TABLE 1: Production figures comparing the 13 week period before the outbreak of blue-eared pig disease and the 13 week period during infection

Performance parameters	Before the outbreak of infection	During blue-eared pig disease infection
Conception/pregnancy failures	24	53
Farrowing rate (%)	90·2	88·0
Born alive/sow/year	25·69	23·79
Born dead/litter	1·0	1·2
Born mummified/litter	0·1	0·5
Pre-weaning deaths/litter (%)	8·5	21·6
Piglets weaned/sow/year	23·51	18·65

cent, to below 75 per cent. Finally, there was also a recorded rise in the number of mummified piglets born compared to the units normal level of 1·0 per cent.

This unit illustrates one of the variations in the BEPD phenomenon that can be seen in the field. The low incidence of concurrent endemic disease in this unit appears to have reduced the losses from secondary infections. Treatments with salicylic acid, flunixin, oral electrolytes and prophylactic antibiotics were considered by the unit's owner and veterinary surgeon to have been beneficial. Overall losses though were dramatic and depressing and total productivity remained affected for some time (Table 1). Four months after the first signs of disease were identified in the unit the after effects of BEPD were still identifiable. Litter sizes were extremely variable; mummified piglet numbers were still slightly raised; there was a second peak of sow returns thought to relate to post infection infertility of the boars; and many sows debilitated by disease had to be culled earlier than planned.

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Antibodies to phocine distemper virus in Canadian seals

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THE mass mortality of harbour seals (*Phoca vitulina*) in Europe in 1988 resulting from infection with phocid distemper virus-1 (PDV-1) has been extensively described (Dietz and others 1989b, Osterhaus and others 1990, Örvell and Sheshberadaran 1991). PDV-1 is a newly-identified member of the genus *Morbillivirus*, and is distinct from both phocid distemper virus-2 (PDV-2), which resulted in the deaths of several thousand Baikal seals (*Phoca sibirica*) in 1987-88 (Grachev and others 1989, Osterhaus and others 1989a, Visser and others 1990), and canine distemper virus (CDV). The 1988 PDV-1 epidemic in Europe, which left about 60 per cent of the harbour seal population dead, led to speculation about the origin and nature of the virus, and the role that pollution may have played in weakening the immune systems of the seals (Dietz and others 1989b). During the European epidemic, there were no reports of unusual mortalities or sick seals on the east coast of Canada, which suggested at that time that Canadian seal populations had not been exposed to PDV-1. In fact, harbour seal pup production on Sable Island, a major breeding site in eastern Canada, doubled between 1978 and 1990 (W.T. Stobo, personal communication), suggesting no unusual additional mortality in the population during that period.

In a routine veterinary screen of serum samples collected from harbour and grey seals (*Halichoerus grypus*) on Sable Island, Nova Scotia, Canada (43°55'N; 60°00'W), in January 1989, virus neutralising antibodies to CDV were found in the majority of the samples (Table 1). In addition, two out of five serum samples from captive adult harbour seals at Dalhousie University, Nova Scotia, showed the presence of CDV-neutralising antibodies. Captured on Sable Island in May of 1988, these seals showed no signs of illness in captivity, and were isolated for the five month period before blood was sampled, suggesting infection before that time.

In an effort to identify the virus that induced these antibodies, and lacking adequate volumes of the original samples from 1988 and 1989, the authors collected further serum samples from nine adult female harbour seals on Sable Island in May of 1991. These samples were first screened in a CDV enzyme-linked immunosorbent assay (ELISA) described by Visser and others (1989). Again, CDV-positive samples were found: three of the nine serum samples were ELISA-positive (titre > 40). These three samples, along with one of the negative samples, were tested for antibody specificity in virus neutralisation (VN) as-

TABLE 1: CDV-neutralising antibody titres in serum collected from free-ranging adult grey (n=25) and harbour (n=5) seals on Sable Island in January 1989, and captive harbour seals (n=5) from Dalhousie University that were captured on Sable Island in May 1988

Species	Sex	CDV-VN titre
Grey seal	Male	<64
"	Female	<64
"	Male	<64
"	Female	<64
"	Male	<64
"	"	<64
"	"	<64
"	"	<64
"	Unknown	<64
"	"	64
"	Male	64
"	"	90
"	"	90
"	"	90
"	Unknown	90
"	Male	128
"	"	128
"	Unknown	128
"	Male	183
"	"	367
"	Female	512
"	Male	512
"	Unknown	512
"	Female	734
"	Female	734
Harbour seal	Female	<64
"	"	<64
"	"	64
"	"	256
"	"	1453
Captive harbour seal	Female	<64
"	"	<64
"	"	<64
"	"	512
"	"	734

Titres greater than or equal to 1:64 are considered positive. These preliminary assays were undertaken by Dr Campbell Cornwell, Department of Veterinary Pathology, University of Glasgow

