Swine vesicular disease: Clinical signs, diagnosis, epidemiology and control*

J.G. LOXAM** and R.S. HEDGER***

Summary: In its acute form swine vesicular disease (SVD) is clinically indistinguishable from foot and mouth disease (FMD) and can be differentiated only by the use of laboratory tests. The emergence of mild SVD, with transient signs which often go unnoticed or disregarded, has resulted in cases of the disease being not reported and detected only after careful veterinary examination and often only as a consequence of investigation into positive blood test results disclosed in SVD serological surveys.

A variety of sensitive and specific laboratory tests is now available which provide a reliable basis for the surveillance, differential diagnosis and confirmation of the existence of SVD.

The counter-immunoelectro-osmophoresis test (CIEPT) is ideally suited to screening sera for diagnosis, for use in epidemiological investigations and for routine surveillance.

The epidemiology of SVD is related essentially to the extraordinary persistence of the causal virus both in pig meat and outside the host. Consequently strict control is necessary of the handling and processing of waste food (i.e. household scraps, etc.) for feeding to pigs, of the marketing of pigs and of vehicles used to transport pigs.

The eradication of SVD is of vital importance in countries in which the control of FMD is based on a policy of non-vaccination and stamping-out.

INTRODUCTION

Swine vesicular disease (SVD) is a contagious disease of pigs caused by an enterovirus and characterised by a mild fever and vesication of the coronary


** Assistant Chief Veterinary Officer, Ministry of Agriculture, Fisheries and Food, Tolworth, Surbiton, Surrey (U.K.).

*** Principal Veterinary Research Officer, The Animal Virus Research Institute, Pirbright, Woking, Surrey (U.K.).
band, the heels, skin of limbs and less frequently of the snout, lips, tongue and teats. Clinically indistinguishable from foot and mouth disease (FMD) its control is vital in countries free of vesicular disease.

First seen in the Lombardy region of Italy in 1966, and identified at the Animal Virus Research Institute, Pirbright, Surrey, as a new disease, it was recognised in Hong Kong in 1971. Simultaneous outbreaks occurred in a number of European countries in 1972/73 and by the end of 1981 outbreaks had been reported in a total of 13 countries (Table I).

**TABLE I**

*World distribution of reported SVD outbreaks*

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Britain</td>
<td>13</td>
<td>137</td>
<td>187</td>
<td>45</td>
<td>3</td>
<td>18</td>
<td>43</td>
<td>60</td>
<td>12</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Austria</td>
<td>3</td>
<td></td>
<td>5</td>
<td>3</td>
<td>6</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Belgium</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>France</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Greece</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hong Kong</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Italy</td>
<td>2</td>
<td>23</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Japan</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malta</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Netherlands</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poland</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Switzerland</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>West Germany</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

× = Actual number of outbreaks not recorded.

The disease first appeared in Great Britain, in Staffordshire, in December 1972. Up to 31 December 1981 there have been a total of 518 outbreaks in Great Britain and the longest period during which no outbreaks of SVD were confirmed was from June 1977 to February 1979, a total of 20 months (Figures 1 and 2).

**CLINICAL SIGNS**

SVD may appear in a variety of clinical syndromes, varying from inapparent infection to severe clinical disease indistinguishable from FMD. Detection of mild disease under some modern labour-saving husbandry conditions, particularly on premises where waste food is fed, is often difficult.

The incubation period varies from 2 to 7 days and is dependent on the infecting dose and on the route of infection. On occasion incubation periods
Figure 1
Annual incidence of SVD in Great Britain, 1972-1981

No. of outbreaks

FIGURE 2
SVD in Great Britain, 1979-81

No. of outbreaks

MONTH
YEAR

1979

1980

1981
longer than 7 days have been suspected but in such cases it is possible that the initially infected pigs were not recognised. Experimentally, intradermal injection of virus into the foot produces lesions within 48 hours.

Exposure to virus by inhalation or by ingestion may result in subclinical infection, with an associated antibody response. There is, so far, no evidence that infected pigs excrete significant amounts of virus in the absence of clinical signs of disease. In Great Britain there is no field evidence of a virus carrier state nor has it been possible to demonstrate persistence of virus after experimental infection.

