injection of one cubic centimeter of blood from a sick pig, which had been injected with infected blood by W. B. Niles, at the Hog Cholera Research Station in Iowa, and shipped to Bethesda. The dosage was gradually increased to 400 cc. of virus blood. The hog remained normal. Eleven days after the last injection of virus blood, the end of its tail was cut off with a chisel and hammer, and 100 cc. of blood were collected in a pan.

The blood was defibrinated (prevented from clotting) by constant stirring and then tested for potency by injecting 20 cc. of it into each of two susceptible pigs with and without virus blood. The two pigs were tested 5 weeks later by injecting infected infected blood. The pig that received only serum died. The one that got serum and virus lived. Thus the first hyperimmune serum was produced. It is referred to as anti-hog-cholera serum.

Many experiments were carried out at the Hog Cholera Research Station, Ames, Iowa, from 1905 to 1907 to make sure that adequate protection was afforded in almost 100 percent of the pigs. In 1907 the serum was put to a practical test by vaccinating farm herds. The results were good. The method of preparing the serum was then patented by Dr. Dorset, who had discovered the methods, and all rights were dedicated to the public.

Notice of the successful results was sent by A. D. Melvin, chief of the Federal Bureau of Animal Industry, to livestock officials of every State, with an invitation to send representatives to the Hog Cholera Research Station to observe the work and consider plans for the practical applications of this method of combating hog cholera. Representatives from 25 States attended a conference in Ames in 1908.

A number of States began to produce and distribute the new serum to their farmers. The preparation and sale of serum was undertaken by commercial concerns. As the commercial establishments began to increase in size and number, the States discontinued making the serum. Only commercial firms now make anti-hog-cholera serum.

The basic principles of producing serum are the same as when the first hyperimmune serum was made from hog number 844. The mechanics of producing serum have been improved greatly by research workers in the United States Department of Agriculture, in various States, and in commercial companies. These improvements give a more refined standard serum free from the disease-producing organisms.

The serum is prepared by the intravenous injection of 5 cc. of virus blood
for each pound of body weight into the hogs that have been immunized against hog cholera for at least 90 days. After 11 days, blood is drawn from the tail three times at intervals of 7 days. The next week—that is, after the three intervals—all the blood is taken by bleeding from the throat. The blood is mechanically defibrinated as it is collected. The fibrin is removed by a centrifuge.

The red blood cells are removed by passing the blood through a milk separator after a small amount of bean extract (Wisconsin pea bean) and salt solution are added to the blood to facilitate the removal of the cells. The clear serum is then pasteurized at 58.5° C. for 30 minutes. Phenol is added so as to make a 0.5-percent concentration.

The serum is tested for potency by injecting 15 cc. of serum and 2 cc. of virus into 40- to 90-pound pigs. The pigs must pass a satisfactory test before the serum is released for sale. Purity tests for swine erysipelas and other contaminating organisms as well are made. No serum more than 3 years old can be sold.

Hog-cholera virus is prepared from the blood of the pigs that have cholera, show the characteristic symptoms, and on postmortem examination disclose lesions typical of hog cholera but no lesions of other infections. Five or seven days after the injection of the virus, the blood is drawn aseptically and defibrinated. Phenol is added as a preservative. This virus is the active agent that produces disease. If it is improperly handled it can cause an outbreak of cholera.

Two methods of serum treatment are used.

The serum-alone treatment consists in injecting hyperimmune serum into the pig at one of three places—the axillary space between the shoulder and body, the loose skin in the flank, or the abdominal cavity.

The length of time immunity will last varies with the individual pig. Some will be protected for 2 weeks and others 2 months.

Serum alone is used when only a temporary immunity is required. It may be recommended for pregnant sows and in an emergency when it is necessary to immunize pigs affected by a condition or disease, other than cholera, that has lowered their vitality so that they are not in proper condition to receive virus. The dosage of serum must be judged according to the size of the pig and the conditions under which it is given.

In simultaneous inoculation, hog-cholera virus and anti-hog-cholera serum are administered at the same time but in different places. The combined use of the two products results in permanent immunity in most herds. The serum protects the pig until the body has had time to develop immunity to the virus. For some unknown reason, some pigs do not develop immunity and are a source of danger and a loss to the owner. This failure to develop immunity after treatment with serum and virus is one of the reasons hog cholera cannot be eradicated by the simultaneous immunization method.

The dosage of virus remains the same for pigs of all sizes—that is, 2 cc. each. Some veterinarians prefer, however, to give larger doses of virus, 3 to 5 cc., and increase the serum accordingly. We have no experimental evidence to support this practice. Virus may be injected into the axillary space on the opposite side from where the serum is administered or it may be injected into muscles in the hind leg.

The dosage of serum is adjusted to the size of the pigs. The dosage recommended on the label of the serum bottle varies from 20 cc. for suckling pigs to 75 cc. or more for grown hogs. Many veterinarians increase the recommended dosage 50 to 100 percent in the belief that it is better to give a large dose of serum than to give too little. Good judgment and experience must determine how much serum to use. The serum may be injected into the axillary space between the front leg and body.
into the loose tissue in the flank, or into the abdominal cavity. A large dose should not be injected in one place nor should it be injected too fast, lest the surrounding tissue be destroyed and an abscess form.

**Hog cholera vaccines** may be called modified hog-cholera-virus vaccines. They may be either live virus or killed virus.

