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Swine Fever in South Africa.

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INTRODUCTION.

EXTENSIVE outbreaks of swine fever amongst pigs from 1933 onwards both in the Transvaal and Cape Province afforded an opportunity of further observations and experiments and of reviewing the whole swine fever problem in the Union of South Africa.

In the latter part of 1933 a number of very severe outbreaks of an infectious disease of pigs occurred in the Western Province and in the Transvaal. In the latter province the disease was mainly confined to the Witwatersrand area. Difficulty was experienced in establishing the true nature of the disease. It was at first thought to be of the nature of a bacterial infection, but early in 1934 cases were encountered in an extensive outbreak in Johannesburg which proved beyond doubt that the disease was swine fever.

(A) SWINE FEVER IN THE WITWATERSRAND AREA (JOHANNESBURG).

Since 1933 a number of outbreaks occurred in this area, some of which were recurrences on the same farm. In the first of these outbreaks at the Lombardy Estate the owner had about thirty pigs all of which died. The symptoms shown by the pigs were high fever followed by death in two to three days. At post-mortem there was swelling of the spleen, haemorrhagic gastro-enteritis and in one case haemorrhages in the kidney. One of the pigs brought to Onderstepoort showed lesions of an acute septicaemia, with splenic enlargement, haemorrhages in the kidneys and a haemorrhagic condition of the periportal lymphatic glands. A pig inoculated with blood from this case died in 72 hours with lesions of acute swine fever.

Another farm in the area, the Cairngorm Estate was apparently infected from the one previously mentioned. In this outbreak the Government Veterinary Officer described reddening of the skin of the ears and abdomen, haemorrhagic gastro-enteritis, haemorrhages in the kidneys and a reddish black colour of the mesenteric glands. This outbreak was not confirmed by subinoculation into susceptible pigs.

In February, 1934, an extensive outbreak occurred on the Savoy Estate, amongst about two thousand pigs. An infectious disease had occurred in October, 1933, but the mortality was attributed by the owner to a pneumonia and was not reported. The whole stock of pigs was sent to the abattoir and after disinfection, restocking took place six weeks later. No further mortality occurred until February, 1934, when about fifty pigs died within a few days. Many of the pigs died within twelve to twenty-four hours of showing symptoms. A wobbling and uneven gait was noticed. A characteristic symptom was the occurrence of reddish patches on the body and legs. In some cases death occurred suddenly without symptoms having been noticed. Bloodstained mucus was seen issuing from the nose. In one pig the ears were very red and swollen. Small haemorrhages of a bluish red colour could be seen under the skin of the body and on the face, causing bleeding at the inner canthus of the left eye. In another pig haemorrhages were seen under the skin of various parts of the body, most marked round the eyes which were swollen.

In this outbreak the following lesions were encountered:—

(*a*) Multiple irregularly circumscribed haemorrhages in the skin from ½—2 cm. increasing to large sugillations-like areas of the subcutaneous connective tissues, chiefly over the abdomen near the pubis, shoulders and on to the limbs.

(*b*) Extensive haemorrhages and marked swelling of lymphatic glands, especially those of the periportal region, resembling a cluster of very large grapes, and in one case leading to rupture and haematoperitoneum.

(*c*) Multiple haemorrhages into the serosa of parts of the intestines. In one case there were extensive diffuse heamorrhages on the epicardium and endocardium of the left ventricle.

(*d*) Kidneys showed slight degenerative changes and in some, small infrequent petechiae. In one case there was extensive diffuse haemorrhage of the kidney medulla in the vicinity of the pelvis.

(*e*) In every case small consolidated areas were seen in the cardiac lobes of the lung while the rest of the lung was normal. In one case it was complicated by multiple haemorrhages varying from ·5—1 cm. in diameter.

(*f*) Stomach and intestines in some showed slight catarrh with patches of hyperaemia, while in others patches resembling haemorrhages occurred in the submucosa. In all cases except one there was light to acute hyperaemia and swelling of the ileo-caecal valve with early to older ulcerations varying from ·2 to 1 cm. in diameter (probably *Balantidium infection*).

(*g*) Spleen only showed a moderate tumor splenis and in some there were raised reddish areas while the lymphoid tissue was prominent.

(*h*) The wall of the gall bladder was markedly thickened and in a few cases contained blood.

Defibrinated or citrated blood from four of these pigs was injected into pigs at Onderstepoort with positive results. The strain of swine fever virus obtained from this outbreak was used in most of the experiments at Onderstepoort.

(B) Swine Fever in the Northern Transvaal.

In the routine examination of carcases of pigs at the Johannesburg abattoir at the time when the Johannesburg outbreaks occurred in 1934, cases were met with in which there was no clinical evidence of the disease, whereas changes in the lymphatic glands strongly suggestive of swine fever were encountered. In a few instances inoculation of material from these pigs set up swine fever. An attempt was made to trace the origin of infection in some of these cases, but without success. Inspections were carried out in a number of districts in the Transvaal but nothing resembling swine fever could be traced. In some cases blood or organs from pigs which had died on farms were used for inoculation into susceptible pigs, but without positive results.

During the year 1934-35 no cases were reported from Potgietersrust, Waterberg and Zoutpansberg, whereas in 1935-36 one outbreak occurred in Potgietersrust, two in Waterberg and three in Zoutpansberg. During 1937 two outbreaks were diagnosed on two adjoining farms in the Pietersburg district. During 1938 two outbreaks again occurred in the Pietersburg district. In addition there were 2 suspected outbreaks in the Potgietersrust district but these were not confirmed because all the pigs had succumbed. This small number of active infections is probably not a true reflection of the real incidence of the disease in the Northern Transvaal, because apart from the difficulty caused by the fact that wild pigs are natural carriers of swine fever, the control of the disease in domestic pigs is rendered most difficult in that area. From a preliminary census it would appear that in these northern districts there were probably more than 20,000 pigs. These are owned by about 900 European and 1,800 natives. Only about 15 per cent. of these pigs are well bred, whereas 25 per cent. run free and are not under control. The possibility therefore exists that such pigs may from time to time come into contact with wild pig carriers and become infected. These sporadic outbreaks in Northern Transvaal clearly show the need for the present quarantine restrictions. Pigs from there are either allowed to the quarantine section of an approved abattoir for immediate slaughter or in the case of well bred pigs under proper control " a system of quarantining out " is permitted.

(C) Swine Fever in the Western Province.

A disease with high mortality was reported in October, 1933, at the Imperial Cold Storage, Gouda, Tulbagh district, in pigs for slaughter. At first arsenical poisoning was suspected, but analysis of the organs gave negative results. From blood samples *B. suipestifer* was isolated but a vaccine made did not have any protective action. The blood inoculated into pigs at Onderstepoort produced symptoms

of an acute septicaemia with death in three to four days. In these experimental pigs an organism of the *Pasteurella type* was found, and a form of haemorrhagic septicaemia was suspected. Subsequently, however, filtered blood produced similar cases and the conclusion arrived at was that the disease was due to the virus of swine fever.

The infection was probably introduced by pigs from Johannesburg. The Gouda outbreak established a focus of infection for the surrounding districts. Infection broke out on the farm Onverwacht, Wellington, probably introduced by infected pigs' heads and feet purchased by coloured labourers from Hiscocks Bacon Factory at Wellington, which had become infected probably also by pigs introduced from the Transvaal. The spread from these centres was at first thought to be limited owing to the isolation of the infected pigs at the Bacon Factory and Gold Storage. Unfortunately the disease had spread further than was realised. Early in 1934, a further centre was discovered on the outskirts of Cape Town in the pigs of a dealer, but it was possible to dispose of the pigs. Towards the end of 1934 a big outbreak occurred in Retreat, a suburb of Cape Town, and about 300 pigs died. This outbreak was fortunately localized as no distribution of pigs took place. In January to March, 1934, reports were received of a disease in pigs with a high mortality, from a village called Halfmanshof near Gouda and from Riebeek West in the Malmesbury district. In March, 1934, a severe outbreak occurred at a farm Fruit Grove, Wellington. The owner got rid of his pigs not yet affected to a dealer, who lost the majority of them. A farmer at Paarl bought manure from the Paarl Abattoirs and his pigs became infected, heavy losses resulting. By this time the disease had become widespread and quarantine measures with prohibition of movements of pigs were introduced. A large area of country was involved including Cape Town, Wynberg, Simonstown, Belville, Wellington, Tulbagh, Stellenbosch, Malmesbury, Moorreesburg, and Piquetberg, i.e., an area of about 600 square miles.

A natural mountain barrier with only a few passes hindered the spread of the disease to the north and the disease remained confined almost exclusively to the districts mentioned, on the southern aspect of the mountain. The northern mountain barrier was, however, twice penetrated by the disease. In one case the infection apparently went via the Bain's Kloof Pass and Mitchell's Pass to Prince Alfred's Hamlet, Ceres. This outbreak was suppressed at once and did not spread. The nearest infection by road was fifty miles away. The other extension occurred through a pass known as The Rest, to Clanwilliam district, and a farm on the Piquetberg-Clanwilliam road became infected. The outbreak was 70 miles from the nearest infection in the Malmesbury district. The pigs on the farm were slaughtered out and no further outbreaks occurred in the district.

In the infected areas the farming is mainly fruit and wine production with pigs as a sideline to provide fresh meat for coloured labourers. In the Malmesbury and Piquetberg areas grain farming is the main occupation, pigs being allowed free range on the stubble and grasslands. These areas are all closely settled so that spread of infection was facilitated.

During the year 1936, after a quiescent period of some months, the disease again made its appearance in this province under some mysterious circumstances. There was no evidence that infection had been introduced from outside and the indications were that the recurrent outbreaks had their origin in local foci of infection where the disease had remained latent since the 1934-35 outbreak. The outbreaks during the year 1936 occurred as follows: Wellington 2; Caledon 1, Franschhoek 1, and Worcester 1. These infections involved a total of 148 pigs which were all slaughtered off. In respect of the Caledon outbreak (*vide* file 18/16/1 of 1936) the infection extended from Elgin to Villiersdorp.

Several factors apparently contributed to the spread of the disease in the Western Province. Speculators disseminated the disease by the movement of pigs from one place to another, very often in vehicles from which the excreta infected clean premises. A further factor contributing to the spread of infection was the local custom of distributing portions of meat from a carcase to the neighbours. Owing to the custom of using manure from the abattoirs for fertilizing purposes in vineyards and orchards, infection was apparently also spread in this way. Pigs are often allowed to have free range in the vineyards and orchards and not many are kept in styes continuously. The movement of coloured labourers with food supplies such as pig products from farm to farm in the course of harvesting may also have been a source of setting up new infections.

In the control of the infection in the Western Province an attempt was made to forward as many as possible of the healthy pigs on the infected farms to the abattoirs to minimize financial loss. Farmers with farms in contact with infection were advised to evacuate their pigs to the abattoirs to create buffer farms without pigs. Five abattoirs, conveniently situated, were selected for this slaughtering under direct veterinary supervision. Buffer areas were created by keeping pigs on farms adjoining infected ones in styes and temperaturing weekly while a strict census was kept at the same time. The creation of buffer zones enabled many unsuspected outbreaks to be found. The buffer zones extended to those farms situated within ten miles of the in-contact zone. Early in 1935 the disease had burnt itself out in the areas where it had first appeared and when the disease was arrested in March, 1935, about 1,500 farms were under observation.

The disease did not progress as a wave of infection but more often appeared at farms remotely distant from other outbreaks. In some cases the spread was from farm to farm but long jumps were frequent. In the wheat-lands where the pigs ranged freely on the stubble, the spread was very rapid.

A remarkable instance of the spread of infection from a "carrier" occurred on a farm in the Piquetberg area. The farm was infected in July, 1934, when the disease was raging in the area. Nine pigs survived the outbreak one of which was a sow which produced a litter and the sow and young pigs were kept in isolation. When the pigs were four to five months old it was thought that it would be safe for them to mix with the survivors, but when they were allowed to do so they all died of swine fever, seven months after

the original outbreak. Pigs from two of the adjoining farms where the disease had not broken out were allowed to mix with the survivors as well and an outbreak amongst them was the result.

In connection with the carrier question two interesting outbreaks of swine fever occurred in 1939 in the Piquetberg district of the Western Province. The first occurred in April (O.P. file 18/61/1 of 1939) on a farm where the disease appeared in 1934 and after a heavy mortality was slaughtered out. In spite of every effort being made to trace the source of the 1939 infection it remained obscure until a second outbreak occurred on an adjoining farm in October, 1939, and a connection between the two was established. In both the outbreaks the diagnosis was confirmed by histopathological examination and subinoculation into pigs, and paratyphoid was excluded. It was eventually found that a mortality occurred every year about October on the farm where this second outbreak was discovered, but it had not been reported and it would appear that carriers probably existed. A pig from this second farm had been sent to the first farm some months previously and a connecting link between the two was thus established.

These outbreaks are remarkable in this way that since the epizootic which ended in 1935 there have been no further outbreaks in that vicinity until 1939 in spite of not infrequent inspections by officers of the department. It is not clear how this virus was maintained on this infected farm and remained localised for such a considerable period without setting up new outbreaks in the neighbourhood. An investigation is being carried out to ascertain whether other domestic animals such as cattle and sheep may have been carriers of the virus. Recenty Zichis (1939) showed that hog cholera virus was transmitted to sheep both by intravenous injection and cohabitation with infected pigs. Although the virus produced inapparent infection in sheep their blood was pathogenic for at least 21 days following infection.

The slaughter out policy adopted by the Department (see control measures: Appendix VIII) proved a successful method of stamping out the disease. It brought the active outbreaks to a close in March, 1935. Attention was then directed to the "carrier element" namely the surviving pigs as a potential source of reinfection. Steps were taken to ascertain the number of surviving pigs in the gazetted areas and to dispose of them with a view of preventing recurrence of infection from this source. This offered a problem that was not easy of solution. Efforts were directed towards evacuation of surviving pigs to one of the approved abattoirs from:—

(*a*) Outbreaks that occurred at Gouda and Wellington in October and November, 1933, to the date of Government Notice No. 616 of 11.5.1934 and which were not reported.

(*b*) Known outbreaks from the date of the publication of the first prohibition order (Government Notice No. 616 of 11.5.1934) to the application of the slaughter out policy.

In carrying out the slaughter policy in relation to these surviving pigs an attempt was made in all cases to obtain the goodwill of the farmers concerned. Where possible time was allowed to fatten pigs

and allow sows to farrow and wean the piglets. The danger of the carrier element was appreciated by the Farmers' Association and sub-committees were formed and were remarkably helpful in discovering unknown infections and getting the pigs evacuated.

From the initial outbreaks in 1933 to the commencement of the slaughter out policy (December, 1934) about 11,000 were involved and of these more than 8,000 died and about nineteen hundred were evacuated to the Abattoirs. There were 862 survivors left over from the outbreaks and all were slaughtered. Compulsory slaughter was only enforced in three instances, which speaks well for the goodwill of the farmers. The slaughter policy was usually applied on a farm before many pigs had died and in eighty-two outbreaks there were only about 328 deaths from the disease itself. A total of 3,610 pigs was destroyed between December, 1934, and June, 1935, with a valuation of about £1,638, representing compensation at a quarter of the actual value.

COMMENTS ON SYMPTOMATOLOGY.

Steyn (1928) mentions that farmers in the portion of the Transvaal where the disease was first reported, described it as affecting animals of all ages. The disease was sudden in onset and the animals were noticed to be dull, breathing heavily, grinding the teeth and foaming at the mouth. Death might occur within twenty-four hours of the first symptoms but some pigs remained sick for several days, showing a swaying gait. Affected pigs rarely recovered, but if they did they were immune but might develop a further attack later which was usually not fatal. In his own experiments Steyn found the a erage incubation period to be two days but it varied from thirty-six hours to four days. The temperature rose to between 105° and 107° F. but occasionally reached 108° F. Towards the end the animals became comatose and the temperature dropped to subnormal. In about 50 per cent. of cases he noticed bronchitis sometimes associated with broncho-pneumonia, but a cough was rarely noticed. Breathing was laboured and the pulse rate was sometimes 180 to the minute. Occasionally diarrhoea was noticed with blood in the faeces, but more commonly the faeces were hard and dry. When walking the pigs showed an arched back and staggering gait.

Walker (1933) in his article on East African Swine Fever describes symptoms almost identical with those mentioned by Steyn. In addition he mentions cyanosis of the skin, ears, hairless parts of the body such as the under side of the abdomen, and insides of thighs and fore legs.

In his experimental work which covered at least 200 animals the average incubation period was three and a half days and the duration of the reaction four days. Montgomery (1921) had two cases which lived for fourteen days.

Other symptoms mentioned by Walker are vomiting in some cases and abortion in pregnant sows. Vomiting as a symptom in the African disease was noticed by Steyn in an outbreak which he investigated after the publication of his original article.

During the recent outbreaks in the Union, the course of the disease has been very similar to that described by Steyn (1928) and Walker (1933). In the earlier reports from the Western Province, it was stated that many pigs were found dead in the morning without having been noticed to have been sick. The majority, however, lived for two or three days. Blood was noticed in the feaces in some cases and was seen to be oozing from the nose and anus after death in others. In white pigs dark red patches were seen on the ears and under the abdomen. Swelling of the ears was noticed in some cases and the eyes were very bloodshot.

In the Witwatersrand outbreak some of the pigs showed haemorrhages of a bluish red colour under the skin of the body and face. Some of these involved the eyes causing bleeding from them. A number of the pigs were described as having died suddenly without showing signs of illness previously and others died after a very short illness. The history was again very similar to that previously described. A number of pigs, however, recovered but they remained in poor condition and showed a scaly condition of the skin.

During the slaughtering out in the course of the eradication campaign in the Western Province, the opportunity was afforded to examine a few recovered animals, some of which were known to have shown symptoms at the time of the outbreak a few months previously. Eight of these cases were examined. Six of them were in excellent condition and apart from slight evidence of swelling of the joints were quite normal in appearance. One of the others was in poor condition and showed marked swelling of the joints. Another showed poor condition with slight swelling of the joints.