Clinical signs include pyrexia initially (up to 41°C), but temperatures usually fall to normal within 2-3 days. Vesicles develop on the coronary bands of the feet (including accessory digits), less frequently on the snout and more rarely on the lips and tongue and teats. On the feet vesicles are not necessarily restricted to the junction of skin and horn and may, on occasion, extend up the limb. In the early stages of disease, affected animals may be anorexic and lame. However, in SVD vesicles normally rupture easily; following rupture lameness disappears and a shallow area of granulation tissue remains and, where vesication occurs on the coronary band, the associated wall of the hoof separates from the underlying tissue. It is rare for the complete hoof to become detached, as occurs with FMD, but the line of separation at the coronary band is later visible as a horizontal line, dark in colour, which moves downwards with horn growth. Old disease may be suspected by such horizontal marks and their distance downwards from the coronary band may be used to assess the time at which infection occurred (Watson, 1981).

No evidence has been found in Great Britain to suggest that lesions of vastly differing ages may be found in the same pig, although it is not uncommon to get a primary lesion followed 48 hours later by a crop of secondary lesions.

The severity of the disease may vary greatly. Younger pigs tend to show more severe clinical signs than older pigs but even in very young pigs recovery is usually fairly rapid and mortality is negligible.

Morbidity in the herd is variable but in individual pens may reach 100%. Spread between pens is also variable and is dependent upon movement of infected pigs or contaminated material.

**CLINICAL DIAGNOSIS**

Swine vesicular disease must be considered clinically indistinguishable from FMD. In Great Britain any vesicular condition in pigs is treated initially as suspected FMD, unless there is a known and established connection with a proven case of SVD. Confirmation of SVD on clinical grounds alone is, therefore, rare. In early disease a tentative clinical diagnosis is based on the presence of ruptured or unruptured vesicles on the coronary band and the
snout, lameness, anorexia, and pyrexia. In the later stages of disease it is based on horn separation and the presence of horizontal lines indicative of earlier separation of the hoof wall particularly when more than one digit and limb shows similar lesions. In making a differential diagnosis, other vesicular diseases, including FMD, must be considered and confirmation of SVD depends on laboratory tests.

LABORATORY DIAGNOSIS

The laboratory diagnosis of SVD depends on either the isolation and characterisation of the virus, the demonstration of specific antibodies or a combination of both. It is characteristic of SVD that infection is usually of longer standing than is initially suspected from the appearance of the lesions. Even in outbreaks where only unruptured and recently ruptured vesicles are described suggesting very early disease, if the affected pigs and their contacts are bled antibody in high titre will be found in some of the group in the great majority of cases. Serological tests are particularly useful, therefore, not only in long standing disease when vesicular epithelium for the demonstration or isolation of virus is no longer available, but also in new disease when the amount of material submitted may be insufficient to obtain a quick result by direct complement fixation (CF) test.

A. VIRUS

a) Direct complement fixation test.

The direct CF test is carried out on microtitre plates (Casey, 1965) using clarified 1 in 10 and 1 in 30 suspensions of ground vesicular epithelium as antigen. Reference antisera in the test include those for SVD and all seven immunological types of FMD virus. Fixation times vary from one hour at 37°C to the more sensitive overnight at 4°C. The time taken for a result may, therefore, vary. If the sample submitted is adequate in amount (0.3 g or more) and from a fresh unruptured or recently ruptured vesicle, then a result may be obtained in as little as 2 1/2 hours after arrival of the specimen in the laboratory. Where samples are insufficient or of poor quality it is necessary to enhance the virus by tissue culture.

b) Tissue culture.

Suspensions of lesion material are routinely inoculated on to monolayer cultures of primary calf thyroid, primary calf kidney and IBRS₂ cells in roller tubes. Foot and mouth disease virus grows in all three cell systems but SVD virus only replicates in IBRS₂ cells. When cytopathic effect is complete, usually in from 24 to 48 hours, the supernatant tissue culture fluid is harvested and subjected to the CF test for virus identification. Negative samples are given one blind passage at 48 hours in the appropriate cell systems.
c) Strain differentiation.