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says. Although antibodies to different morbilliviruses crossreact to a certain degree in this assay, their relative specificities can be estimated by differential VN assays (Visser and others 1990). The authors used a panel of eight members of the genus *Morbillivirus* in the VN assays. The panel tested included CDV, PDV-1 and PDV-2, the recently isolated porpoise and dolphin morbilliviruses (PMV and DMV) (Osterhaus and others 1992), measles virus (MV Bussel strain), rinderpest virus (RPV RBOK strain), and peste des petits ruminants virus (PPRV Nigeria 75-1). VN assays were undertaken as described elsewhere (Visser and others 1990), with titres reflecting the ability of heat-inactivated serum to neutralise 30 $TCID_{50}$ of virus cultured in Vero cells. Whereas the control serum (CDV ELISA-negative) did not neutralise any of the viruses, all three CDV-ELISA positive serum samples showed neutralising activity to most or all of the morbilliviruses tested. Although there were considerable differences among the titres, they were consistently highest against PDV-1 (Fig 1). Transformation of titre values on the basis of PDV-1 = 1·0 demonstrates the relative specificity of the serum antibodies in the Canadian harbour seals for the viruses tested.

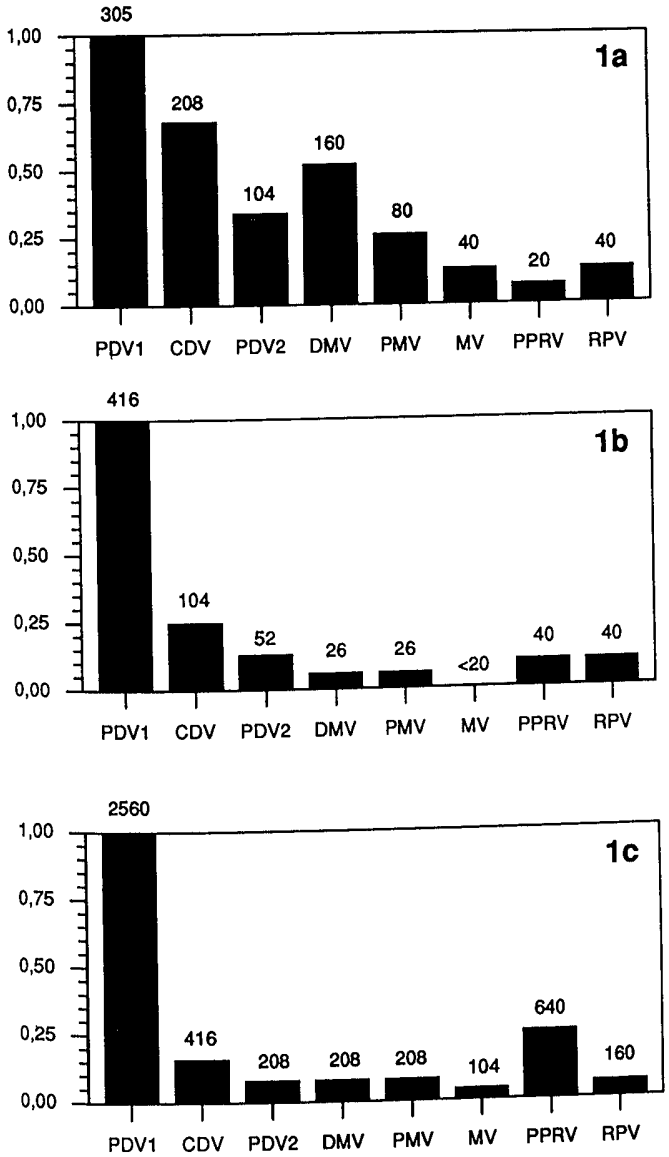

FIG 1: Proportional distribution of serum virus neutralising titres in three Canadian harbour seals (a, b, and c), as tested against different morbilliviruses. Virus isolates used include the known seal morbilliviruses PDV-1 and PDV-2; its canine equivalent CDV (Bussel); the two recently identified cetacean morbilliviruses DMV and PMV; the ruminant morbilliviruses PPRV (Nigeria 75-1) and RPV (RBOK); and the human measles virus MV (Edmonston B). Absolute titres are given above each bar, and reflect the ability of serum to neutralise 30 $TCID_{50}$ of virus. Bars represent the mean of duplicate assays, with variation between assays being consistently within one dilution factor

The serological data suggest that the virus which infected the Canadian seals is most closely related, if not identical, to the virus which caused the 1988 mass mortality in Europe. The authors' collective results also suggest that the virus is currently enzootic in the harbour, and probably grey, seal populations of southeastern Canada. It has probably been so since May of 1988 or before, although the authors cannot exclude the possibility that a different morbillivirus had induced the antibodies found in the samples from 1988 and 1989. Since the European outbreak began in the spring of 1988, further questions may be posed about the origin of PDV-1. Dietz and others (1989a) found canine distemper neutralising antibodies in 12 out of 40 harp seals (*Phoca groenlandica*) from the west coast of Greenland in 1985-86. The authors suggested that the virus responsible for these antibodies was PDV-1, and the subsequent mass migration of harp seals in 1986-87 southwards to northern Europe may have led to the 1988 epizootic. Aside from one low-titred sample collected from a harbour seal in 1986, no antibodies against CDV were found in European harbour seals before 1988 (Osterhaus and others 1989b).