In the recent experiments carried out at Onderstepoort the great majority of the cases produced have been acute. The most obvious symptom noted besides the severe hyperthermia (temperature up to 108° C.) is weakness in the hindquarters shown by swaying gait. The average incubation period has been three days and the duration of the visible illness just over three days. The shortest incubation period has been between thirty-six and forty-eight hours. In some of the cases produced by the blood of recovered pigs the incubation period has varied from five days up to even twenty-two days. The addition of swine fever antiserum delayed the reaction up to eleven days in one case and eighteen days in another.

These long incubation periods are unusual and never seem to occur with material from actively infected cases except if antiserum be added to the virus.

The duration of the disease has usually been three to four days, but periods of seven to fourteen days have occurred. Only two pigs have survived out of over one hundred experimental animals. In both these cases recovery was slow and the temperature fell gradually to normal over a period of one to two weeks. In one case constipation was very marked for about a week after the temperature fell to normal but in the other case recovery was uneventful. This latter pig was tested two months after recovery with a virus from a different source to that with which it had previously been inoculated but showed no reaction. The control pig died on the sixth day after inoculation from acute swine fever.

PATHOLOGICAL CHANGES IN ACUTE CASES.*

In acute cases general changes such as loss of condition as a result of the disease are almost unknown, in fact the animals which succumb are frequently reported as found dead without previously having been noticed sick. Rigor mortis sets in early.

Integument.

The skin almost invariably has a patchy or diffuse purplish colour (cyanosis) which naturally is more striking in light coloured animals, but can be seen even in black animals, e.g., in the axilla and other parts covered by thin skin. More rarely there may be isolated single or multiple irregular, small dark red patches or spots up to one or two cm. in diameter (see Fig. 1). These usually have a raised appearance covered with tense, shiny epidermis and are distributed particularly on the abdomen. Microscopically one finds engorgement and stasis of the smaller blood vessels and capillaries to account for the cyanotic appearance.

Fig. 1.—Skin showing multiple haemorrhages.

Endovascular hyalinisation and thrombosis can be seen in some cases and, when advanced, leads to localised blood extravasation into the cutis. Other evidence of necrosis, such as karyorrhexis and rarely total disintegration with secondary infection, may be seen according to circumstances.

(Note.—* The histological examination was carried out by Dr. A. D. Thomas, of the Pathological Section, Onderstepoort.)

Abdomen.

As the animals feed well till near the end, the abdomen is usually well filled and presents no great departure from the normal. The peritoneal cavity may contain a variable amount of clear fluid, which sometimes may be somewhat turbid or show fibrinous tufts and strands. The peritoneum is usually smooth and glistening. At times, however, there may be ecchymoses and even extensive reddening with or without fibrinous adhesions.

Thoracic Cavity.

The thoracic fluid is usually slightly increased and may reach a considerable amount where pulmonary oedema is marked. On occasion a turbid, almost milky exudate may be present, in which fibrinous shreds may be seen.

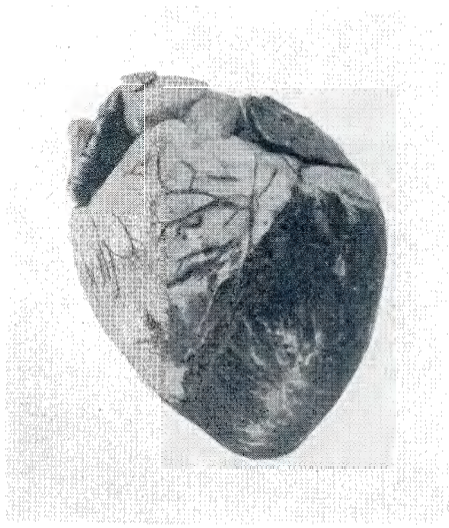

Fig. 2.—Heart. (Spec. No. P.S. 14742.) Extensive subepicardial haemorrhages. ×½.

Heart.

The pericardial sac often contains an increased amount of clear watery fluid. At times the fluid may be turbid even to the point of being milky, when fibrinous strands and adhesions may form important constituents of the exudate.

Sub-epicardial and sub-endocardial ecchymoses are not infrequent and on occasions may be very extensive and confluent. (See Fig. 2.)

Haemorrhages of variable extent are also to be seen in the substance of the myocard. The endocardium, valves and atria do not as a rule present any changes. The haemorrhages in the

myocardium can usually be found associated in histological sections with vascular obstruction (thrombosis), hyalinisation of wall, karyorrhexis, etc. It is of course possible to find microscopic interstitial haemorrhages even where these may not be visible with the naked eye.

Lungs.

In many cases the lungs show little or no change. Cyanosis in a variety of shades and pattern is quite a common feature. In some cases oedema may be pronounced, to the point of showing wide infiltrated interlobular spaces, and profuse froth and serum oozing from cut surfaces. In other cases reddish irregular patches or spots (haemorrhages) appear singly or disseminated throughout the whole organ. In such cases also the consistence of the organ appears altered. There is consolidation, partial or complete, of some areas (broncho-pneumonia). Sometimes the darker areas seem partly atelectatic rather than simply inflammatory. Exudate in appearance varying from frothy blood to turbid, greyish slime can be expressed from the cut surfaces.

Fig. 3.—Lung. (Spec. No. P.S. 7556.) Thrombosis, oedema and hyperaemia. ×200.

Microscopically the picture includes any one or more of the following changes according to circumstance, hyperaemia, oedema (alveolar and interstitial), collapse of lung (partial atelectasis), broncho-pneumonia, pleuro-pneumonia, with varying amounts and nature of exudate (serous, catarrhal, haemorrhagic, fibrinous, necrotic) (see Fig. 3). Hyalinisation of the alveolar walls (capillaries) and blood vessels can be pronounced in outstanding cases, but is not easily recognised in the majority of cases. Hyalinisation and thrombosis of pulmonary veins is a relatively frequent occurrence, and necrotic effects especially diffuse karyorrhexis usually accompany them.

Spleen.

The spleen may show little or no change visible to the naked eye, but usually it is swollen to a variable extent, occasionally reaching a size two or three times the normal. This swelling may be uniform and affect the whole organ, or it may involve one extremity only say $\frac{1}{3}$ or $\frac{1}{2}$ of its length, or even be in the form of isolated, bulging, darker areas distributed at random in the pulpa. The swollen portions have a tense shiny capsule, thick rounded borders, and a greyish blue black colour as seen through the capsule. On section the colour is the deep purplish black of stagnant blood. The trabeculae are indistinct and seem to be stretched and overflowed by the pulpa. The lymphoid follicles are sparse, small and obscured by the bulging pulpa. The colour and consistence of this swelling indicate masses of coagulated blood in the pulpa rather than proliferative processes. In those cases in which swelling is not evident the pulpa appears normal in structure and of the usual brick red colour. In some, however, one may see small brighter red spots or zones scattered in the pulpa usually associated with the follicles.

Histologically the most striking and constant changes are briefly:—

(*a*) Hyalinisation and effacement of the Schweiger-Seidel sheaths or splenic ellipsoids.

(*b*) Congestion of pulpa, haemorrhage or infarction.

(*c*) Spreading apart and atrophy of lymphoid follicles.

(*d*) Pyknosis and karyorrhexis of reticulo-endothelial and lymphocytic cells.

(*e*) Endo-vascular changes—hyalinisation of wall, desquamation of endothelium, thrombosis and necrosis.

(*a*) *Effacement of Schweiger-Seidel Sheaths.*

The Schweiger-Seidel sheaths or so-called splenic ellipsoids are bulb-like terminal enlargements of the final twigs of the pencil arteries. In the pig, as is well known, they are particularly prominent and distinct as roundish or oval structures composed of a mass of concentrically arranged endothelial cells. These bodies appear to form a delicate but loose meshwork through which the blood from the arterioles has finally to pass before it reaches the

pulpa and sinuses. They are distributed throughout the spleen roughly in the proportion of 10 to 20 to each malpighian follicle around which they tend to form an irregular ring.

In all cases of Swine Fever it was noticed that the Schweiger-Seidel sheaths were affected to a greater or lesser extent. The majority show disintegration, smudging or total disappearance of the ellipsoid structure, so much so that no indication whatever may be left to mark their previous existence and situation.

Very often, however, shreds, or islands of hyaline substance with faint oval outline indicate where these structures existed. These islands which are usually loose-meshed, generally contain much blood, and sometimes there are indications of fibrin deposition and even of complete thrombosis. In a very few cases only do the ellipsoids remain fairly distinct in outline, although even here one may see very definite indicatons of necrobiosis of the delicate cells forming the spongy mass.

Probably these changes represent progressive steps in the destruction of the ellipsoids. At first the sheaths retain their form, only the delicate cells which make them up undergo pyknosis and beginning karyorrhexis. Then the cytoplasm becomes eosinophilic, hyalinised and fuses to a homogeneous mass or which is more usual, retains a certain sponginess with erythrocytes packing its meshes. In advanced stages in which haemorrhage is very extensive even this hyaline substance tends to disappear. It would seem as if the flood of blood here causes compression or disruption of these hyaline remnants or both, so that nothing is left eventually whereby these structures could be identified and their place is taken by the all pervading blood which congests the pulpa.

(b) Congestion of the Spleen.

The bluish black colour and swelling of the spleen mentioned earlier is seen histologically to be due to excessive accumulation of blood in the splenic pulp. Of course it is difficult to see whether this is to be considered as hyperaemia, haemorrhage or infarction on account of the complicated vascular system of the spleen. The fact that this accumulation of blood is often localised to more or less defined areas, the presence of fibrin in the ellipsoids and thrombosis of vessels are indications in favour of regarding it as of the nature of a haemorrhagic process.

(c) Spreading apart and Atrophy of Lymphoid Follicles.

In a spleen markedly swollen and congested as above the histological picture is quite characteristic. The pulpa cells are spread apart by the masses of blood between them and the same thing takes place as regards the malpighian follicles. Not only do the lymphoid centres appear fewer in a given field, i.e. further apart in the expanse of blood laden pulpa, but the follicles themselves are often much reduced in size and contain less lymphocytic cells, many of which may have undergone karyorrhexis. This process of disintegration seems to take place especially just around the central artery.

The shrinkage of the lymphoid follicle may also partly be due to atrophy resulting from the the pressure exerted by the accumulated blood as has been suggested by Seifried (1934). The latter goes so far as to link up the leucopaenia seen in swine fever with this phenomenon.

(*d*) *Pyknosis and Karyorrhexis of Reticulo-endothelial and Lymphatic Cells.*

Karyorrhexis of endothelial cell nuclei in all tissues is characteristic of this disease. Pyknosis and to a lesser extent other regressive nuclear and cellular changes are also seen. This condensation and fragmentation of nuclear chromatin is generally extensive and affects endothelial as well as lymphocytic and allied cell types. It can be seen in the lining cells of blood and lymphatic vessels everywhere, but is particularly noticeable in the lymphatic glands and spleen where the reticulum and splenic cells appear very susceptible. *This change so conspicuously affecting endothelial and lymphocytic cells everywhere would seem to be the direct result of damage done by the virus* and can scarcely be attributed to, or confused with the more localised necrotic changes that arise secondarily as a result of thrombosis and infarction.

(*e*) *Endo-vascular Changes.*

Apart from the alterations mentioned above in regard to the Schweiger-Seidel Sheaths the spleen on account of its peculiar anatomical blood vascular arrangement, does not present such marked intravascular lesions as other organs. Nevertheless it is possible to demonstrate hyalinisation, swelling and desquamation of endothelium and thrombosis in some of the splenic vessels.

Lymphatic Glands.

The lymphatic tissues in general and certain groups of the lymphatic glands in particular afford perhaps the best and most constant single naked eye characteristic lesion in Swine Fever. The changes to be noted consist essentially in extravasation of blood into the reticular spaces and lymph sinuses. The glands so affected may show anything from a deep bluish black colour, through and through, to lighter diffuse or speckled red shading or to the normal appearance. Between these extremes all grades of intensity and distribution of the haemorrhagic effects may be seen (see Fig. 4). It is quite common to find glands showing a more or less well demarcated dark red peripheral zone or rind where the haemorrhage has taken place into the outer lymh sinuses mostly.

Outwardly the glands are swollen, and on section the cut surface may be moist or even watery, but in most cases presents a more or less firm and mat appearance indicating the presence of coagulated fibrin. Occasionally lymph glands may show a greyish mottled effect, an indication that thrombosis and subsequent necrosis are already far advanced.

Of all the lymphatic glands of the body the most constantly and prominently affected groups are the gastric and periportal. Next come the mesenteric, renal, sublumbar, thoracic, maxillary and then the other superficial groups. In the latter it is very unusual to find anything but a diffuse light reddening and swelling.

It is interesting to note that any of the groups mentioned may without any apparent reason show pronounced haemorrhagic changes over and above some or all of the others in a given case, and conversely in some cases the majority of glands may show damage to a similar extent.

Fig. 4.—Lymphatic glands. (Spec. No. 11700.) Extensive haemorrhagic appearance. $\times\frac{3}{4}$.

Histologically the lymphatic glands also present a fairly typical picture which may be summarised in the following two sets of alterations:—

A.—Primary damage to the endothelial and lymphatic system as evidenced by the diffuse karyorrhexis of these cells, and B. secondary circulatory disturbances and their complications arising from A., e.g. blood extravasations, lymph stasis, thrombosis and necrosis.

A.—Extensive and diffuse karyorrhexis of the reticulo-endothelial nuclei in all the lymphatic tissue is *such a constant and characteristic feature of the histological picture that it deserves special attention.*

In the earlier stages of the disease or in organs so mildly affected that other histological changes are not yet apparent, karyorrhexis is usually already discernible here and there in a number of cells to an extent which at any rate enables one to suspect if not to diagnose the disease definitely . A study of stained smear preparations made from the pulp of glands or spleen gives one a very good insight into some of the nuclear changes which take place in the endothelial cells and lymphocytes. The nuclear chromatin of some of these cells seems to become flocculated into what appears to be a loose and bulkier, fluffy mass of chromatin within the nuclear membrane. Another and more striking phenomenon is seen in a great proportion of cells. It consists of " condensation " of the chromatin (pyknosis) into denser and darker shining masses which tend to divide rosette wise into smaller globular or tear-drop shaped fragments of chromatin (see Fig. 5). In many instances it appears as if the chromatin was

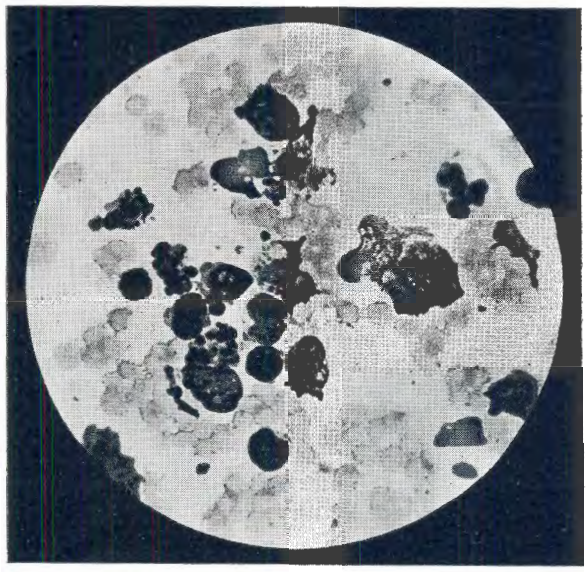

Fig. 5.—Specimen No. 1173. Smear periportal lymphatic gland. Group of karyorrhetic cells. ×700.

being squeezed out not only from the nuclear membrane but from the cytoplasm as well, so as to be outside the cell entirely. Such spongy nucleus-less cell remnants are not infrequently encountered in smear preparations. In addition to the nuclear changes one frequently sees in gland smears, but also blood and other organ smears, globular fragments of cytoplasm floating in the plasma. These vary in size from 1 or 2 to 10 or 15 μ. They stain very faintly with Giemsa solution and often appear as mere bubbles,

their colouration approximating that of the lymphocyte cytoplasm, i.e. very light grey blue. Actually the cytoplasm of a number of large lymphocytes show the bulging pseudopodia which might be taken as indications that these fragments are " pinched " off from parent cells. On the other hand these fragments which at times may be very numerous, are more likely to originate from endothelial cells which have broken up or desquamated at the time that they lost their nuclei as indicated above.

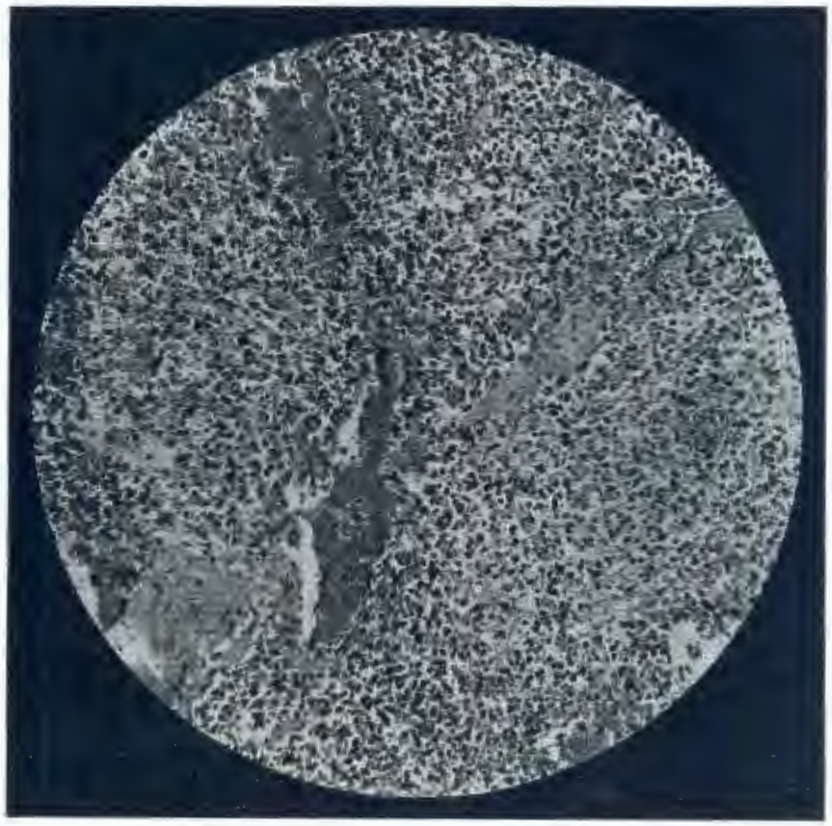

Fig. 6.—Spleen. (Spec. No. 14472.) Thrombosis and hyalinisation of S.S. sheaths, also karyorrhexis and lymphoid atrophy. ×135.