Differences between strains of SVD virus are small and difficult to detect by neutralisation tests. The polyacrylamide gel electrophoresis (PAGE) test (Harris and Brown, 1975) which detects differences in the structural polypeptides of the virus has, however, been used successfully in attempts to determine the origin of obscure outbreaks.

B. SEROLOGY

Counter immuno-electroosmophoresis (CIEP), enzyme-linked immunosorbent assay (ELISA) and double immunodiffusion (DID) tests are all routinely used in the diagnosis and in epidemiological studies of SVD. Results from the CIEP and ELISA tests can be obtained very rapidly within 2 to 2 1/2 hours and DID takes a little longer, from 8 to 20 hours. The micro-neutralisation test which is used to corroborate and quantify results of other tests, takes 3 days and is mainly used in epidemiological investigations.

In practice, it is found that the lesser sensitivity of precipitin tests and the ELISA test used can be overcome by increasing the number of sera tested from each farm or outbreak.

a) Counter immuno-electroosmophoresis.

The CIEP test (Bellhouse et al., in press) is carried out in 1% agarose gel in disposable Petri dishes, measuring 105 x 105 mm. The virus used is either formalin or acetyleneimine (AEI) inactivated tissue culture harvest concentrated by ammonium sulphate precipitation or hollow fibre ultrafiltration. Forty-eight sera may be screened in one dish. The test is specific, easy to perform, rapid and economic of reagents. It is thus ideally suited to screening sera for diagnosis, for epidemiological investigation and particularly for serological surveillance. Another advantage of CIEP is that, because inactivated antigen is employed, the test may be used outside the confines of a high disease security laboratory.

b) Enzyme-linked immunosorbent assay.

The rapid indirect ELISA test developed by Hamblin and Crowther (in press) is now used routinely for the serological confirmation of diagnosis of SVD. Results correlate well with those from serum neutralisation (SN), and although less sensitive than the SN test, this ELISA test compares favourably with the CIEP test and is more sensitive than DID. It is more complicated, however, in operation and needs more skill in interpretation than do the precipitin tests.

c) Micro serum neutralisation.

Serum neutralisation tests (Golding et al., 1976) are carried out on monolayers of IBRS2 cells in flat bottomed tissue culture grade microtitre plates
and SN titres are expressed as the reciprocal of the final dilution of serum present in the serum virus mixture at the 50% end point.

The SN test has the advantage of being a quantitative test. It is sensitive and specific but laborious and time consuming. It is therefore less useful as a diagnostic aid, when quick results are needed, or for the screening of large number of sera.

d) Double immuno-diffusion.

The DID test (Pereira et al., 1976) has been extensively used for the diagnosis and surveillance of SVD. Although now it has been largely replaced by CIEP and ELISA, which are more economical of antigen and give speedier results, it remains a valuable technique. Continuity of the precipitation line with that given by control positive sera, provides unequivocal evidence of serum specificity. The simplicity of DID and the ease of its performance make it particularly suitable for use by unpractised personnel in emergency situations at night and out of hours.

EPIDEMIOLOGY

The first appearance of SVD in Great Britain in 1972 is believed to have been as a result of the introduction of the virus in pig meat from abroad and subsequent feeding of improperly processed waste food containing that meat to domestic pigs. Later spread and persistence of the disease has, in large measure, been due to the resistance of the virus to environmental factors including pH changes and disinfection procedures which would inactivate most other pathogenic micro-organisms.

It is accepted that, despite the adoption of a stamping-out policy there have been occasions when infected pigs have been sent for slaughter before disease has been diagnosed in a herd. In consequence, despite considerable improvements in our control of the processing of waste food prior to its being fed to pigs, there is evidence that re-cycled virus in waste food has been the origin of infection on a number of occasions since 1972.