Available data indicate that there are approximately 13,000 harbour seals (Malouf 1986), 100,000 grey seals (Zwanenburg and Bowen 1990), and 2,500,000 harp seals (Roff and Bowen 1983) in eastern Canadian waters, representing a significant pool available for virus infection. Given the authors' evidence of a past PDV-1 like infection among these seals, the following questions arise: why was there no mass mortality or other signs of disease in Canada?; when and how was this virus introduced into the Canadian population?; did harp seals play a role as a vector, bridging the seal populations of Europe and North America? More extensive historical and epidemiological mapping of antibodies from seals in Greenland and North America is clearly needed to better define the origin and movement of phocine distemper virus. Differences between conditions during the infection in North America and in Europe may have led to a differential response to a similar or identical virus. One may hypothesise about lower burdens of immunosuppressive pollutants in Canadian seals, less stressful environmental conditions such as food availability or climate, differences in population density and distribution patterns, or possible genetic differences in the host seal populations or the virus itself. In the end, however, identifying the factors responsible for the severity of the disease in Europe and the apparent lack of effect in Canada may prove very difficult.

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Coronal cemental hyperplasia in a cow

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LESIONS that have been interpreted as cementomas are common in horses and have been reviewed by Miles and Grigson (1990). They are rare in other animals. Woods (1907) reported one in an ox; that specimen is also illustrated and described by Miles and Grigson. The present article describes a lesion in a cow, similar to that described by Woods.

It was detected at slaughter in an approximately three-and-a-half year-old cow and consisted of an overgrowth of cementum protruding from the lingual enamel surface of the crown of the right mandibular permanent second incisor (Fig 1). Histological examination of the demineralised specimen revealed that the cemental growth was attached to the coronal cementum which was continuous with the radicular cementum (Fig 2a, b). The cementum of the overgrowth was not laminated and exhibited numerous spaces which probably originally contained blood vessels; bone marrow was present in one area.

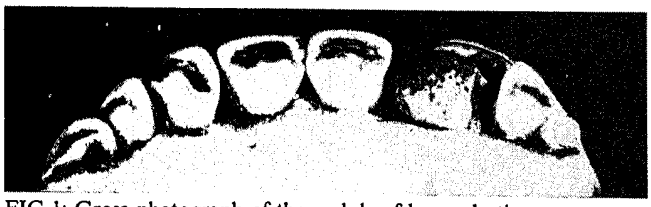

FIG 1: Gross photograph of the nodule of hyperplastic cementum

FIG 2 (a): Low power photomicrograph of the pathological section. Haematoxylin and eosin × 3

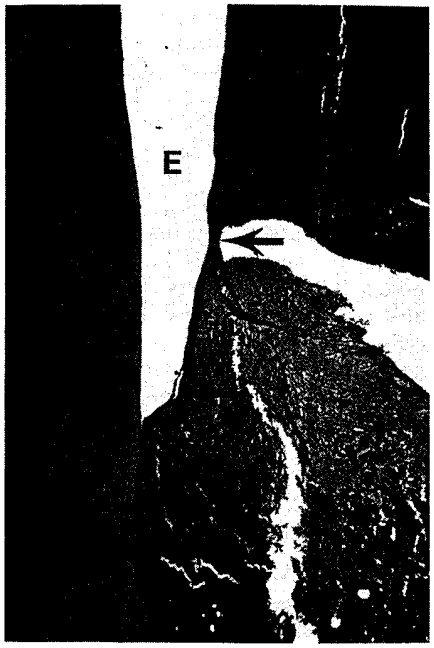

FIG 2 (b): Higher power photomicrograph of the area indicated in Fig 2a. The hyperplastic cementum (HC) is continuous with the coronal cementum (CC) which is continuous with the radicular cementum (RC). E Enamel space, D Dentin and G Gingiva. Demineralised tissue. Haematoxylin and eosin × 15

The crowns of unerupted teeth in cattle are normally covered by a thin layer of cementum. The present lesion apparently developed from this coronal cementum before the tooth erupted and represents hyperplastic cementum. Consequently, it should not be referred to as a cementoma, a term that implies neoplasia, although the so-called cementomas in human beings are probably not neoplastic either.

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Abstract

Dogs that bite

OF 146 patients bitten by dogs and referred for plastic surgery between1982 and 1989 all but three required out of hours surgery and the average length of stay was four days; 74 males and 72 females were attacked and 79 of them were less than 15 years old. Many breeds of dog were responsible, but the most commonly involved were Staffordshire bull terriers, medium-sized mongrels (particularly in attacks on children), Jack Russell terriers (particularly in attacks on adults), German shepherd dogs and German shepherd/labrador crosses; 85 per cent of the dogs were males. Most of the attacks occurred in the dog's own home and the dog and victim were familiar to one another. Four sets of circumstances accounted for most of the attacks: the victim 'invaded' the dog's territory, or was seen as a threat to the dog's family, or to the dog itself, or the dog became jealous.

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