B.—Even in glands mildly affected there is always an engorgement of the capillaries and pre-capillaries in the reticular tissue. Normally these capillaries are scarcely distinguishable as endothelial tubes unless they contain blood since their wall consisting as it does of a single layer of endothelial blends with the rest of the reticulum. As the disease develops, this thin one-celled wall becomes thickened, swollen, blurred and macerated looking. This hyalinisation may present a perfectly homogeneous pink staining appearance (H.E.) sometimes having a finely lamellated, spongy or even vacuolated structure (see Figs. 7 and 8).

Fig. 7.—Specimen No. 15168. Hyalinisation of one vessel wall. Karyorrhexis. ×240.

In the earlier stages of the disease or in lightly affected glands the reddening may be due to hyperaemia only, and the vessel walls may scarcely show any recognisable changes. In the more advanced stages, however, such as is usually the case with the periportal or renal lymphatic glands, the deep reddening and swelling is due to extensive blood extravasation into the reticulum and even into the lymph sinuses. This extravasation may reach such proportions as to involve the whole gland in one large practically confluent haemorrhage or haematoma, in which the remnants of lymphoid follicles lie embedded, compressed, and isolated from one another. Not infrequently the haemorrhage into the peripheral and other lymph sinuses forms large pools of blood which overshadow the blood contents of the glandular tissue proper and give rise to the "black rind" or "nutmeg" aspect of these organs. Occasionally very little extravasated blood may be seen in the gland and yet the tissue may be extensively damaged showing necrosis, advanced karyorrhexis, hyalinisation of vessels and cellular disintegration.

The lymph spaces in such instances usually contain a network of fibrin and the blood vessels are more or less completely plugged by thrombi, thus probably interfering early with the blood supply.

The very extensive blood extravasation seems definitely to follow on the damaged state of the blood vessels especially capillaries and precapillaries. The red blood cells may frequently be seen caught in the meshes of a loose and spongy (hyalinised) vessel wall. In arterioles and precapillaries the inner lining of endothelial cells may be seen to swell up, disintegrate or desquamate in shreds, and even to become incorporated in the thrombi that may form here.

Fig. 8.—Specimen No. 15168. Hyalinisation of several vessel walls. Haemorrhages and karyorrhexis. ×200.

Stomach.

This organ shows hyperaemia with a fair degree of constancy. This occasionally may be severe even to the point of giving rise to haemorrhage. The stomach is usually full of food, the mucosa is covered with mucus and the fundus portion a light diffuse greyish pink to red, sometimes mottled and with erosions or ulcerations. In

the worst cases brownish deposit or actual fibrin pseudomembrane or blood clots are present. In the milder cases, histologically one can see engorgement of the capillaries of the mucous membrane. In more advanced cases there is also fibrin present which then usually forms a clot occluding the vessels completely or in part. Vessels which undergo complete thrombosis rapidly lose their distinctive structure and become a blurred mass of protoplasm and fragmented chromatin. The mucosa over areas so affected becomes eroded at an early stage, with a variable loss of epithelium which leads more rarely to ulceration and bleeding. Infarction as a result of thrombosis of blood vessels is not infrequent.

Intestine (small).

The small intestine usually contains normal looking ingesta, and may not show any obvious abnormality. On the other hand a slight reddening of the mucosa with catarrh is a common occurrence and may be accompanied by subserous ecchymoses. Occasionally there may be a marked hyperaemia and a few cases presenting a severe haemorrhagic or even diphtheroid enteritis have been encountered.

Intestine (large).

The caecum, especially in the vicinity of the ileo-caecal valve frequently shows some degree of swelling and reddening. This region deserves special attention, since even the normal gut may have a misleading appearance. This small area close to the ileo-caecal valve of the caecum consists of lymphatic tissue mostly. It has a thicker, darker and wrinkled appearance and is grossly pitted by the numerous glandular crypts in this region which in turn are commonly filled with necrotic plugs and mucus which can be expressed out or scraped away without great difficulty. It is of course a well known fact that "boutons" in swine fever do occur in this region, but it is important *not* to confuse them with the dead detritus plugs of the normal crypts. The colon itself may show degrees of hyperaemia patchy or diffuse which in some cases become haemorrhagic. Extensive diffuse diphtheroid inflammation is noted occasionally. The lymphoid follicles may be prominent and sometimes show evidence of haemorrhages and necrosis, but it is noteworthy that typical "boutons" have not yet been seen in acute cases of swine fever in this country. In microscopic sections extensive haemorrhagic and also diphtheroid enteritis have been seen on rare occasions. At a rule, however, the vascular changes, haemorrhages and karyorrhexis are limited to lymphoid tissue, i.e. the solitary follicles of the colon and more particularly that of the ileo-caecal valve.

Liver.

The liver usually appears swollen and richer in blood than normal. When cut the surface thus exposed oozes out with a thick coat of blood. Irregular darker red patches or spots are sometimes seen through the capsule (haemorrhages). The lobulation remains distinct, but a speckled or nutmeg appearance may be seen due to lighter lobules or portions thereof and darker blood-filled ones.

Microscopically the marked congestion of the parenchyma is in the nature of a stasis which is not infrequently accompanied by atrophy of the liver cells. The liver sinuses are widened and blood-filled in parts to the point of forming small blood pools. The stasis usually extends over irregular portions of the liver lobules mostly towards its centre. The hepatic cells here may be atrophic or merely show compression, distortion due to blood accumulation or even necrosis with karyorrhectic nuclei of scattered cells.

The necrotic changes in the liver are fairly typical though inconstant and rather indistinct. Unlike those seen in paratyphoid the necrosis is quite diffuse, not sharply demarcated; in fact it seems at times as if single scattered cells are affected, while sometimes all the cells in part of the lobule seem to suffer. The stellate cells show karyorrhexis fairly constant, and the liver cells also tend to hyalinise and their nuclei to break up and disappear.

The gall bladder has been seen sometimes to show haemorrhagic and fibrinous inflammation. In such cases the wall is greatly swollen, red and oedematous and fibrin and blood clots are mixed with the bile.

Pancreas.

In a certtain proportion of cases this organ may show quite extensive and severe lesions while at times it remains practically unaffected. The following have been noticed:—interstitial and interlobular haemorrhages from small scattered ecchymoses to large extravasations reaching haematoma proportions. This organ frequently shows extensive and characteristic necrosis. This change is better seen when cutting through the gland, thus exposing the marmorated effect due to a combination of necrosis (greyish white, mat streaks and patches), haemorrhages (dark red patches) and fat saponification (yellowish white blotches) of the interlobular fat; the whole giving rise to a variegated mottling. In severe and advanced cases the whole pancreas may appear like a soft brownish mass, with deep greyish mottling interspersed with dark brown or black streaks of extravasated blood. These changes are very striking in histological sections where necrosis of the parenchyma in certain areas with attendant thrombosis and general karyorrhexis and disintegration of gland cells give rise to the greyish mat discolouration. The mottling is completed by the irregular haemorrhages and by the contrast between the normal fat and patches of saponified fat (fat necrosis) with their large fat cells filled with fan shaped fine acicular crystals of fatty acids or soaps. When the areas of necrosis are large the pancreas cells disintegrate rapidly and become a chromatinless mass of cell remnants, the typical karyorrhexis being seen only at edges near the healthy tissue.

Kidneys.

The lesions in the kidneys are not constant. In a large proportion of cases there is nothing to be seen with the naked eye. For the rest haemorrhages in various grades of intensity and localisation may be seen. These vary from the smallest pinpoint red spots scattered

in the cortical substance, to almost confluent red mottling of the outer and even inner zones. In extreme cases these may lead to large haematomata between the renal capsule and the kidney even extending into the renal fat. Pale or light patches and striations of the cortex may also be seen. The usual vascular changes in the kidney when present take the form of multiple pin point haemorrhages which arise apparently by extravasation from the smaller intertubular vessels (see Fig. 9).

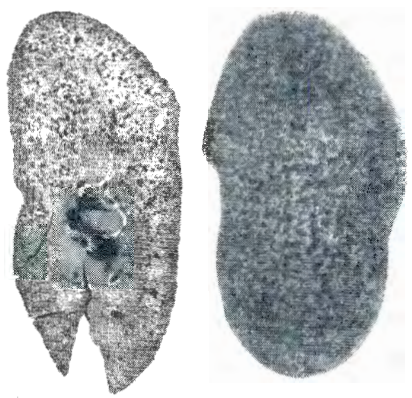

Fig. 9.—Kidney. (Spec. No. 6409.) Multiple haemorrhages. ×½.

Although the glomeruli are composed largely of endothelial cells there is remarkably little change (karyorrhexis, etc.) in this structure except in rare and very severe cases of lesion of the kidney, (see Fig. 10) where the hyalinisation and porosity of the blood vessel wall is clear and the consequent manner of extravasation obvious.

Degenerative processes of varying degree are also commonly seen. Albuminous hyaline substance in the tubular lumen is a common occurrence. This usually goes paired with a granular cytoplasmic disintegration which may even go over to hyaline droplet formation. Karyorrhexis of the tubular epithelium is rare and even the endothelium of the glomerulus shows this only in exceptional cases as mentioned above.

Adrenals.

The adrenals have been found on several occasions to show haemorrhages in the cortex and medulla. Other lesions of significance were not seen.

Urogenital System.

The urinary bladder, ureters, urethra, uterus and ovaries and testes of cases examined have not shown significant lesions beyond small occasional haemorrhages which could be correlated with similar occurrences elsewhere. No specific microscopic changes could be found either.

Fig. 10.—Kidney. (Spec. No. 15385.) Advanced hyalinisation and extravasation, glomeruli. ×200.

Brain.

Congestion of the meninges is a fairly frequent occurrence and it is not always easy to assess its significance. Actual haemorrhages may also be present but are usually very small and not easily seen with the unaided eye. Microscopically small perivascular haemorrhages are seen in some of the cases. They are usually localised immediately around and along the blood vessel or under the pia mater (see Fig. 11).

Fig. 11.—Brain. (Spec. No. 14818.) Extravasations from brain showing karyorrhexis. ×135.

Occasionally the extravasation may extend into the brain substance proper. The blood vessels and capillaries of the brain do show hyalinisation of their wall and karyorrhexis of their endothelial lining, but this is nowhere as distinct or constant as that seen in the lymphatic glands. The vessels frequently contain clots of hyaline or fibrinous nature (thrombi) which may fill the lumen totally or only in part. There is little doubt that the extravasation taking place originates from the blood vessels themselves since the blood very frequently forms a sort of mantle along the adventitia. Perivascular round cell infiltrations such as are described by most workers on swine fever are conspicuous by their absence. Of course, Seifried (1934) maintains that the occurrence of these infiltrations as a diagnostic lesion can be very misleading and it is even being suggested that they are the result of a totally different aetiological cause (virus) which often but not necessarily coincide or precedes Swine Fever.

Blood.

Only a few and rather preliminary observations were made to date on the blood changes. These, however, will serve to show that there are important changes in the blood and that some of these agree to some extent with those recorded in swine fever in Europe.

The following figures were obtained in counts made before inoculating and during the course of the disease, on experimentally produced cases:—

Table I.

Pig. No.	Date.	W.C.	R.C. Million.	Red. precip.	L.	N.	E.	M.	B.	Remarks.
1137	7/7/36	7,400	7·84	32	29	60	1	9	1	Inoculated virus 7/7/36,
	10/7/36	5,400	8·41	40	49	48	1	2	0	died S.F. 11/7/36.
1148	7/7/36	18,600	9·87	51	80	14	3	3	0	Inoculated virus 7/7/36,
	10/7/36	11,100	7·97	34	51	45	3	1	0	killed *in extremis*, 11/7/36
	11/7/36	14,000	7·12	40	66	26	0	8	0	
1147	17/7/36	16,400	5·77	27	34	56	0	10	0	Inoculated virus 17/7/36, died 19/7/36, before 2nd count.
1154	17/7/36	8,800	7·91	37	68	27	1	4	0	Inoculated virus 17/7/36,
	20/7/36	10,300	5·52	28	60	34	3	2	1	died S.F. 20/7/36.
1165	25/7/36	27,100	8·89	37	32	64	0	4	0	Inoculated virus 25/7/36,
	27/7/36	7,300	5·49	27	54	39	1	6	1	died S.F. 30/7/36.
1185	25/7/36	20,200	8·03	37	42	53	0	5	0	Inoculated virus 25/7/36,
	27/7/36	11,400	7·74	34	43	55	0	2	0	killed 31/6/36 *in extremis*.

It will be noted that a decrease in the white count as well as in the red count is a frequent occurrence although by no means an absolute rule. The differential count as it stands does not present much of significance, the figures being too erratic and based on too few observations necessitated by the very short course of the disease before the fatal ending. Blood smears made from advanced cases show as a rule various degrees of poikilocytosis and anisocytosis. Often there is quite a fair percentage of normoblasts and Jolly bodies. Of some diagnostic significance is the karyorrhexis of the nuclei of the large lymphocytes and monocytes as seen in preparations made from the pulp of glands or spleen. The nature of this karyorrhexis and the resultant broken chromatin is fairly typical in that the chromatin appears to be squeezed out of the nuclear membrane in the form of " tear drop " or irregular blobs of various sizes and shapes. A number of these chromatin fragments are to be found lying free in the preparation separate from large lace-like or loose spongy masses which would appear to be the nuclear membrane and spongy tissue divested of its chromatin. Small rounded and very faintly staining bubbles or globules are also frequently encountered. They vary in size from about a fifth to double the size of an ordinary red blood cell. They are to be found lying free among the cells of a gland or spleen pulp preparation. Often they tend to adhere to other cells particularly monocytes and may also be phagocytozed by large macrophages. These bodies are regarded as

fragments of cytoplasm probably arising from disrupted cells having lost their nuclei or by a process of pinching off a cytoplasmic pseudopodia such as are seen protruding from the cytoplasm of large lymphocytes or monocytes.

PATHOLOGICAL CHANGES IN CHRONIC CASES.

The changes recorded below were observed in natural chronic cases in the Western Province outbreak in 1934. The sick pigs were usually one or more of the few remaining alive 14 days and up to a month after the onset of acute outbreak and mortality.

It is interesting to note that in many of the farms visited the only surviving animals were litters of very young suckling piglets farrowed at the time of the outbreak. These appeared not to have contracted the disease at all and grew up perfectly healthy.

The recovering or chronic cases were usually young pigs but occasionally mature pigs were also seen in the chronic stages. These animals were all emaciated and in a very miserable condition. The younger animals showed swellings—usually on the joints (carpal and tarsal)—but also along the tendon sheaths, stifle, phalangeal joints, under the maxilla and on the snout. These swellings were non-painful, soft and sometimes fluctuating slightly. On section they consisted of large accumulations of yellowish grey matter (fibrin) within a fibrous tissue walled cavity. The skin around was also oedematous.

Some of the swellings were due to enlarged lymphatic glands, e.g. sub-maxillary, which on section showed a greyish yellow firm necrotic appearance.

The heart and heart sac in many cases showed extensive pericarditis. The pericardial sac being distended with a milk or dirty greyish fluid in which numerous floccules and shreds of fibrin were present. The epicardium was usually thickened and covered with a greyish white, soft deposit of exudate of necrotic appearance.

The spleen often was normal in appearance but in some cases there were dark firm enlargements of somewhat nutmeg appearance. On section pericapsular fibrinous and fibrous adhesions were also seen.

The lymphatic glands varied a good deal in appearance. Some were swollen and necrotic, i.e. with greyish mat pulp, others were dark reddish grey and firm, while others still were swollen and red or mottled or even very little changed.

The lungs often showed varying degrees of catarrhal bronchopneumonia. Occasionally the lung was affected in patches but more often only in portions of the apical and cardiac lobes.

The intestinal tract was carefully examined for possible changes but in no single instance were lesions even suggesting boutons encountered.

The kidneys did not present much of significance but degenerative changes were to be seen.

Blindness in one or both eyes, accompanied by atrophy of the bulb and opacity of the cornea which developed during or after the disease was a feature seen in three pigs.

Incidence of Lesions.

The foregoing description of lesions represents a summary of the pathological changes seen in over 150 cases of Swine Fever, both natural and experimentally produced.

The histological examinations for the purpose of this study included material from some 197 pigs, 155 of which died or were killed while suffering from the disease. There were thus 42 animals dead from a variety of causes other than Swine fever for a comparison of the organ changes and determination of their relative value or specificity in diagnosis.

An analysis of the relative frequency of significant lesions in the chief organs as given below is of some interest as an indication of the organs best suited for histological diagnosis of this disease. In all cases included in this table, swine fever was diagnosed histologically in one or other organ.

Table II.

Organ.	No. Examined.	Positive Lesions.	Negative.	Percentage Positive.
Lymphatic gland (periportal)	131	127	4	97
Spleen	133	128	5	96
Liver	106	86	20	81
Lung	61	39	22	64
Kidney	117	60	57	51
Brain	76	37	39	48

The number of other organs examined was too small to permit of their inclusion on a comparative percentage basis. It will be seen, therefore, that positive histological diagnosis using one organ can only be established roughly to the extent of the order indicated in the percentage column.