A further experience in Great Britain has been that, because of the extraordinary persistence of the virus outside the host, many outbreaks have been caused by the contamination of hauliers vehicles by infective pigs. Figures 3 and 4 illustrate the attributed origins of outbreaks of SVD in the two periods 1972 to 1977 and 1979 to 1981.

Unlike FMD virus, which is excreted in exhaled respiratory air, SVD virus appears to originate primarily from ruptured vesicles. Spread of disease locally from one premises to adjoining premises does not usually occur in the absence of the movement of infected pigs or contaminated materials. Airborne spread of SVD has not been recorded in Great Britain. Even on infected premises spread from one pen to another may not occur in the absence of a common...
No. of outbreaks

FIGURE 3
SVD in Great Britain, 1972 to 1977: Origin of disease
No. of outbreaks

Figure 4
SVD in Great Britain, 1979 to 1981: Origin of disease

Movement of pigs
Contaminated vehicles
Waste food
Market contact
Movement personnel or equipment
Local spread
Recrudescence
Obscure

ORIGIN
(open) drainage system, or of frequent movement of pigs between pens. It has been said that SVD is a «pen» disease rather than a «farm» disease.

The epidemiology of SVD is related essentially to the extraordinary persistence of the virus outside the host. SVD virus resists treatment with many commonly used disinfectants. In the presence of organic matter, it resists dessication. It withstands freezing and can survive normal fermentation and smoking processes used to preserve foods. It is destroyed by a temperature of 69°C, but is stable in the pH range between 2.5 and 12.0. It has been known to survive 400 days in dried salami and pepperoni sausages and in processed intestinal casings has survived for at least 780 days.

**CONTROL**

In Great Britain SVD is a notifiable disease and statutory powers necessary for its control are based on FMD control procedures. Legislation allows slaughter of all affected and contact pigs on a premises when the presence of SVD is confirmed and compensation for the pigs, at full market value, is paid to the owner.

All the movements onto and off infected premises, from 28 days before the estimated time of introduction of infection until the time of disease confirmation, are traced. Such tracings include movement of pigs, vehicles, equipment, personnel, etc. and, where it is considered necessary, pig movement restrictions are imposed on all premises from and to which such movements took place.

To comply with the requirements of the European Economic Community (EEC) notices prohibiting the movements of pigs except for slaughter are served on all the premises with pigs within a 2 km radius of an infected premises. These restrictions remain in force at least 15 days.

Following the confirmation of disease and slaughter of the pigs, carcasses are normally disposed of by incineration or burial on the premises. In some circumstances, under supervision, carcasses are moved off the premises, in special leak-proof vehicles, to an approved rendering plant for processing adequate to destroy all SVD virus.

Infected premises are then cleansed and disinfected under the control of Ministry of Agriculture staff, and usually at Ministry expense. Following a preliminary spraying with a disinfectant known to be effective against SVD virus, all surfaces and equipment are thoroughly cleansed, where necessary using a degreasing agent based on sodium metasilicate. The premises are then sprayed with 1% sodium hydroxide solution and 48 hours later all non-inflammable parts are flame-gunned. The sodium hydroxide/flame-gunning procedure is repeated after 14 days.

Limited restocking is allowed 8 weeks after completion of the final cleansing and disinfection. Initially up to 50% of the total number of original
stock (subject generally to an overall maximum of 200) is allowed on to the premises. The pigs are housed in those parts of the premises where infection was known to have been present and these sentinel animals are subjected to veterinary inspection on arrival and thereafter at weekly intervals for 3 weeks. If, after 3 weeks, no clinical signs of SVD appear in the sentinel pigs, full restocking is allowed.

WASTE FOOD LEGISLATION

Because of the persistence of SVD virus it became necessary to introduce stricter controls relating to the feeding of waste food to pigs and the Diseases of Animals (Waste Food) Order 1973 replaced existing legislation. This placed the most stringent requirements on waste food feeders and one effect of this has been the reduction in the number of licensed waste food feeders, from over 4,000 in 1973 to 872 by the end of 1981. This marked reduction must, in itself, have contributed significantly to the decrease in the number of SVD outbreaks in recent years.