The killed-virus type may be made of blood or of tissue from pigs that have cholera. The vaccine made from tissue is prepared by treating lymphoid tissue, principally spleen, from cholera-infected pigs with 1-percent oil of eucalyptol. After about 8 weeks at 45° F., the virus is killed, but the antigenic properties are retained. This vaccine commonly is spoken of as BTV—Boytont's Tissue Vaccine.

The other killed vaccine of blood origin is known as crystal violet-glycerol vaccine. It was first prepared in 1934 under the direction of Dr. Dorset. Since then other men in the Department of Agriculture have gathered and have made many improvements in the production and use of the vaccine.

Crystal violet-glycerol vaccine is prepared by adding 20 parts of a solution of crystal violet in glycerol (also called glycercine) to 80 parts of defibrinated blood from a cholera-infected pig. The crystal violet-glycerol solution is prepared by adding 1 part of crystal violet to 400 parts of glycerol. The method of use and the results of the two killed vaccines are almost the same.

A dose of 5 cc. of the killed vaccine is injected subcutaneously into each pig. Immunity is produced in about 14 days. Although some protection is given in 7 days, the vaccine should not be used in herds where cholera already exists and pigs should not be exposed to cholera within 14 days after treatment. Immunity will last 8 to 12 months in most pigs. In some it will last longer.

The chief advantage of this type of vaccine is that it produces immunity but does not introduce active virus into the hog. Therefore it can be used in herds infected with diseases other than cholera, because the use of virulent virus would be dangerous with them.

**Modified-live-virus vaccines** of rabbit origin are of two types. One type is made from a virus that has been passed from one rabbit to another until only a very slight reaction is observed when the virus is injected into a pig. The virus is injected into pigs, and certain tissues of the pig are harvested, treated, bottled, and dried in vacuum. The vaccine is tested for safety, potency, and purity before it is sold. It is necessary to inject 10 cc. of anti-hog-cholera serum at the same time this vaccine is used.

The other type of rabbit-origin vaccine is made from a virus that has been passed from one rabbit to another more than 200 times so that it lost its disease-producing properties; when it is put back into the pig no lesions or symptoms are observed. This type of vaccine is prepared from rabbit tissues which are collected, treated, and dried in vacuum the same as the other vaccine. No anti-hog-cholera serum is recommended for use with this vaccine. However, serum can be used if desired.

Vaccines were not used very widely to protect pigs against cholera until the modified-live-virus vaccines were produced. Since their introduction in 1951, their use has steadily increased; 11,072,295 doses were sold in the 12 months ending June 1952 and 15,800,577 doses were sold in the year ending June 1953. The production of serum and virus produced declined proportionately.

An important question came up: Are the modified vaccines as good as serum and virulent virus in the prevention of hog cholera? The answer will come after further use of the modified vaccines. In 1955 we might have been in the low phase of the 10-year cycle that characterizes the field outbreaks of the disease. The cycles seem to occur without respect to the amount of vaccina-
tion and therefore should be taken into account when one attempts to evaluate the efficiency of the new vaccines. The incidence of cholera declined from 1952 to 1954, but we cannot attribute the drop to the increased use of modified vaccines until the end of the 10-year cycle.

When the new vaccines were introduced, claims were made that these new forms of virus would be totally incapable of producing cholera. Field usage and experimental evidence have disproved the claims under certain circumstances. Some pigs are highly susceptible to cholera and acquire the disease easily when vaccine is injected into them, although the same vaccine may make other pigs immune.

I know of no way in which to determine which pigs are hypersusceptible. Sows injected with modified-live-virus vaccines during the first 30 days of pregnancy may abort, or many of the fetuses will have abnormalities, such as ascites, edema, asymmetry of the head, lengthening and twisting of the snout, and malformations of the limbs.

Eradication of hog cholera in the United States has been talked about since the first experimental work on it was begun. When the simultaneous method of immunization was discovered, many thought the method would eliminate hog cholera, but the men who developed the procedure were aware that it would never eradicate the disease. The swine industry could never have grown to a major farm industry without this method of immunization, but the availability of other methods of immunization have made the goal of eradication seem more attainable.

The United States Livestock Sanitary Association in 1951 appointed a committee to study further the possibility of stamping out cholera in the United States. The committee presented its first report in 1951. Its recommendations influenced some States—Alabama, Georgia, and Tennessee—to prohibit the sale of virulent virus. Idaho, Utah, and Wyoming require a special permit for each shipment of virus coming into the State.

The recommendations also led to the establishment of a pilot test area, in which more than 2,500 hog raisers participated, in Florida. The first reports of the project were encouraging. Alabama undertook an area plan of eradicating cholera in 1952. After 2 years, the death losses from cholera were negligible.

J. P. Torrey is veterinarian in charge of the Hog Cholera Research Station, United States Department of Agriculture, at Ames, Iowa. He holds degrees from Mississippi State College and Michigan State University.

The Enteritis Complex

L. P. Doyle and L. M. Hutchings

A GROUP of diseases having similar manifestations is called a complex. Several ailments that cause inflammation of the digestive tract of swine make up the enteritis complex—as enteritis means an inflammation of the intestine. Inflammation of the gut is often accompanied by necrosis, or death, of tissues in the gut wall. The word "necrosis" has been shortened to "necro" and is used widely in referring to disease of the intestine. The term