In practice, however, and provided the spleen and lymphatic gland as well as some of the other organs are included the chances of arriving at a satisfactory diagnosis are even better than when relying on a single organ. These figures naturally refer to organs of animals dead from the disease or killed in a fairly advanced stage of its development. The number of positive cases established histologically would probably fall rapidly as soon as organs from animals in the earlier stages of the disease were examined.

Significance of the Lesions of Swine Fever in South Africa.

It is fairly clear from the foregoing descriptions that the main pathological changes in this disease are the results direct or indirect of a marked interference with the vascular system.

Since a virus is accepted as the aetiological agent of this form of Swine Fever we have to assume that it falls in River's third group, namely one which causes a straightforward necrosis of the cells affected. There is also ample evidence of the cell specificity of the virus since the lesions at the onset of pathogenesis are practically confined to cells of the endothelial type, in other words, the lining cells of capillaries, precapillaries, veins, arteries, lymphatic vessels, reticulo-endothelial tissue, etc. In fact some of the more spectacular naked eye lesions like the haemorrhages, extravasations, thrombosis, infarction, etc., are probably secondary vascular accidents and complications arising out of the damaged vessels.

Once the full significance of the changes to the capillary wall is grasped (and lesions in a few advanced cases of this disease leave one in no doubt on this score) it is a fairly simple matter to understand or explain the apparent inconstancy of the vascular changes in various organs e.g. why haemorrhages occur sometimes in the kidney, at other times in the lymphatic glands, in the brain or any other organ, depending apparently only on the degree of damage done to the endothelium of that particular organ or tissue in that animal. In other words it is not the presence of haemorrhages in a particular organ that is diagnostic of the disease but rather the manner in which such haemorrhages arise i.e. hyalinisation of vascular walls.

When these *histological changes* enumerated by Thomas in *the South African cases of Swine Fever* are compared with those under European conditions, as described by Geiger (1937), the very close relationship becomes apparent. Unfortunately Geiger did not have an opportunity of studying the changes brought about by the African virus and relied on the observations of Walker and Steyn who, however, did not undertake a very intensive histological examination.

In the acute form of the *European disease* Geiger records a marked septicaemia characterised macroscopically by haemorrhages in various organs, especially in the lymphatic glands, urinary system, gastro-intestinal canal, and in the skin. The European virus of Swine Fever in the first instance causes changes in the vascular endothelial cells of the intima and a necrobiosis or hyalinisation especially of the capillaries and precapillaries, etc., etc.

EXPERIMENTAL OBSERVATIONS.

The work on the etiology of Swine Fever which has been done in South Africa since 1932 may be conveniently discussed under the following: —

(1) Virus studies.

(2) Complications with secondary bacterial infections.

(3) Bacterial diseases of pigs resembling Swine Fever.

(4) The relationship between the European and African viruses.

(5) Part played by carriers.

(6) Study of the disease in small animals.

(7) Immunisation against Swine Fever in South Africa.

(1) Virus Studies.

In previous outbreaks of swine fever in South Africa, Steyn (1928) found that he was dealing with a virus which would pass a Seitz filter. Both Montgomery (1921) and Walker (1933) were able to show that the virus of East African Swine Fever was filterable.

All investigators who have worked on the subject have come to the conclusion that Swine Fever found in Africa is caused by a filterable virus. Geiger (1937) did a number of filtrations using both Berkefeld and Seitz filters and came to the conclusion that the virus from South Africa was filterable.

With material from the Western Province outbreak in 1933 and 1934 a series of pigs was inoculated and in every case death occurred within a week with a marked temperature reaction and typical post-mortem lesions. Owing to the fact that in a number of cases a *Pasteurella* organism was isolated from them and in the material obtained originally from the Western Province, *Salmonella cholera suis* was found, some element of doubt existed as to whether the disease was really Swine Fever.

In the filtration experiments (see Appendix 1) there seems to be no doubt that the recent outbreaks in the Witwatersrand and in the Western Province were due to the filterable virus of swine fever. In the experiments carried out at Onderstepoort it would appear that mild reactions may occur with some filtrates and that the virus may in some cases fail to pass a Seitz filter.

Survival of the Virus.

Steyn (1932) quotes the experience of Montgomery in East Africa that the virus of that country was viable 536 days in oxalate-carbol-glycerine, that blood allowed to decompose for 16 days was still virulent and that styes left unoccupied for 5 days after a dead pig was removed, were not infective. Steyn found that blood allowed to decompose for 70 to 84 days became non-virulent. He was able to show that blood preserved in an ice-chest for a year was still virulent. He confirmed Montgomery's observation that styes remain infective for less than 5 days after removal of dead pigs. He quotes a case, however, where a farmer put pigs into styes, three weeks after removal of infected pigs, and the new pigs developed the disease.

In an experiment on 23rd June, 1934, two pigs, 1000 and 1003, were inoculated with 10 c.c. each of a mixture of pig bloods kept from Steyn's experiments in 1928. One of these pigs, 1003, reacted and died of acute swine fever in 8 days. The other pig reacted and died 18 days after inoculation. It apparently caught its infection from

contact with the other pig as it reacted after its mate died. The blood in this case was about 6 years old but had been stored in a cold room in the dark.

(2) Complications with Secondary Bacterial Infections.

From what has been described in the previous pages one must conclude that swine fever is caused by a filterable virus, but the question arises to what extent secondary bacterial infections occur in this disease under South African conditions.

In his experiments on swine fever in East Africa Montgomery (1921) mentions that in rare cases he obtained cultures of *P. suisepticus* from the blood of pigs which had died of the form of swine fever he described. Walker (1933) does not mention having isolated either *P. suisepticus* or *S. suipestifer* (*S. cholerae suis*) from his cases of swine fever in East Africa. Steyn (1928) obtained staphylococci and *E. coli* from some of his cases but no other pathogenic type. Geiger (1937) did not find *S. cholera suis* or *P. suisepticus* in his cases of swine fever of the African type.

In the early cases of the disease at Gouda in the Western Province in 1933, swine fever was not suspected at first and specimens of spleen were sent to Onderstepoort in 50 per cent. glycerine saline from which a salmonella organism identified as *S. cholera suis* was isolated, and a vaccine was made from it for use in combating the outbreak.

In the first pigs inoculated with material from the Gouda outbreak, pure cultures of a *pasteurella* organism were isolated from the blood, and occasionally in further sub-inoculated pigs. No lesions of a pneumonic type associated with pasteurella organisms were ever encountered in any of the pigs used in the experiments. The experimental work done on laboratory animals and pigs is given in Appendix II.

In the course of a large number of inoculations into pigs at Onderstepoort about 80 cases have ended fatally and cultures have been made from the heart blood in every case. It is only exceptionally that paratyphoid bacteria have been encountered and in a small percentage of cases bipolar bacteria of the *P. suisepticus* type have been obtained in cultures, especially in primary sub-inoculations from natural cases. One must conclude that if paratyphoid organisms play any rôle in the production of lesions in swine fever in South Africa it must be a very limited one. In one case at Onderstepoort a *S. enteritidis* type has been obtained from a pig which died in a nutrition experiment, but no lesions of the type usually associated with paratyphoid in pigs were seen.

(3) Bacterial Diseases of Pigs Resembling Swine Fever.

Paratyphoid.

Outbreaks of a disease in pigs have been encountered in South Africa in recent years which resembled swine fever, especially macroscopically, but the cause was found to be *S. cholera suis*.

Martinaglia and Robinson (1932) described an outbreak of a disease in pigs in Johannesburg in which *S. cholera suis* was isolated and no association with the virus of swine fever could be demonstrated.

An outbreak of a disease in pigs was investigated in 1937 at a farm in Johannesburg where a big outbreak of swine fever had occurred in 1934. From some of the cases from which material was sent for investigation, paratyphoid was finally diagnosed, although at first swine fever was suspected. The results of the investigations are recorded in Appendix III.

Blood samples from some of the pigs were inoculated into susceptible pigs. One of these pigs developed a slight temperature reaction, so it was bled and its blood inoculated into two susceptible pigs. These did not develop any symptoms of illness.

In view of the fact that no definite diagnosis had been made, a second visit was paid to the farm two weeks later, by which time 109 pigs had died. The mortality was still occurring at random in the pens. The chief symptoms noticed were weakness of the hind limbs and listlessness.

At this second visit seven pigs were available for post-mortem examination. The result of this led to the conclusion that there appeared to be lesions of swine fever but of a slight and subdued type or masked by paratyphoid lesions. It appeared as if swine fever of a less virulent type than usual was being dealt with. The low mortality rate and the presence in some cases of " boutons " in the intestine brought the disease more in line with the European type of swine fever. The result of the histo-pathological examination of the organs from these cases, the results of the cultural examinations and the inoculations of blood of five pigs into susceptible pigs are given in Appendix VIII.

It will be seen that *S. cholerae suis* was obtained from five of the cases and that the blood inoculations were uniformly negative. A series of inoculations into susceptible pigs was carried out with cultures of the *S. cholera suis* strain obtained from the outbreak. Apart from intravenous inoculation which produced acute septicaemia, the other experiments gave negative results. Susceptible pigs injected intravenously with 24 hours broth cultures of the strain *S. cholerae suis* showed a marked febrile reaction commencing on the day of inoculation and continuing until the pigs died on the fourth day. They showed no other symptoms except *reddening of the skin and cyanosis.*

However in the histological examination in no single case were the lesions typical of swine fever seen, although the naked eye lesions, e.g., reddening of the skin, reddening of the lymphatic glands, etc., in some cases suggested it.

A further outbreak occurred on the same estate early in December, 1939. During the investigations carried out (see Appendix III) the post-mortems revealed haemorrhages in the skin, severe gastritis with diphtheritic membranes, enlargement of the spleen, haemorrhagic periportal lymphatic glands, etc. In some,

slight haemorrhage was noted in the kidney. The officer carrying out the first investigations expressed the opinion that the disease was strongly suspicious of swine fever. Further post-mortems subsequently conducted at Onderstepoort again resulted in a tentative diagnosis of swine fever. Blood was inoculated into susceptible pigs, cultures made, and suitable material collected for a careful histological examination. On the 28th December Dr. Thomas of the Pathological Section, Onderstepoort, however reported that although the *macroscopical findings* (e.g., ecchymoses lungs and kidneys, cyanosis and haemorrhosis skin, etc.) in the first lot of pigs were suggestive of swine fever, the typical lesions of this disease could not be confirmed histologically and *a definite diagnosis of paratyphoid was made.*

Typical lesions of paratyphoid were encountered, characterised by scattered pin point necrotic foci in the liver. Isolated necrotic centres were also encountered in the spleen and lymphatic glands of some of the cases. The histological diagnosis was supported by the bacteriological examination of the blood which revealed a pure culture of *S. cholera suis.* Furthermore no reactions occurred in the susceptible pigs inoculated with the blood.

The contributory cause of this outbreak may have been the heavy rains, which greatly affected the hygienic states of the styes, there being a shortage of shavings and sawdust. There was undoubtedly also considerable overcrowding of the pigs. It would appear that the mortality occurred in those pigs which had been introduced during the previous three weeks

Hutyra and Marek (1926) discuss paratyphoid in pigs and maintain that the intestinal lesions are frequently indistinguishable from those of swine fever. In an editorial on swine fever and paratyphoid in pigs in the *Journal of Comparative Pathology* (1933) the question of the relationship of the two conditions is discussed at length. Waldmann (1932) as a result of his studies on swine fever comes to the conclusion, however, that diagnosis by animal inoculation does not yield positive results in more than 50 per cent. of cases. Two further articles on paratyphoid which raise many points of interest are those by Shanks and Lamont (1938) and a circular notice (1938) by the Research Department of the Bacon Development Board of Great Britain. In the former the disease investigated by them was commonest in pigs eight to fourteen weeks and took the form of necrotic enteritis with occasional symptoms of pneumonia. All attempts to show any association with the virus of swine fever failed. In the latter publication reference is made to the importance of *predisposing causes* in cases of necrotic enteritis caused by *S. choleræ suis.*

Conclusions.

Although the lesions in these Savoy Estate outbreaks somewhat resembled swine fever, especially macroscopically, it would, however, appear that the disease was *paratyphoid* for the following reasons:—

(*a*) The blood of infected pigs injected into susceptible pigs failed to produce any reaction;

(*b*) paratyphoid organisms were isolated from most of the cases;

(*c*) the mortality ceased of its own accord after a small percentage of the pigs had died;

(*d*) the microscopical lesions encountered were those of paratyphoid, and not of swine fever;

(*e*) apparently the following predisposing causes played an important part in these outbreaks, viz., newly introduced pigs, excessive rains which markedly affected the hygienic state of styes, overcrowding, etc.

(4) The Relationship between the European and African Swine Fever Viruses.

Walker (1933) mentions that Montgomery sent blood from cases of East African Swine Fever to England but Stockman was unable to record a single survival when pigs immune to European swine fever were inoculated with it. Even hyperimmnue pigs succumbed. Virus and antiserum were obtained by Montgomery from England but when he inoculated pigs immunized with the virus and antiserum with blood from cases of East African swine fever, they invariably succumbed. No success attended attempts to protect against the disease with antiserum obtained from Budapest (Hungary) or from America. Pigs inoculated with the appropriate dose of antiserum and exposed to the East African disease by contact developed the typical acute type of the infection. Owing to the limited success which has attended the immunization of pigs against East African swine fever, it has not been possible to reverse the experiment and test the immunity of pigs immune to the East African disease against the European or American types. It has been shown by Hupbauer (1934) that a plurality of viruses of European swine fever does not appear to exist. If it did the immunization of pigs against swine fever would naturally become much more complicated.

In order to see whether there was any antigenic relationship between the South African and European types of swine fever the following experiments were carried out. The virus used was blood of a pig which died of acute swine fever as a result of inoculation with blood of a pig in the Johannesburg outbreak referred to elsewhere . The virus will be referred to as 1006. The serum used was imported from the United States of America.

In the first series of experiments the virus was inoculated under the skin of one thigh and the serum under the skin of the other one. Later the method was changed, the virus and serum being incubated together for 3 hours at 37° C., left in the refrigerator overnight and then incubated again for one hour before inoculation. This latter method was suggested by Alexander (1935) as a result of his experience with horsesickness serum virus inoculation in white mice.

The results of these experiments are given in Appendix IV.

Conclusions.

The experiments detailed in Appendix IV are still incomplete. Owing to the extreme infectivity of the disease it has not been possible to carry out many experiments at one time.

It would, however, appear that where a small dose of virus is incubated with antiserum, there is complete neutralization without any immunity developing subsequently. In the earlier experiments done without incubation of the serum-virus mixture, there were indications that the serum delayed the onset of symptoms. There is a definite indication that a relationship exists between the viruses of European and South African swine fever as judged by these experiments and that the two viruses have an antigenic factor in common.

(5) The Part played by Carriers.

Walker (1933) was unable to demonstrate that the blood of recovered pigs harboured the virus. Steyn (1931) showed that a recovered pig harboured the virus in its blood two months after recovery. Unfortunately he did not continue the experiment beyond this point. Waldmann (1932) in referring to the persistence of the virus in animals that have recovered from an acute infection, with or without being subsequently unhealthy in appearance, stated that it is not denied that such animals may be carriers of the disease, but little is known as to the proportion of carriers and the form and the duration of the excretion of the virus.

A few experiments were undertaken recently at Onderstepoort with the blood of pigs which had survived attacks of swine fever on several farms in the Piquetberg district of the Western Province. Blood was obtained from eight pigs which were slaughtered during the eradication campaign in the Western Province.

The results of these experiments are given in Appendix V.

Conclusions.

The blood of six out of ten pigs which had shown symptoms of illness during outbreaks of swine fever, proved infective for susceptible pigs. In some of the cases recovered pigs harboured the virus for ten months in the blood. Experiments were not carried out beyond this point.

Wild Pigs as Carriers of Swine Fever.

Montgomery (1921) showed that the blood of warthogs and bush pigs in Kenya might contain the virus of East African swine fever, as demonstrated by inoculation of susceptible pigs. Walker (1933) found that warthogs experimentally infected with the virus did not develop symptoms but harboured the virus in their blood subsequently. Steyn (1931) found that blood of three apparently normal warthogs from the Northern Transvaal harboured the virus of swine fever. Both Montgomery and Walker failed to demonstrate that contact of susceptible pigs with warthogs whose blood harboured

the virus of East African Swine Fever resulted in infection of the pigs. In the case of the warthog, the virus is not excreted in the faeces and urine. For this reason Walker has put forward the idea that the infection is transmitted from warthogs to pigs by an insect vector.

Geiger (1937) mentions that with the European type of virus he was able to infect a young wild boar experimentally and it died on the 12th day after inoculation. A full-grown wild boar showed no symptoms even after inoculation with big doses of a very virulent virus, but it excreted the virus. The American wild pig, the peccary, develops no symptoms but harbours the virus in the blood if inoculated with the European type.

With regard to the part played by wild pigs as carriers of the infection in South Africa, ten samples of blood from warthogs have been examined, with results as set out in Table III.

TABLE III.

Blood Samples of Warthogs injected into Domestic Pigs.

No. of Pig.	Origin of Blood.	Result.
724	Berg en Dal, Potgietersrust.....	Reaction with recovery.
725	Berg en Dal, Potgietersrust.....	Blood subsequently found infective.
726	Gruispan, Potgietersrust.....	Reaction with death.
727	Welgemoed, Potgietersrust.....	Reaction with death.
738	Oranjefontein, Koedoesrand, Potgietersrust.....	No reaction.
737	Umfolosi Game Reserve, Natal.....	No reaction.
737	Umfolosi Game Reserve, Natal.....	No reaction.
1047	Koedoesrand, Potgietersrust.....	Reaction with death.
1058	Koedoesrand, Potgietersrust.....	Reaction with death.
1105	Crown Lands, Louis Trichardt.....	No reaction.
1106	Gravelotte, Pietersburg.....	No reaction.

From the above it will be seen that some of the blood samples from apparently healthy warthogs collected in the Potgietersrust district produced reactions to swine fever with death when injected into susceptible (domestic) pigs. The pathological-anatomical and histological diagnosis of some of these cases are briefly enumerated in Table IV.