MOVEMENT AND SALE OF PIGS LEGISLATION

Legislation was introduced, in 1975, to control further the movement of pigs and the cleansing and disinfection of markets and vehicles used to transport pigs. This legislation requires that :

1. All pigs movements, with certain exceptions, must be licensed.

2. The movement of pigs from premises (except direct to an abattoir for slaughter) is prohibited for 21 days following the movement of any pigs onto the premises.

3. Pigs from premises on which waste food is (or has in the last 3 months been) fed may move off ONLY direct to an abattoir for slaughter or to a « slaughter only » market from which ALL purchases must go direct to an abattoir.

4. Pigs being moved for slaughter are marked in a distinctive manner.

5. Vehicles used for the transport of pigs must be routinely cleansed and disinfected and the categories of pigs which can be carried are restricted.

SEROLOGICAL SURVEYS

Since SVD first appeared in Great Britain, continuing serological surveys have been carried out in an attempt to locate inapparent infection. Recent surveys in 1980 and 1981, involving the testing of over 53,000 serum samples, were as follows:
1. Yorkshire/Lancashire/Humberside waste food feeder survey:

In recent years, SVD appears to have localised in the Yorkshire/Lancashire/Humberside area. Pigs which have been fed on waste food and which originate from this area have been sampled at abattoirs when sent for slaughter. During 1981 this survey resulted in the detection of 3 outbreaks of SVD.

2. Sow feeder/dealer survey:

Evidence accumulated which suggested that many outbreaks, particularly in the last quarter of 1980, might have been the result of illegal movements of pigs. Associated with these movements were a number of sow feeder/dealer farms, especially in the Yorks/Lancs area. Arrangements were made, therefore, to collect blood samples from sows sent to abattoirs by known feeder/dealers. In 1981 this survey resulted in the detection of 7 outbreaks of SVD.

3. National survey of previously infected premises:

During 1981, when the prevalence of SVD in Great Britain appeared to be decreasing it was decided to extend our serum surveys. Arrangements were made to collect representative serum samples from pigs on all premises which had been restocked with pigs following a confirmed outbreak of SVD in 1979, 1980 and 1981. This survey has been completed and no seroconversion was revealed in pigs originating from any of these premises. These negative findings reflect the efficiency of the cleansing and disinfection procedures employed at premises on which SVD has been confirmed to exist.

4. National survey of waste food fed pigs:

In the last quarter of 1981, arrangements were made to collect, at abattoirs, serum samples from pigs originating from all the premises in Great Britain on which waste food is fed. At the end of 1981, pigs from 800 out of 872 eligible premises had been sampled and none had given positive results to tests for SVD antibodies.

5. National survey of meal-fed pigs:

Arrangements are currently in hand to collect, again at abattoirs, serum samples from a statistically significant, representative number of all the pig herds in the country not fed on waste food (i.e. meal-fed herds). The intention is to obtain 5-10 samples from a random-selection of 10% of such herds in G.B. (i.e. 17,500-35,000 blood samples from 3,500 herds) and the aim is to complete this survey by May 1982.

CONCLUSION

In the 518 outbreaks of SVD which have been confirmed in Great Britain during the period 1972-1981 and in which a stamping-out policy has operated,
a total of 311,713 pigs have been slaughtered and their carcasses destroyed, at a cost in compensation of over £11,360,000. In addition, over £4,808,000 has been spent in the cleansing and disinfection of infected premises.

For the last 3 years confirmed outbreaks have been confined mainly to the Yorkshire/Lancashire area of the country or, where they have occurred outside this area, have either been linked directly to outbreaks in that area or have occurred as isolated outbreaks related to the feeding of infected waste food.

The continuing serological surveys demonstrate that SVD is not widespread in Great Britain and therefore, notwithstanding the costs so far incurred and the heavy demands made upon veterinary resources, Great Britain is committed to the eradication of SVD. This is of vital importance in countries normally free of vesicular diseases and where the control of FMD is based on eradication by slaughter and non-vaccination.

* * *

REFERENCES