TABLE IV.

No. of Pig Injected.	Date of Death P.M. No. Spec. No.	Pathological Anatomical Diagnosis.	Histological Diagnosis.
726	24/6/31 P.M. 10391 Spec. No. 11700	Hydropericard : Extensive haemorrhage of lymphatic glands especially periportal. Numerous subcapsular and intracortical haemorrhages kidney. Coagulated blood caecum and colon with numerous haemorrhages in mucosa. Haemorrhage perirectal tissue	Extreme extravasation of blood in sinuses of lymph gland—pigmentation. There is practically no hyalinisation of vessels or karyorrhexis. *Spleen :* Marked haemorrhages in pulpa ; Schweiger-Seidel Sheath disappeared. *Follicles* only slightly atrophied. Relatively slight karyorrhexis. *Kidney :* punctiform haemorrhage cortex ; hyalinisation glomeruli, droplet degeneration. *Liver :* hyperaemia and slight diffuse karyorrhexis.
727	30/6/31 P.M. 10415 No specimens collected	Hydropericard : subepicardial haemorrhages. Marked oedema and hyperaemia lungs. Degeneration myocard. Tumor splenis. Extensive haemorrhage of lymphatic glands especially periportal. Subcapsular and intracortical haemorrhages kidney. Subpleural haemorrhages. Haemorrhages in stomach and small intestines. Croupous typhlitis and colitis.	
1047	6/11/34 P.M. 13720 Spec. No. 15627	Hyperaemia lungs and liver. Mottling of periportal lymphatic glands. Slight tumor splenis. Haemorrhages cortex kidney. Extravasation left endocard. Slight hyperaemia stomach. Extensive haemorrhage peritoneal cavity with blood in pelvic region. Haemorrhage in bladder.	*Spleen :* No trace of Schweiger-Seidel sheath ; follicles are indistinct and some show karyorrhexis (early). There is no marked haemorrhagic effusion. *Lymphatic gland :* Severe haemorrhage ; Karyorrhexis slight ; hyalinisation very slight ; follicles indistinct. *Kidney :* Slight hyalinisation and karyorrhexis of vessels. *Brain :* Nothing unusual. *Heart :* Myocard—nothing unusual. *Liver :* Fairly extensive intralobular patches like blood lagoons from which liver cells have disintegrated ; necrosis-karyorrhexis.
1058	31/1/35 P.M. 13927 Spec. No. 16000	Localised degenerative changes liver. Haemorrhagic infiltration gall bladder. Hyperaemia periportal lymphatic glands. Hyperaemia small intestine. Slight hyperaemia stomach and large intestines. Hydroperitoneum and hydrothorax	*Brain :* Nothing unusual. *Kidney :* Small haemorrhages. *Gland :* Extensive karyorrhexis and hyalinisation ; follicles distinct. *Spleen :* Schweiger - Seidel Sheath disappeared. Karyorrhexis extensive.

From Table IV it would appear that the lesions seen, although perhaps less significant, are nevertheless characteristic in all respects of swine fever. One gets the impression that these cases ran a peracute course and that consequently the lesions did not develop to the usual extent, particularly in respect of hyalinisation, thrombosis and karyorrhexis of the endothelial lining of smaller blood vessels.

(6) Study of the Disease in Small Animals.

As a result of the work of Theiler (1931) with the virus of yellow fever many attempts have been made to establish the filterable viruses associated with many diseases of man and domesticated animals in laboratory animals in order to facilitate research on these viruses. Great progress has been made in research on yellow fever as a result of the discovery that it could be transmitted to white mice by intracerebral inoculation. The discovery that foot and mouth disease could be transmitted to guinea pigs very greatly facilitated research on this disease. More recently Alexander (1933) and Nieschulz (1933) have shown that the virus of horsesickness can be transmitted to white mice and guinea pigs by intracerebral inoculation, the virus becoming neurotropic in mice.

No record could be found in the literature of any attempt to transmit the swine fever of Europe and America to laboratory animals by intracerebral inoculation. It has been attempted in recent years as a result of the success which has been achieved with some of the filterable viruses, but the results have been negative and subsequently not recorded.

Dr. Alexander of this Institute attempted to transmit swine fever to white mice by intracerebral inoculation using the technique which had been so successful in the case of horsesickness. Fresh defibrinated blood from a pig at the height of the swine fever reaction was used but no success attended the experiment. Subsequently the writer has made repeated attempts to transmit the disease to white mice and guinea pigs but without success. Owing to the fact that in the primary inoculations with yellow fever and horsesickness, only some of the inoculated mice developed the disease and sometimes with a long incubation period of up to 18 days, the animals in the experiments were kept for at least 20 days before being discharged. In all the inoculations into white mice, half grown animals were used as in them it is possible to push the needle directly through the skull into the brain instead of inoculating through the occipital foramen. In the case of the guinea pig, again half grown animals were used. For inoculation of guinea pigs intracerebrally a method employed by Mason at Onderstepoort in attempts to transmit blue tongue and heartwater to these animals was used.

The results of these experiments are given in Appendix VI.

Conclusions.

These experiments are in the nature of a preliminary investigation and further attempts will be made to see whether the virus of

swine fever can be acclimatized to laboratory animals. The indications at present are that the virus cannot easily be so adapted, the attempts to transmit the infection to laboratory animals having failed up to the present.

(7) Immunization against Swine Fever.

Walker (1933) and Geiger (1937) record attempts to immunize pigs against African swine fever. Neither had much success with attenuated virus or organ extracts treated with various chemicals. According to Geiger (1937) on account of the trouble in getting recovered pigs, the preparation of an immune serum presents great difficulty. The results of serum virus inoculations are only of limited value. An immunity of a durable nature type cannot be obtained. Walker (1933) as a result of an experiment which he carried out maintains that in contrast to swine fever, African virus shows a plurality of strains.

On account of the fact that the policy of the government in South Africa in the eradication of swine fever was the slaughter one, and the fact that the facilities were unsatisfactory, attempts at the development of a method of immunization were not actively pursued.

In view of the recent success attained by McBryde and Cole (1936) with the immunization of pigs against swine fever with virus attenuated by the addition of crystal violet, some experiments were carried out to study the effect of crystal violet on swine fever virus. The technique employed followed the lines of that recommended by Dorset and described by McBryde and Cole. 90 Parts of virulent defibrinated blood from pigs in the acute stage of the disease were mixed with 10 parts of a 1 per cent. aqueous phenol solution and then with 5 parts of an aqueous 1 per cent. solution of pure crystal violet. The mixture was incubated at 37·5° C. as recommended by Dorset but the time taken to render the virus attenuated had to be established. For this purpose the experiments as detailed in Appendix III were carried out (see Appendix VII).

Conclusions.

No success attended attempts at attenuating the virus of swine fever by the use of the crystal violet method of Dorset.

HISTORICAL.

The earliest report of the existence of swine fever in South Africa is by Hutcheon (1903) who recorded its occurrence at the Government farm, Groot Constantia, Cape Peninsula, and immediately afterwards in the Paarl district. On investigation it was found that this disease had been very widespread and a heavy mortality had occurred. It had not been reported to the veterinary department either, having been concealed or mistaken for a parasitic broncho-pneumonia which had previously caused heavy losses. Hutcheon was of opinion that the disease had probably been in existence for more than two years in the affected areas, particularly on the Cape Flats. He introduced stringent control measures

which included slaughter and burial of infected pigs, disposal of healthy pigs to the butcher and no introduction of fresh pigs to the farm for six months. Destruction of manure and thorough disinfection of the premises were undertaken. Only two outbreaks are recorded in 1905, both in the Malmesbury district of the Western Province.

Robertson (1905) in commenting on these early outbreaks refers to the absence of restrictions on the importation of pigs, either from other South African colonies or from England. He considered the disease may have been introduced prior to 1900, but that it was confused with broncho-pneumonia, or that *occult cases or carriers* had been introduced and had been responsible for the outbreaks. He pointed out that an extended quarantine period would be necessary in order to eliminate these latter cases. In describing the symptoms he refers to staggering gait, weakness of the hindquarters, large blotches on the skin of the neck, at the back of the ears, on the thighs, etc. A cough might be observed. The course was two to three days or longer in some cases. He laid emphasis on the changes in the digestive organs whereas pneumonia occurred in a number of cases. He described ulcers, single or confluent, varying in shape, from a yellowish grey to a black colour and usually circular in form. In the absence of these lesions Robertson considered a diagnosis of swine fever doubtful without further evidence. He noted the haemorrhagic appearance of some of the lymphatic glands, particularly the inguinal and those at the base of the lung.

As early as 1903 Stockman refers to outbreaks of swine fever in the Transvaal and considered that it had not as yet gained a footing. Provisions were made in Government Notice No. 834 of 1903 to deal with the disease. An outbreak of what was undoubtedly swine fever was diagnosed in the Krugersdorp district of the Transvaal in 1903. The pigs had been bought on the Johannesburg market but their origin could not be traced further. The outbreak was stamped out successfully. Stockman mentioned the part played by chronic cases or those not showing symptoms in the spread of the disease.

Gray (1904) records further outbreaks in the Pretoria and Krugersdorp districts of the Transvaal. These were stamped out by the usual methods, but the fear was expressed that the disease had gained a footing.

Theiler (1905) was inclined to connect the outbreaks in the Transvaal with those in the Cape and therefore prohibited the introduction of swine from that Colony. The pathological anatomical diagnosis of swine fever appears to have been beyond doubt (the photographs of Theiler show typical " boutons ").

In the blood inoculation experiments carried out by Theiler in 1905 interesting lesions were manifested. Changes in the mucous membrane of the gastro-intestinal tract varied from deep congestion with areas of commencing necrosis to advanced areas of necrosis in the large intestine. In a number of cases no changes are recorded in the spleen, kidneys and liver, and in some diffuse reddening and

haemorrhages under the skin are mentioned. In a number consolidated areas in the lung are referred to and in a few, small haemorhages in the kidneys are recorded. In some the lymphatic glands are said to be congested and swollen.

Theiler came to the conclusion that the disease investigated by him in the Transvaal resembled hog cholera of America and the swine fever of Europe. He was however never able to trace *B. suipestifer* and concluded that the presence of this bacillus was not necessary for the development of swine fever.

With reference to the socalled swine plague which he also investigated at that time, Theiler maintained that in South Africa under natural conditions the lesions of swine plague are found as a rule associated with true swine fever. There was only one spasmodic case of swine plague in which *B. suisepticus* was found and where swine fever had to be excluded.

Gray (1906) refers to further outbreaks in the Transvaal, some stated to be complicated with swine plague, and he was of the opinion that the number of outbreaks reported did not truly reflect the actual position as many outbreaks were probably not reported.

In the reports of the Principal Veterinary Officer of the Union of South Africa from 1910-1918 references are made to swine fever outbreaks. Three outbreaks were reported in 1910 and in 1912 twenty outbreaks in the Western Province were effectively dealt with. In 1917 ten outbreaks with a heavy mortality were reported from five districts of the Western Province. Further outbreaks occurred in these areas in 1918 and cases were reported in the Eastern Province at Somerset East and Longhope. These outbreaks were effectively dealt with. It must be emphasized that these outbreaks were diagnosed on clinical and post-mortem findings, and it was assumed that they were the European type of swine fever.

No further outbreaks were recorded until 1926, when an investigation was made into a very virulent disease of pigs occurring in the Potgietersrust district of the Northern Transvaal. The farmers in the locality were unable to keep pigs on account of this disease and the mortality in some instances was 100 per cent. Pigs running free on the farms were most affected and warthogs were reported to occur in large numbers on the infected farms. Steyn (1928) investigated this disease and came to the conclusion that it was the same as that described by Montgomery (1921) in East Africa and called "East African swine fever". Steyn (1932) showed that the disease was caused by a virus which could pass a Seitz filter. After subinoculation of the virus the incubation period was thirty-six hours to four days. After the initial temperature the period up to the time of death was usually two to three days, but in one case it lasted as long as eleven days. The disease was therefore very acute and the temperature sometimes reached 108° F. The most important post-mortem changes described by Steyn were a croupous pneumonia, haemorrhage foci in the lungs, haemorrhagic infiltration of all the lymphatic glands and the kidneys and a croupous typhlitis. In 1931, 1932 and 1933 several fairly large outbreaks occurred in both the Zoutpansberg and Pietersburg districts of the Northern Transvaal

with heavy mortality. The infection could not be traced to direct contact with warthogs but it was strongly suspected that that was the source of infection. Blood from some warthogs harboured the virus whereas recovered pigs might harbour the virus for two months in their blood. Steyn suggested that farmers in warthog areas should not keep pigs on account of the danger to the pig industry in general.

DISCUSSION.

According to the above historical review it would appear that swine fever of the European type and resembling hog cholera of America appeared in South Africa about 1900. According to Robertson the disease was probably introduced into South Africa by means of infected animal products, occult cases, or carriers. The symptoms described by Robertson in many respects resemble the acute cases of swine fever seen in the Western Province and Transvaal from 1933 onwards, namely staggering gait, weakness of the hindquarters, and large blotches on the skin of the neck. Robertson also refers to the haemorrhagic appearance of some of the lymphatic glands. Theiler connected the 1905 outbreaks in the Transvaal with those in the Cape and prohibited the introduction of pigs from the Cape Colony. In experiments carried out, Theiler came to the conclusion that the *disease in pigs produced by the inoculation of blood* resembled hog cholera in America and European swine fever. Amongst the post-mortem changes described, the presence of haemorrhages under the skin and in a restricted number of cases small haemorrhages in the kidneys are mentioned besides consolidated areas in the lung and swelling and congestion of the lymphatic glands. Theiler also referred to the presence of " boutons " in the large intestine and in his photographs these lesions are very well represented. The disease in each of the larger outbreaks from 1900 onwards died out or was eradicated by the methods and precautions undertaken.

From the records it would appear that the disease did not make its re-appearance in South Africa in an epizootic form for several years. After 1926 intermittent outbreaks of a very virulent and infectious disease occurred in the Northern Transvaal, i.e. some considerable distance inland. Some of these outbreaks were investigated by Steyn and others and they were of the opinion that the disease was identical with the African virus disease of pigs described by Montgomery in East Africa. On post mortem there were invariably present subendocardial haemorrhages in the left ventricle, marked tumor splenis, haemorrhages in the kidneys, haemorrhagic changes in most organs especially in the periportal lymphatic glands. From his description of the disease and from the description of the cases in the recent Witwatersrand-Western Province outbreaks it would appear that the disease investigated by Steyn and by the authors was one and the same.

The macroscopical and microscopical changes described in this paper resemble the acute cases described by Seifried, Geiger and others, in case of the European virus, namely, haemorrhages in various organs, especially in the lymphatic glands, urinary apparatus, stomach, intestinal tract and in the skin. Reference is made to the

haemorrhagic and marble-like appearance of the lymphatic glands. Geiger states that in the first instance damage to the vascular walls takes place in the form of a degeneration of the vascular endothelial cells and necrosis, a hyalinisation of the capillaries and precapillaries.

The swine fever lesions observed in South Africa are in general perhaps more acute, especially in respect of capillary injury. The absence of cerebral peri-vascular cellular infiltrations in the South African specimens should be noted but it is interesting to state that Seigfried has already expressed doubt as to the specificity or significance of these cerebral lesions in true swine fever.

Geiger reports that the hyper-immune serum prepared from the European virus had no effect on the virus from South Africa and from various data collected by him these two diseases should be regarded as aetiologically different. Although the earlier experiments on cross immunity between the European and African viruses gave no indication of a relationship between the two, the recent experiments at Onderstepoort would seem to give an indication that the two diseases have an antigenic relationship.

It is therefore quite likely that probably more than one " strain " of swine fever virus exists and that the South African one is particularly virulent. Analogies of this are furnished in case of the other viruses, e.g. horsesickness, blue tongue, foot and mouth disease, etc. In case of the Tzaneen horsesickness strains obtained by passage through a number of generations it was found that the one group remained extremely virulent with a mortality of about 100 per cent. in susceptible horses, whereas in the other group attenuation took place and the mortality was reduced to about 15 per cent. In their respective passages the highly virulent Tzaneen strain continued to produce symptoms and pathological changes associated with the respiratory system, whereas the more attenuated strain invariably produced the cardiac form of horsesickness. Although there was a definite antigenic relationship between them (both derived from the same virus), the more attenuated strain only partially protected against the virulent type in a small percentage of animals. The study of the possible existence of " several strains " of swine fever virus is to be continued. Experiments have also been planned to study the effect of the passage of the European strain of virus in warthogs.

Swine fever in the Onderstepoort experiments has been of such a virulent nature that there has been little opportunity of studying the problem of immunity, especially in recovered animals. Although some limited success has attended attempts to immunise pigs, the immunity produced does not appear to be very strong. Unfortunately experiments of this nature have been hampered by the fact that up to the present it has not been possible to transmit the virus of swine fever to laboratory animals more suitable for immunity observations on a large scale.

In respect of the spread of the disease during the recent outbreaks, especially in the Western Province, it will be seen that it was comparable to that of the European disease as described by Geiger.

From the above remarks it would therefore appear that the disease described by Hutcheon and Robertson in 1903 and later by Theiler in 1905 was the same as the disease investigated by Steyn in 1928 and by the authors from 1933 onwards. Furthermore the disease investigated on several occasions in South Africa resembles *the acute form of European swine fever.*

The disease in South Africa is evidently of a particularly virulent character and usually manifests itself in the form of *an epizootic.* Judged by the absence of reports of mortality in pigs it would seem that the Union has for periods of several years been free of this disease, whereas in most European countries it has remained *enzootic.* Has this been due to the elimination of the virus carrier by the slaughter out policy adopted as soon as outbreaks were reported? Did environmental factors and the fact that few pigs in the larger part of the Union are scattered over wide areas contribute to this? After these long spells of freedom from the disease where did the recent outbreaks originate? Apparently the recent Western Province outbreaks stand in relation to the occurrence of the disease in the Witwatersrand area. How was the virus introduced into this latter area, seeing that it is situated fairly far inland and was probably not due to the introduction of infected animal products or ship's garbage, responsible for the 1927-1928 outbreaks in Australia according to Seddon and Blumen (1931)? Indications are that the disease during the recent Witwatersrand outbreak came from the Northern Transvaal in pigs moved to the Johannesburg market. It is doubtful whether this disease could have smouldered in domesticated pigs in Northern Transvaal without manifesting itself sooner or later. The disease is of such a virulent nature with high mortality that it could not have been suppressed for long intervals.

Is the virus in South Africa maintained in a limited number of wild pigs in certain areas of the Northern Transvaal? It is not yet known to what extent wild pigs are infected in this area and how infection is transmitted to domesticated pigs (whether by contact or insect transmission). Such transmission from the wild pig to the domesticated pig probably does not occur frequently and that may explain the peculiar intermittent incidence of this disease in South Africa. In the investigations carried out it was shown that the virus collected from clinically healthy warthogs could produce a form of disease indistinguishable from the one derived from domesticated pigs. A point to be investigated in the near future is to ascertain whether the European " strain " of virus in passage through the warthog becomes exalted. Has the warthog adapted itself to the virus and did the original swine fever virus of domestic pigs come from wild pigs? (*c.f.* trypanosomes in game).

Steyn refers to the fact that recovered domesticated pigs harbour the virus for a period of two months. Unfortunately he did not carry the investigations beyond this point. From the investigations with pigs which survived during the recent outbreaks in the Western Province it was found that the blood of some remained infected for susceptible pigs for a period of ten months. In some of the outbreaks a few pigs recovered from the acute disease but remained in poor

condition and showed a scaly condition of the skin. In the Western Province swelling of the joints of the legs was noticed in some of the recovered cases. The virus of swine fever belongs to the class of very resistant viruses. From experiments carried out it was shown that it survived for a period of six years if kept in a refrigerator in the dark, but decomposition will render it non-infective in about 70 days.

A bacteriological examination was carried out in all cases investigated during the recent outbreaks, and complications with secondary bacterial infections such as *P. suisepticus* and *S. cholerae suis* occurred only in a limited number of cases. In two recent outbreaks of a disease in pigs with fairly high mortality the aetiological factor was suspected to be *S. cholerae suis*. In most of these cases the symptoms were acute and macroscopically some of the lesions resembled acute swine fever. The disease in both outbreaks died out without causing a high mortality and the blood of infected pigs did not produce swine fever. Waldmann, however, states that in herds infected with the acute or chronic form of the disease, diagnosis by animal inoculation did not yield positive results in more than 50 per cent. of the cases. It would appear that in South Africa as in Europe and elsewhere outbreaks of paratyphoid do occur and may sometimes assume a septicaemic form.

It will be interesting to ascertain how far *B. suipestifer* is mainly or entirely responsible for the structural alterations in the bowel, especially the diffuse diphtheritic inflammation and the formation of " boutons " in the caecum and colon. In the recent outbreaks of Swine Fever in South Africa such changes were significant for their absence and from the large number of investigations carried out in which blood cultures were undertaken it would appear that these organisms were rarely present. Probably the disease in South Africa is of such a virulent nature that secondary organisms are not afforded a chance of developing the lesions of diphtheresis, " boutons ", etc.

CONCLUSIONS.

1. It is believed that various outbreaks of swine fever in South Africa have probably occurred since 1900. The disease in domestic pigs produced by the blood of warthogs resembled the European disease. Probably in swine fever, as in many virus diseases, more than one " strain " of virus exists.

2. Reference was made to the virulent nature of this disease in South Africa, and the fact that it does not progress as an enzootic as in European countries. A number of recovered animals harboured the virus for long periods.

3. It is quite likely that the virus in South Africa may also be maintained in a limited number of warthogs in certain areas of Northern Transvaal where domestic pigs occasionally become infected. That may explain the intermittent incidence of this disease in South Africa.

4. From the investigations carried out it would appear that the following may occur in South Africa :—

(*a*) Uncomplicated very acute form of swine fever without manifestations of " boutons ", diphtheresis, hepatization of the lungs, etc.

(*b*) Swine fever possibly complicated by such secondary infections as *S. cholerae suis* and *B. suisepticus*.

(*c*) A septicaemic type of *S. cholerae suis* infection in an enzootic form and probably associated with such predisposing causes as bad hygiene, overcrowding, defective feeding, etc.

5. A description of the main pathological changes is given, and the value of the histological examination of organs for diagnostic purposes is discussed.

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APPENDIX I.

Filtration experiments carried out to ascertain whether the outbreaks were due to the virus of swine fever or to a bacterial infection, and to study the filtration properties of the virus.

Experiment 1.—

Pig 952 was inoculated with 5 c.c. of filtrate of blood from a pig which had just died from swine fever. The blood was laked by the addition of distilled water and salt was added to make the concentration .85% A Seitz filter with one asbestos disc was used. The inoculated pig did not develop any symptoms and was found to be susceptible when subsequently inoculated with virulent unfiltered virus.

Experiment 2.—

Pig 955 was inoculated with 5 c.c. of filtrate of blood from a case, and made in the same way as in the previous experiment. No reaction occurred.

Experiment 3.—

Pig 957 was inoculated with 10 c.c. subcutaneously of a filtrate from virulent swine fever blood. Again no reaction occurred.

As the filtration experiments with the Seitz filter had not shown the virus to be filterable it was decided to try Berkefeld filters. Infective blood was prepared in the same way as for the Seitz filtration experiments and passed through a Berkefeld N filter under low pressure. Larger

quantities of this filtrate were used than of the Seitz as it was thought that perhaps the quantities used had been too small. In Steyn's experiments (1928) 2.5 c.c. of a Seitz filtrate was fatal. The Berkefeld filtrate was sterile on the two occasions when it was made.

Experiment 4.—

Pig 934 received 50 c.c. of a Berkefeld filtrate subcutaneously. It showed a typical reaction and died of swine fever on the 6th day. Another pig 935 received 40 c.c. of another Berkefeld filtrate subcutaneously and died of acute swine fever on the 6th day. In both cases a few staphylococcus colonies were obtained from the heart blood.

With blood from cases of swine fever which died in the Johannesburg outbreak in 1934, further filtration experiments were carried out. The blood was prepared in the same way as described in the previous experiments.

Experiment 5.—

Two pigs 979 and 991 inoculated with a Berkefeld filtrate. Pig 979 was inoculated subcutaneously with 5 c.c. and pig 991 with 5 c.c. into the lung tissue. No reaction occurred for 14 days.

Pig 979 then recived 5 c.c. of the filtrate intravenously and died of swine fever on 6th day. Pig 991 received 20 c.c. subcutaneously and died on 5th day after a typical reaction. It is possible the reactions were due to the first inoculations but this seems unlikely and the route of infection in one case and the higher dose in the other were probably the real factors involved.

Conclusions.

It was found that the virus of swine fever would not pass a Seitz filter. Filtration experiments carried out with a Berkefeld filter were however successful.

APPENDIX II.

Experiments on laboratory animals and pigs with bacteria isolated from swine fever cases.

The blood of the pigs which died in the first transmission experiment was inoculated into two rabbits. Each rabbit received 1 c.c. intravenously and died in less than 24 hours. Smears from the blood and cultures from it showed organisms of the *P. suisepticus* type in both cases.

Two white mice inoculated subcutaneously each with 1 c.c. of a mixture of the pigs' blood died in about 48 hours. Smears from the blood and cultures showed organisms of the *P. suisepticus* type in both cases.

Two rabbits were inoculated subcutaneously each with 1 c.c. of the pigs' blood. One died on the sixth day after inoculation showing a purulent pleuritis and peritonitis. Lesions of a general septicaemia were present and an organism of the *P. suisepticus* type was isolated from the blood. The second rabbit died on the eleventh day after inoculation but no lesions of pasteurellosis could be demonstrated and cultures from the blood were negative.

Owing to the fact that in some experiments with Seitz filtrates of blood failure was experienced in setting up any disease, the possibility had to be considered that an acute bacterial disease was being dealt with. In the later experiments with the disease, the bacterial complications seemed to disappear, though occasionally coccus types were isolated from the blood of experimental cases.

As both the *salmonella* and *pasteurella* types isolated from these cases were available for experiments it was decided to see what symptoms they would produce in pigs when inoculated with them.

Experiment 1:

Pigs 955 and 957 each received 3 c.c. intravenously of a 24 hour broth culture of *P. suisepticus* isolated from the cases previously mentioned. The pigs were inoculated on 23rd November, 1933, and the same day a high febrile reaction was shown in both cases, which persisted until death occurred. One pig died on 26th November, 1933, and the other on 27th November, 1933. The pigs remained lying down from the day of inoculation and could not be made to rise. There was some reddening of the skin. At post-mortem there were lesions of an acute septicaemia, with hyperaemia and oedema of the lungs, tumor splenis, hyperaemia of liver and kidneys and a diffuse gastro-enteritis. The lesions were very similar to those seen in swine fever, but there was a very short incubation period.

Experiment 2:

Pig 956 was inoculated on 14th November, 1933, with 2 c.c. of a 24 hour broth culture of *P. suisepticus* subcutaneously. No reaction occurred and the pig was inoculated on 23rd November, 1937, with 5 c.c. of virulent swine fever blood. It died of the disease on the 6th day after inoculation.

Experiment 3:

Pig 952 was inoculated intravenously on 22 November, 1933, with 3 c.c. of a 24 hour broth culture of the strain of *S. cholerae suis* from the Western Province. A marked febrile reaction commenced the same day and continued until the pig died on the 4th day. It remained lying down and would not rise but showed no other symptoms except a slight reddening of the skin and cyanosis. At post-mortem there was slight hyperaemia of the lungs, marked hyperaemia of the liver and spleen.

Cultures were made from the blood and spleen in five cases post mortemed during the big outbreak of swine fever which occurred in Johannesburg in February, 1934. One of these pigs showed a few organisms of the *P. suisepticus* type in the blood, but the others were negative with the exception of one which showed a few chromogenic cocci in the blood.

In November, 1933, a pig owner in Johannesburg reported that his pigs, 35 in number, had all died of an acute disease and one was brought to Onderstepoort for post-mortem. It showed lesions typical of swine fever and a susceptible pig inoculated died on the 6th day with typical lesions. Cultures from the blood showed *P. suisepticus* in pure culture.

In November, 1934, one of us (A.D.T.) paid a visit to the Western Province during the outbreak in the Malmesbury district. Several farms were visited where the disease had occurred prior to the visit. On one farm (de Kock, Vogelgesang) three sick pigs in poor condition were seen. One was killed but at post-mortem the only lesion was a broncho-pneumonia. Two pigs which had died that morning were exhumed and post-mortemed. Both showed a broncho-pneumonia, one this lesion only, but the other pin point haemorrhages on the kidneys, slight lymphadenitis and a gastric catarrh. Typical swine fever cases had occurred on this farm. Cultures from the blood and spleen were made but only colonies of staphylococci grew in them.

On 15th November, 1934, a pig 1060 was inoculated with 5 c.c. blood from the case which was killed. The pig died on 28th day after having shown two distinct temperature curves. Blood was taken from the pig during the first reaction and inoculated into pig 1057 on 15/12/34. This pig died of acute swine fever on the ninth day.

At the second farm (Jordaan, Radyn) the owner had lost all his pigs except a sow and five piglets. The sow had been sick for 14 days but had shown signs of recovery in the previous two days. The animal was killed and at post-mortem a subacute pericarditis and broncho-pneumonia were found. No lesions

of the alimentary tract were seen. Cultures were made from the organs in glycerine. A few colonies of staphylococci and *E. coli* were obtained. Blood from the pig was inoculated into a pig 1049 which died of acute swine fever on the sixth day.

At the third farm (Richter, Bontheuvel) a small pig, two months old, was seen showing swollen glands and joints. It was stated to be recovering and the owner had lost all but a few piglets from swine fever. The pig was killed and at post-mortem, showed pericarditis and swelling of the joints, which contained a thick turbid fluid. Cultures from the fluid in the joint cavity showed colonies of streptococci. On 15th November, 1924, a pig 1051 was inoculated with blood from this case. It died on 20th day after inoculation but at post-mortem the lesions were those of an acute enteritis with a heavy ascaris infection but no lesions of swine fever.

Serum of this pig was tested with strains of *S. suipestifer and S. enteritidis* (Dublin) but no reaction was obtained.

At the fourth farm (Kitshoff, Spes Bona) heavy losses had occurred. A small pig was seen showing swollen joints and apparently recovering. It was killed and at post-mortem showed swollen necrotic submaxillary glands, a slight pericarditis, and a large caseous necrotic abscess on one carpal joint. No sub-inoculations were done from this case. Cultures showed staphylococci and streptococci in the material from the joint. The serum of the pig gave negative reactions with salmonella *S. cholerae suis* and *S. enteritidis*.

At the last farm visited (Burger, Ideal Hill) there were only a few piglets left, some of which had swollen joints. Two of the pigs with swollen joints were killed and at post-mortem extensive serous arthritis was seen and swollen glands. No sub-inoculations were done from these pigs. Cultures from the material from the joints showed a few coliform colonies of the *E. coli* type. Streptococci were present as well.

In March, 1936, an outbreak of swine fever occurred in the Louis Trichardt district of the Northern Transvaal and sub-inoculations with the blood of cases produced the acute disease. In some of the sub-inoculated pigs, *P. suisepticus* infection was observed.

The presence of *P. suisepticus* was demonstrated in some cases of swine fever and other organisms such as staphylococci were found in others. The organisms did not appear to have any etiological significance.

APPENDIX III.

Recent Outbreaks of Paratyphoid in Pigs on the Savoy Estate, Johannesburg.

The 1937 Outbreak.

(*a*) *Cases in January*, 1937.

An investigation *re* mortality in pigs was carried out in January, 1937, at the Savoy Estates. Two dead pigs and three *in extremis* were available for post-mortem. During the previous month 26 pigs had died out of about 2,500 in the styes. The mortality was not confined to any particular pen but was distributed throughout the various pens.

Case 1.

Black sow, 6 months old. There was reddening of the skin of the abdomen and between the legs.

The liver showed hyperaemia but the periportal glands were unchanged. Slight enlargement and hyperaemia was noted in the *spleen*. The mucous membrane of the stomach showed a diffuse hyperaemia.

Diagnosis.—A definite diagnosis could not be made at the time, but *S. cholera suis* was isolated from the spleen.

Case 2.

Black hog about 6-8 months old. Reddening present on skin of abdomen.

Lungs.—Marked hyperaemia. Several small areas of hepatization.

Spleen.—Marked hyperaemia.

Liver.—Marked hyperaemia. Periportal lymphatic glands mottled, reddish grey, but not intensely hyperaemic.

Kidneys.—Marked hyperaemia. Numerous small pin point haemorrhages in cortex.

Stomach.—Deep red colour, with ulceration and diphtheritic deposit.

A tentative diagnosis of swine fever was made, pending the result of cultures and inoculation of blood into susceptible pigs.

Case 3.

Black and white sow, about 6 months. The only lesions found were hyperaemia of the lungs with small areas of hepatization.

No diagnosis was made pending results of cultures and histological report.

Case 4.

Black and white sow, about 6 months old, very thin, and with swelling of the left carpal joint.

Lungs.—Atelectasis of portions of both lungs.

Kidneys.—Large yellow areas in the cortex giving a mottled appearance.

The only other lesion was an extensive fibrinous deposit on the serosa of the intestines, liver and spleen.

Case 5.

Black hog 6 to 8 months, in good condition. There was reddening of the skin of the whole body.

Lungs.—Mark hyperaemia.

Liver.—Marked hyperaemia. Marked mottling of the periportal glands, of a dark reddish grey type.

Spleen.—Greatly enlarged. Marked hyperaemia.

Kidneys.—Marked hyperaemia. Numerous pin point haemorrhages in the cortex.

Stomach.—Diffuse bluish red colour of the mucous membrane, with ulceration and diphtheritic deposit. A tentative diagnosis of swine fever was made.

Blood from pigs 2 and 5 were mixed and inoculated into susceptible pigs at Onderstepoort but no symptoms developed as a result of the inoculation. One pig showed a slight temperature reaction, so it was bled and two further pigs inoculated. These did not develop any symptoms.

(*b*) *Cases in February*, 1937.

Case 1.

Black hog. It showed a slight hydropericardium and hydrothorax, pulmonary oedema and atelectatic areas in the lung. All the lymphatic glands of the body showed a marked redness round the periphery. The *stomach* showed a tenacious diphtheritic pseudo-membrane on the folds with haemorrhages in between. The *skin* showed an intense cyanosis. This case was tentatively considered to be one of swine fever.

Cases 2 *and* 3.

Were too decomposed for a proper examination. Pig 3 showed an extensive fibrinous pleuro-pneumonia and pericarditis.

Case 4.

Showed fibrinous pericarditis and pleuropneumonia.

Case 5.

Blotchy cyanosis of the skin.

Spleen.—Slight hyperaemia.

Liver.—Extensive and disseminated miliary necrosis. (Typical paratyphoid lesions.)

Case 6.

Black hog (killed).

Lung.—Adherent to thoracic wall. Extensive commencing consolidation. Adhesive fibrinous pericarditis.

Caecum.—Ileo-caecal value and part of colon showed a yellow flaky pseudo-membrane raised above the surface. Around this there was a red margin with slight concentric ringing. There was a diphtheroid enteritis with characteristic " bouton " formation.

Case 7.

Black sow. Poor condtion. Swollen off knee with necrosis of the joint.

Hydrothorax.—The only lesions seen were in the lungs which showed large atelectatic areas. Bronchial lymphatic glands were swollen and mottled.

Bacteriological work and sub-inoculations.

Pure cultures of *S. cholerae suis* were obtained from the blood of pigs 1, 4, 5, 6 and 7. Blood from pig 1 was inoculated into two susceptible pigs at Onderstepoort, and a mixture of bloods of 4, 5, 6 and 7 into two others. No reactions were observed in these pigs subsequently. A tentative diagnosis of swine fever had been made as a result of post mortem examination of the seven pigs but it could not be confirmed biologically.

In order to see what the effect would be of inoculating or dosing pigs with the paratyphoid cultures obtained from the cases the following experiment was carried out. A pig (1195) was given 5 c.c. of a mixture of cultures of *S. cholera* intravenously, the dose corresponding to about half an agar slope or culture. The pig died in 36 hours from an acute septicaemia. Pig 1197 received 500 c.c. per os of the same culture emulsion. It did not show any symptoms subsequently. A further experiment was then carried out in which two pigs were starved overnight and each given 500 c.c. of a dense emulsion of several strains of *S. cholera suis* from the cases in the outbreak. Each pig was given a mixture of four carbonates (to counteract gastric acidity) one with the culture and one half an hour before the culture to neutralize the gastric juice. These pigs did not show any symptoms of illness subsequently. Attempts to produce typical paratyphoid cases in pigs experimentally with cultures from the outbreak, therefore, failed.

In summarising the 1937 outbreaks at the Savoy Estate, it may be stated that at post-mortem examination one found fairly marked peripheral haemorrhagic lymphadenitis, hydrothorax and hydropericardium; pulmonary oedema and bronchial pneumonia; diphtheroid an haemorrhagic gastritis and enteritis (boutons); cyanosis of skin, fat saponification and necrosis of the pancreas; disseminated necrosis of liver (paratyphoid). Very few pin point haemorrhages in the kidney.

Microscopically the examination of organs from these cases showed in the majority disseminated necrosis with endothelioid proliferation typical of paratyphoid in the liver. In addition there were lesions of catarrhal bronchopneumonia and oedema. Acute gastro-enteritis—some catarrhal and a few diphtheroid in type. The lymphatic glands showed a varying amount of blood extravasation in the sinuses, but only a very slight degree of vascular hyalinisation and karyorrhexis. In the spleen there were the usual disseminated necrotic foci but the Schweiger Seidel sheaths were mostly intact or only slight karyorrhexis was present. Other organs also showed slight karyorrhexis and scattered haemorrhages.

The 1939 *Outbreaks.*

A further outbreak occurred on the same estate early in December, 1939. Throughout the year the average number of pigs running on the estate has been about 3,000 with an average daily mortality of three pigs. There was a sudden rise in the death rate from the 6th of December.

The following is the record of deaths reported:—

6th—6, 7th—8, 8th—4, 9th—3, 10th—5, 11th—7, 12th—16, 13th—15, 14th—7, 15th—12, 16th—1, 17th—7, 18th—8, 19th—8, 20th—2, 21st—1, 22nd—2.

A number of small pigs of the pork type had been purchased for the Christmas period. There was also a number of poor quality pigs which did not seem to be thriving.

Post-mortems on 16 pigs on the 13th practically in all cases revealed the following: haemorrhages of the skin, severe gastritis with diphtheric membranes, enlargement of the spleen, haemorrhagic periportal, mesenteric, and gastric lymphatic glands, slight haemorrhages noted on the kidneys. These were regarded strongly suspicious of swine fever.

A number of carcasses were brought to Onderstepoort for post-mortem, histological and bacteriological examinations. Blood from some of these pigs inoculated into susceptible ones gave no reactions. *S. cholerae suis* in pure culture was obtained from the blood. As regards the pathology Dr. Thomas reported as follows:—

Pig No. 1 (Specimen No. 24184):

Cyanosis of the skin; phlegmosis of the sheath, general haemorrhagic appearance of the lymphatic glands, localised acute pneumonia, ulcerative gastritis, extensive catarrhal haemorrhagic enteritis.

Gland (pptl).—Marked extravasation in cortical sinuses with fibrin in parts and very little karyorrhexis and no hyalinisation of arterioles.

Spleen.—Rich in blood, s.s.s. are well preserved, practically no karyorrhexis, lymphoid centres not affected markedly, a few isolated necrobiotic centres.

Pig No. 2 (Specimen No. 24185):

Cyanosis of the skin with extensive multiple localised ulcers and haemorrhages in various stages, slight tumour splenis, diphtheroid gastritis with slight catarrhal enteritis, haemorrhages lymphatic glands, slight acute pneumonia.

Gland.—Same as above, small centres of fibrinous exudate, but less extravasations.

Spleen.—As above, less marked.

Skin.—Multiple crush covered-necrotic defects scattered over skin, some with a dark red edge, to blood filled cutis around, bacteria in necrotic bed.

Pig No. 3 (Specimen No. 24186):

Gland.—Irregular necrotic areas with thrombosed vessel, fibrin, haemorrhages in sinuses, slight karyorrhexis, but lymph follicles unaffected.

Spleen.—Pulpa rich in blood, s.s.s. still visible though much distorted, many necrotic areas isolated karyorrhexis not marked.

No liver was collected.

Diagnosis.—Paratyphoid.

Pig No. 4 (Spec. No. 24191) killed *in extremis*:

Haemorrhages lymphatic glands, ecchymoses kidneys, slight acute catarrhal gastro-enteritis, extensive acute pneumonia.

Lung.—Hyperaemia and irregular patches of consolid. with fibrin, serum and red cells.

Gland.—Slight peripheral extravasation.

Liver.—Scattered pin point lesions, some filled with red cells, others just an accumulation of endothelial cells.

Spleen.—Practically normal appearance.

Pig No. 6 (Spec. No. 24193) killed *in extremis*:

Ecchymoses lung and kidneys, fatty degeneration liver, few haemorrhages and cyanosis skin, slight acute gastro-enteritis.

Gland.—Hyperaemia and few necrotic centres.

Spleen.—Small indistinct necrotic foci. s.s.s. still present.

Liver.—As above but more extensive.

Pig No. 5 (Spec. No. 24192) killed *in extremis*:

Intensive patchy cyanosis of the skin, intense acute pneumonia and haemorrhages bronchial lymph glands, slight acute gastro-enteritis.

Kidney.—Hyalinisation of some glomeruli with consequent local haemotaria and degeneration of some tubules.

Spleen.—No great change.

Liver.—Disseminated endothelial, prolif, in irregular foci.

A definite diagnosis of *paratyphoid* was made histologically, supported by bacteriological examination and the fact that the blood inoculation into susceptible pigs was negative.

APPENDIX IV.

Experiments on *the relationship of the European and African viruses of swine fever.* In most of these experiments the virus used, 1006, was obtained from the Witwatersrand outbreaks.

Experiment 1:

12th June, 1934. Two pigs were inoculated with 2 c.c. of virus 1006 subcutaneously, one pig receiving 50 c.c. of Swine Fever Antiserum (Lederle) in the opposite hind leg. The pig that received virus alone died on 4th day from acute swine fever and the pig that received serum died on 6th day after inoculation.

Experiment 2:

11th June, 1934. Two pigs were put in an infected stable. One pig received 50 c.c. Swine Fever Antiserum (Lederle) subcutaneously. This pig died of acute swine fever on 15th day, the control animal dying on 11th day.

Experiment 3:

25th June, 1934. Two pigs were again placed in an infected stable, one pig receiving 30 c.c. antiserum (Lederle) subcutaneously. Both pigs survived, apparently owing to the infection in the stable having died out.

Experiment 4:

17th August, 1934. Two pigs were inoculated subcutaneously each with ·2 c.c. of a virus obtained from another outbreak in the Northern Transvaal. One pig received 30 c.c. antiserum (Lederle) subcutaneously. Both pigs died of acute swine fever on 6th day.

Experiment 5:

17th August, 1934. Two pigs were inoculated subcutaneously each with ·2 c.c. of a virus from Gouda in the Western Province. One pig received 30 c.c. antiserum (Lederle) subcutaneously. Both pigs died of acute swine fever on 7th day.

Experiment 6:

26th July, 1934. Two pigs were inoculated with virus 1006 (Johannesburg) each receiving ·2 c.c. subcutaneously. One pig received 30 c.c. antiserum (Lederle) subcutaneously. The latter pig survived but when reinoculated on 16th August, 1934, with ·2 c.c. of the same virus it died of acute swine fever. The control pig died on 7th day.

As this survival of the initial inoculation with virus was of great interest, the experiment was repeated (Experiment 7) but this time both pigs died.

Experiment 7:

16th August, 1934. Two pigs were inoculated with virus 1006 (Johannesburg) each receiving ·2 c.c. subcutaneously. One pig was given 30 c.c. antiserum (Lederle) subcutaneously. The latter died on 6th day from acute swine fever and the control pig on 5th day.

At this point it was realized that perhaps the dose of virus was too big, so an attempt was made to roughly determine the minimum lethal dose of virus 1006. It was found that ·01 c.c. would kill in 7 days so this dose was used as probably representing several lethal doses.

It was decided to change over to the method of incubation of the serum virus mixture as previously mentioned and in all the subsequent experiments this method was used.

Experiment 8:

1st September, 1934. One pig was inoculated with ·2 c.c. virus (1006) and another with an incubated mixture of ·2 c.c. virus 1006 and 30 c.c. antiserum (Lederle). The virus pig died on 5th day but the serum virus pig survived to the 13th day. In all the experiments where incubated serum virus mixture was used the pigs were kept in separate boxes for fear that contact with the reacting virus pig would increase the exposure of the serum-virus pig.

Experiment 9:

25th September, 1940. Two pigs were inoculated subcutaneously each with ·01 c.c. of virus 1006. One pig received 30 c.c. antiserum (Lederle) which had been incubated with the virus. The pig which received virus alone died on 7th day from acute swine fever. The serum virus pig survived until 19th October, 1934, when it developed swine fever and died on 24th October, 1934.

There are two possible explanations of this late reaction, one being that accidental infection occurred, but in view of experiments on the recovered pig as a carrier it is possible that the virus was not completely neutralized by the serum.

Experiment 10:

11th October, 1934. This experiment was carried out to see whether normal pig serum would have any neutralizing effect on the virus. A pig was given ·01 c.c. virus which had been incubated with 30 c.c. normal pig serum under the same conditions as used for antiserum and virus. The pig died on 5th day from acute swine fever, so one may conclude that normal pig serum was without effect on the virus.

Experiment 11:

24th November, 1934. This was a repetition of experiment 9. The pig received ·01 c.c. virus 1006 alone, and died on 5th day. The pig which received serum survived. The survivor was not tested for immunity in this case.

In order to see whether serum obtained from other sources would give results similar to those obtained with the American antiserum (Lederle), some was obtained from Germany (Bayer) and from Hungary.

Experiment 12:

20th March, 1935. One pig was inoculated subcutaneously with 50 c.c. Bayer antiserum with ·01 c.c. virus (1006), and another with 50 c.c. of the Hungarian antiserum and ·01 c.c. of the same virus. A third pig received ·01 c.c. virus alone. This pig died on the 6th day from acute swine fever. The two pigs which received serum both survived. They were kept for 6 weeks and then inoculated with ·01 c.c. virus (1006) to see whether they had acquired any immunity. Both pigs died of acute swine fever on 6th day after inoculation so that apparently no immunity had been acquired.

Experiment 13:

7th November, 1935. This was a repetition of experiment 12. Two pigs received ·01 c.c. virus (1006) with 50 c.c. Bayer antiserum. A control pig was given ·01 c.c. virus (1006) alone. This pig died of acute swine fever on the 8th day. The two serum pigs survived. It was proposed to carry out further virus tests with these two pigs to see if a basic immunity can be developed on which immunization experiments could be carried out.

These two pigs were not found to have developed any immunity when tested with virus later, and both died of the acute disease.

Conclusions.

The experiments show that there is some evidence of a common antigenic factor between the African and European viruses of swine fever.

APPENDIX V.

Experimental work carried out with the blood of pigs suspected of being *carriers of swine fever virus.* Blood was obtained from a number of pigs in the recent Western Province outbreak.

Pig 1.

The outbreak occurred during November, 1934. The pig was ill for three weeks and showed reddening of the skin and swelling of the joints.

At post-mortem in April, 1935, small abscesses were seen in the lungs, pericardial adhesions, slight enlargement of the spleen, hyperaemia of the mesenteric glands. There were definite traces of the previous acute condition.

Two pigs 1070 and no number (S. 5679) were inoculated on 3rd May, 1935, each with 5 c.c. of blood. One pig reacted from 12th day and died on 20th from acute swine fever; the other reacted from 24th day and died on 28th. It is possible that the second pig became infected from the first as they were in contact.

Pig 2.

The outbreak occurred in September, 1934. The pig was ill for four weeks. At the time of the post mortem in April, 1935, the pig showed no symptoms. At post-mortem there were haemorrhages in the kidneys, and an adhesive pericarditis but no other lesions.

Two pigs 1068 and 1071 (S. 5670) were inoculated on 3rd May, 1935, each with 5 c.c. of citrated blood. No symptoms developed in the following two months. The blood of pig 2 was therefore not infective.

Pig 3.

The outbreak occurred in September, 1934. There was no information available as to whether the pig showed any symptoms at the time.

At the post-mortem in April, 1935, the following lesions were found. An adhesive pericarditis, an abscess in the myocardium, slight hyperaemia of the intestines, marked tumor splenis, marbled appearance of the periportal lymphatic glands and some haemorrhages in the kidneys. There was definite evidence of a previous infection.

Two pigs 1091 and 1094 (S. 5675) were inoculated each with 4 c.c. blood on 17th May, 1935. One pig reacted on 16th day and died on 20th from acute swine fever. The second pig reacted from 20th day and died on 26th. This pig was in contact with the other and may have become infected from it.

Pig 4.

The outbreak occurred in September, 1934. The pig was ill for four weeks and showed swelling of the joints.

At the post-mortem in April, 1935, it was in poor condition and showed pericarditis, mottling of the mesenteric glands, and slight splenic enlargement.

Two pigs 1092 and 1093 (S. 5675) were inoculated on 17th May, 1935. One pig reacted from the 25th day with death on 31st and the other from 24th with death on 30th. The inoculation period in these two cases was exceptionally long.

Pig 5.

The outbreak occurred in June, 1934. The pig was ill for seven weeks at the time of the outbreak.

At the time of the post-mortem in April, 1935, it was in good condition. The following lesions were seen at post-mortem: Old standing pericarditis, slight swelling of the joints, areas of broncho-pneumonia, mottling of the periportal glands, petechiae of kidneys.

One pig (S. 5679) was inoculated with 5 c.c. of citrated blood on 25th May, 1935. It reacted on 12th day and died on 16th from acute swine fever

Pig 6.

The outbreak occurred in June, 1934. There was no information available as to symptoms shown at the time of the outbreak.
At the time of the post-mortem there was evidence of swollen joints. At post-mortem the periportal glands showed a mottled appearance and old haemorrhages were seen in the kidneys.

A pig (S. 5679) was inoculated on 25th May, 1935, with 5 c.c. citrated blood. It reacted from 8th day and died on 12th with symptoms of acute swine fever.

Pig 7.

The outbreak occurred in December, 1934. The pig was ill for three weeks at the time of the outbreak.

At post-mortem in April, 1935, old pleural adhesions were found and haemorrhages in the kidneys, slight mottling and hyperaemia of the mesenteric glands.

Two pigs 1095 and 1096 (S. 5684) were inoculated, each with 5 c.c. citrated blood on 5th June, 1935. One pig reacted from 5th day with death on 15th from acute swine fever. The second pig reacted from 11th day with death on 16th. It may have become infected from the other pig, with which it was in contact.

Pig 8.

The outbreak occurred in September, 1934. At the time of the outbreak, the pig was sick for two weeks, showing staggering gait, swollen joints, swelling of the ears and eyes and blood oozed from the tips of the ears. At post-mortem on 26th June, 1935, the pig was in excellent condition. The spleen showed hyperaemia and the mesenteric and peri-portal glands showed enlargement and mottling.

Two pigs (S. 5699) were inoculated on 26th June, 1935, each with 5 c.c. of blood. Neither of these pigs showed any symptoms subsequently.

In addition to the eight pigs just referred to, blood was sent from two pigs killed at the Wellington Bacon Factory in January, 1935. Both these pigs had shown symptoms during an outbreak of Swine Fever in the Malmesburg District of the Western Province, nine months previously. Both cases showed evidence of previous swelling of the joints. There was slight enlargement of the spleen, but the main lesions were those of a bronchopneumonia and pleural adhesions. On inoculation of blood from these two pigs into susceptible pigs, no symptoms developed.

Two pigs 1104 and 1180 survived in experiments carried out at Onderstepoort. Both were subsequently reinoculated with viruses of different origin and survived.

Pig 1104 was inoculated again two months afterwards but did not show a reaction. It was kept for transmission experiments and its blood was inoculated into susceptible pigs at three monthly intervals for a year but the blood did not at any time produce a reaction, so the animal was apparently not a carrier.

Pig 1180 was bled twice after passing through the reaction, at five months and at eight months. No reaction occurred when the blood was inoculated into susceptible pigs. A susceptible pig was placed in contact with 1104 for several months but did not show any sign of a reaction.

Conclusions.

The existence of carriers was demonstrated in a number of pigs which had survived swine fever outbreaks in the Western Province which had occurred up to eight months previously.

APPENDIX VI.

Experimental work carried out on mice and guinea-pigs in the attempt to transmit swine fever to them.

For all the inoculations on mice a quantity of ·05 c.c. was used and for guinea-pigs ·1 c.c.

Experiment 1.

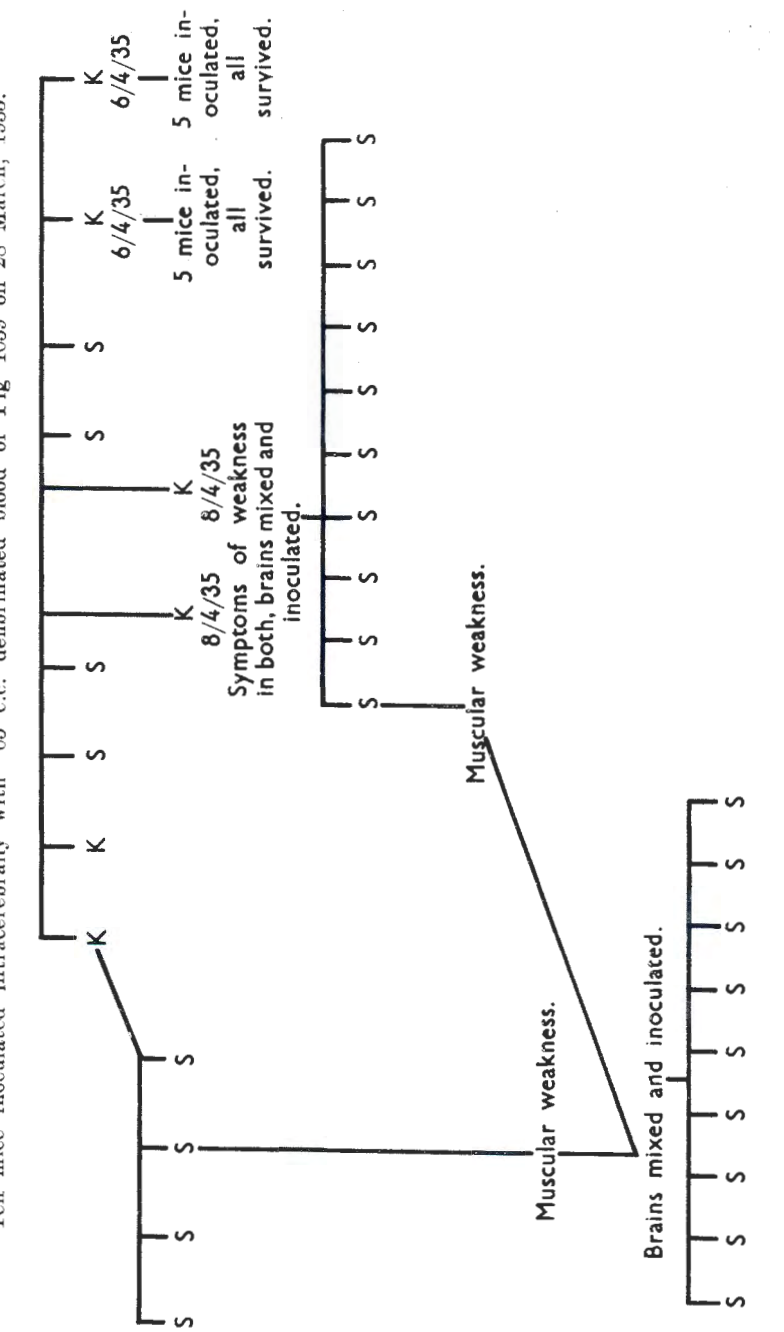
Defibrinated blood was taken from a pig 1059 on 28th March, 1935, immediately after death. The particular strain is referred to elsewhere in the swine fever experiments as the Savoy Estate one.

Ten white mice were inoculated intracerebrally, each with ·05 c.c. of defibrinated blood on 28th March, 1935. On 5th April, 1935, two of the mice were killed and the brains removed. The brains were emulsified in normal saline, using 20 c.c. to each brain, and ·05 c.c. of each of the two inoculated into 5 more mice. On 17th April, 1935, twelve days later, one of the mice from one lot showed muscular weakness, so it was killed and its brain added to that of a mouse showing weakness in another sub-inoculated lot from mice in the original ten. No symptoms developed subsequently in the sub-inoculated mice. Two other mice of the original ten were killed although showing no symptoms, and their brains inoculated into mice without results. The inoculations of the mice are schematically represented in Table I.

The surviving mice were all kept for at least twenty days after inoculation in order to see whether symptoms would develop.

TABLE I.

Ten mice inoculated intracerebrally with ·05 c.c. defibrinated blood of Pig 1059 on 28 March, 1935.



Experiment 2.

Defibrinated blood was taken from a pig 1096 on 24th June, 1935, shortly after death.

Ten white mice were inoculated intracerebrally on 24th June, 1935, each with ·05 c.c. of defibrinated blood. Five of the mice received an intramuscular inoculation of ·05 c.c. of India ink as well. The ink was given with the idea of reducing the resistance of the animals.

On 1st July, 1935, seven days after inoculation, one mouse injected with ink and one which had not, were killed and a mixture of the brains inoculated into six mice, intracerebrally. One of these showed weakness on 6th July, 1935, and was killed, six more being inoculated with its brain. On 12th July, 1935, two of the apparently healthy mice were killed, and a mixture of their brains inoculated into seven mice intracerebrally. One of these mice died on 18th July, 1935, and its brain was inoculated into five mice intracerebrally. On 6th August ,1935, two of the inoculated mice showing signs of weakness were killed. A mixture of their brains was inoculated into six mice intracerebrally but these animals did not show any symptoms subsequently.

Table II shows a schematic representation of the inoculations.

Experiment 3:

In addition to the mice, six half-grown guinea-pigs were inoculated intracerebrally each with ·1 c.c. defibrinated blood from pig 1096 on 24th June, 1935. On the day of inoculation and on the following day each guinea-pig was given ·5 c.c. of India ink intramuscularly.

One of these guinea-pigs died on 30th June, 1935, and its brain was inoculated intracerebrally into four more half-grown guinea-pigs. These animals did not show any symptoms subsequently. Two of the other guinea-pigs were killed on 5th July, 1935, and their brains removed. A mixture of these two brains was emulsified in saline and four more guinea-pigs inoculated intracerebrally. These guinea-pigs remained healthy. No success attended the attempt to infect six white mice intranasally. Temperature charts were kept of the first six guinea-pigs, but apart from an initial rise of temperature in the first 24 to 48 hours the temperatures remained normal.

Experiment 4:

To see whether the virus inoculated into the brains of mice could maintain itself or whether it disappeared.

Two pigs were inoculated with brain material from a mouse and a guinea-pig. One pig, 1103, was inoculated subcutaneously with the whole brain of a mouse inoculated eight days previously with virulent swine fever blood. The other one, 1104, was inoculated subcutaneously with the whole brain of a guinea-pig inoculated eight days previously with virulent swine fever blood. In neither case did any temperature reaction or other symptoms occur in the following six weeks.

Two pigs, 1101 and 1102, were inoculated on 12th August, 1935, each with 5 c.c. of an emulsion of the brain of a mouse inoculated four days previously. This mouse was one of the fourth generation of sub-inoculations from virulent blood (see Expt. 2). No symptoms developed subsequently in either of the pigs and both subsequently died of swine fever when tested for immunity.

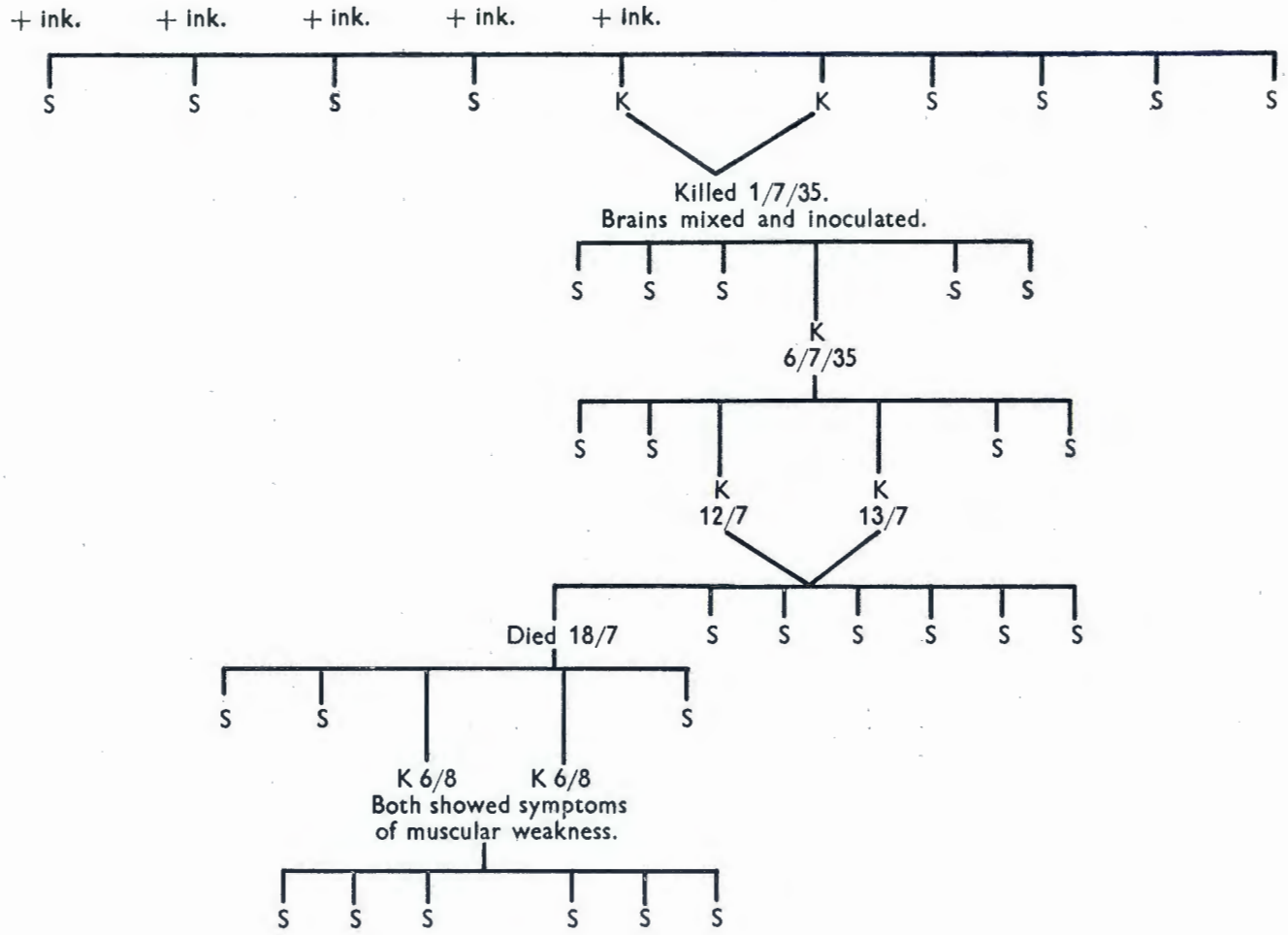
Two pigs, 1105 and 1106, were inoculated subcutaneously each with 5 c.c. of an emulsion of the mixed brains of two mice killed for transmission experiments. These mice were in the third generation from virulent blood (see Expt. 2). No symptoms were noted in the pigs for two months, when they were exposed to swine fever infection and one died, the other recovering after a very severe reaction.

Conclusions.

No success attended attempts to transmit the virus of swine fever to laboratory animals.

TABLE II.

Ten mice inoculated intracerebrally with ·05 c.c. defibrinated blood of pigs 1096 on 24th June, 1935.



APPENDIX VII.

Experiments on immunization of pigs against swine fever.

Experiment 1:

Virus incubated with crystal violet solution for 14 days at 37·5° C. was inoculated subcutaneously into two pigs, 1078 (1 c.c.) and 1081 (5 c.c.). These pigs did not show any reaction for two months. They were then tested to see if any immunity had developed. Both developed acute swine fever and died on 7th day after inoculation. As the result of this test indicated that the virus had apparently been rendered completely inactive in 14 days and the inoculated pigs had developed no immunity, it was decided to try the virus incubated with crystal violet for a shorter period.

Experiment 2:

Two pigs, 1149 and 1150, each received 5 c.c. subcutaneously of a virus-crystal violet mixture incubated at 37·5° C. for 7 days. Both pigs died of acute swine fever on 7th day after inoculation. The period of 7 days incubation did not have any effect on the virus.

Experiment 3:

A pig was inoculated subcutaneously with 5 c.c. of a sample of defibrinated blood from a case of African swine fever at the height of the reaction and which had been incubated with crystal violet solution for 10 days. The pig died on 5th day after inoculation from the acute disease.

Experiment 4:

The same material as was used in experiment 3 was incubated for 16 days and then 5 c.c. were inoculated into pig 1170 subcutaneously. The pig died on 6th day from swine fever.

The few preliminary experiments described do not give any indication as to the value of crystal violet vaccine for swine fever immunization. In the one experiment where the virus was avirulent after 14 days incubation, there was no immunity conferred on the pigs.

The time taken to attenuate the virus appears to be variable. Further experiments on the action of crystal violet vaccine on pigs should be carried out though it seems unlikely that any method of immunization will produce a durable immunity in this disease.

Conclusions.

No success attended attempts to immunize pigs against swine fever of the S. African " strain " by the crystal violet method of Dorset.

APPENDIX VIII.

CONTROL MEASURES.

The control of an infectious disease like Swine Fever in the Union of South Africa is hampered by many difficulties, e.g., distance between farms and between farms and markets, lack of proper hygiene and sanitation, lack of control (only a small proportion of pigs are kept in styes), the large veterinary areas, large numbers of European and Native owners, especially in Northern Transvaal, the wild pig as a carrier of the virus in certain areas, etc. Control and eradication of this disease were more or less effected on the following lines:—

(1) According to the provisions of the Stock Diseases Act every suspicion of Swine Fever, and in fact every mortality in pigs where a number of animals had died suddenly, had immediately to be reported.

(2) Immediately swine fever or suspicious cases occurred on a farm or elsewhere, the farmer or owner of the pigs had to stop the movement of pigs, meat, offal, etc., from his farm or property. All meat used on such a farm or property had to be boiled.

(3) The farmer or owner had to take immediate steps to report to his neighbours any suspected cases of Swine Fever on his farm or property. No affected pigs were allowed to be treated or slaughtered on such farms or properties, but were placed under effective control in yards or pens. The owner had to do everything in his power to prevent swine fever from being carried to uninfected farms or properties, by disinfecting as far as possible shoes, hands, implements, etc., coming in contact with infected pigs.

(4) If the veterinary officer or his representative had diagnosed Swine Fever, the affected farm was immediately placed under quarantine, and all the pigs on it placed under strict control. A valuation was made of all the pigs, and authority obtained from the Minister for slaughter with compensation on the scale prescribed in the Stock Diseases Act, valuation being made on existing market prices. The Department had to be satisfied that the owner took all precautionary measures and destroyed and buried all pigs according to prescribed instructions.

(5) The farmer had to do everything in his power to disinfect or burn out the styes and remove the dung from the styes for use as manure on land not utilised for pigs.

(6) No new pigs were introduced for a period of at least three months after the farm had been cleared of pigs.

(7) Restocking of the farm or property was only allowed from uninfected farms or properties under permit. The chief veterinary officer had to satisfy himself that all due precautionary measures had been taken. The introduction of new pigs were carried out gradually. All pigs passed through a system of quarantine pens, in which they were confined for at least three weeks before being introduced into the herd.

(8) No pigs were allowed to be moved out of any area declared infected with Swine Fever unless the person in charge had written permission from the Veterinary Officer. All vehicles used for the transport of these pigs were thoroughly disinfected immediately after use.

(9) No person was allowed to have access to swine affected or suspected of being affected with Swine Fever unless it was a person whose access was necessary for the proper care and treatment of such swine, or an officer acting under the powers of the Act or the regulations.

(10) No person who was in contact with swine affected or suspected of being affected with Swine Fever was allowed to approach other swine, or leave the place until his hands and boots (or, in the case where boots are not worn, his feet) were properly disinfected.

(11) It was the duty of the person in charge of swine which had been in contact with other swine suffering from Swine Fever to isolate the same for such a time and in such a manner as the Government Veterinary Officer prescribed.

(12) No person was allowed to move manure or litter from premises occupied by swine suffering or suspected to be suffering from Swine Fever except by permission of the Government Veterinary Officer and subject to such precautions as he prescribed.

(13) No hair, claws, heads, offal, excreta, or any other products derived from pigs was allowed to be moved out of or from any one place to any other place within any area declared infected with Swine Fever, except on the authority of a permit issued by the Government Veterinary Officer and subject to such conditions as he might impose.

(14) When Swine Fever had died out on a farm or property and a number of pigs had survived, such farm or property was kept under quarantine and no pigs were allowed on it unless all the remaining pigs were either destroyed or removed, under permit, to an approved abattoir for slaughter.

(15) As wild pigs (warthogs and bush pigs) and domestic pigs which have become wild, may become carriers of the virus of Swine Fever, all possible measures had to be taken to eradicate them from farms or properties in infected areas.

According to Administrator's Proclamation No. 40 of 1936, both warthogs and bush pigs were classed as vermin in the Transvaal and farmers were urged to destroy them.

(16) *Vigilance Committees* of farmers' associations were established in the quarantine areas to assist the Department:—

(*a*) By making propaganda amongst the farmers in their areas as regards the importance of *carriers* of African Swine Fever, symptoms of the disease, post-mortems, changes, regulations etc.

(*b*) By working in close association with the Government Veterinary Officer of the district in an advisory capacity and to persuade farmers to report immediately suspected outbreaks of the disease in pigs to the Veterinary Officer of the district, to assist in the control of illicit movements, to obtain information from farmers as regards occurrence of wild pigs, to assist the Department in the destruction of such wild pigs, and to compile a census of pigs in the quarantine areas.

The following Movement Notices were published to control Swine Fever:—

1. For the Western Province:
   G.N. 616 of 11.5.1934.
   G.N. 715 of 1.6.1934.
   G.N. 342 of 13.3.1936.
   G.N. 1740 of 20.11.1936.
   G.N. 1950 of 31.12.1936.
2. For the Witwatersrand (Transvaal):
   G.N. 344 of 16.3.1934.
3. For the Northern Transvaal:
   G.N. 636 of 10.5.1935.
   G.N. 920 of 28.6.1935.

In Northern Transvaal the Magisterial Districts of Pietersburg, Zoutpansberg, Lydenburg, Potgietersrust, Waterberg and Letaba were declared an area infected with swine fever and from there the movement of swine is only permitted to the quarantine section of an approved abattoir for immediate slaughter.

This legislation was provided to curtail the movements of swine from an area believed to harbour warthogs and bush pigs, amongst which a limited number may be carriers of swine fever virus.