Gross and Histopathological Findings in Unusual Lesions caused by *Streptococcus suis* in Pigs I. Cardiac Lesions

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ABSTRACT

Unusual lesions caused by *Streptococcus suis* involved the heart of 48 pigs necropsied over a four-year period. Lesions included fibrinopurulent pericarditis (six pigs), hemorrhagic, necrotizing myocarditis resembling mulberry heart disease (21 pigs), and vegetative valvular endocarditis (21 pigs). Histologically, the myocarditis was characterized by focally extensive and diffuse hemorrhages and necrosis, with infiltrations of neutrophils and mononuclear cells, and thrombosis of small and large myocardial vessels.

The 21 pigs with vegetative valvular endocarditis generally were over ten weeks old and had died suddenly. The left atrioventricular valve was affected in 18 hearts. Most of an affected valve was replaced by a layer of granulation tissue covered by a mixture of fibrin, inflammatory cells, necrotic debris and large numbers of coccoid bacteria.

Key words: Pigs, *Streptococcus suis*, cardiac lesions, pericarditis, necrotizing myocarditis, valvular endocarditis.

RÉSUMÉ

Au cours d'une période de quatre ans, l'auteur a constaté la présence de lésions inhabituelles dans le coeur de 48 des porcs dont il effectua la nécropsie. Chez six d'entre eux, il nota une péricardite fibrino-purulente; chez 21, une myocardite hémorragique et nécrotique, semblable à la cardiopathie mûriforme; chez les 21 autres, une endocardite valvulaire végétante. L'histopathologie démontra que la myocardite se caractérisait par de la nécrose, ainsi que par des hémorragies focales et extensives, ou diffuses, qu'accompagnaient une infiltration par des neutrophiles et des mononucléaires, ainsi que de la thrombose des gros et des petits vaisseaux sanguins du myocarde.

Les 21 porcs qui arboraient une endocardite valvulaire végétante avaient généralement au delà de dix semaines d'âge et étaient morts subitement. La valvule mitrale était lésée, chez 18 d'entre eux. La majeure partie d'une valve affectée était remplacée par une couche de tissu de granulation, recouverte d'un mélange de fibrine, de cellules inflammatoires, de débris nécrotiques et d'une flore luxuriante de microcoques.

Mots clés: porcs, *Streptococcus suis*, lésions cardiaques, péricardite, myocardite nécrotique, endocardite valvulaire.

INTRODUCTION

Streptococcus suis type 2 primarily causes septicemia and meningitis in weaned pigs (1,2). Reports from North America usually have depicted a broader range of diseases associated with *S. suis* type 2 in pigs (3,4,5). Seventeen serotypes of *S. suis* now have been identified (6, Henrichsen J, personal communication), but *S. suis* type 2 still is the serotype most frequently isolated from diseased pigs.

This report describes several infrequently reported or totally unreported lesions seen in pigs submitted to the Huron Park Veterinary Diagnostic Laboratory (HPVDL) with diseases caused by *S. suis* over the four-year period 1982-1985. Lesions in and around the heart are documented in this paper and those of the central nervous system in a second publication (7).

MATERIALS AND METHODS

Case records of all pigs from which *S. suis* was isolated at the HPVDL between January 1982 and December 1985 were reviewed. All cases with cardiac lesions were selected for this study. Pertinent clinical data and necropsy findings were noted and formalinized tissues embedded in paraffin blocks were sectioned at 6 μm, stained with hematoxylin and eosin and reexamined by light microscopy. Methods used at the HPVDL for identification of *S. suis* type 2 have previously been reported (3). In 1984 and 1985 slide coagglutination with serogroup 2 antiserum linked to staphylococcal protein A was used to identify *S. suis* type 2. Serotypes other than type 2 were identified as *S. suis* and not typed further.

RESULTS

The various lesions associated with the heart over the four-year period are recorded in Table I. One of the six pigs with fibrinopurulent pericarditis was one week old, one was five weeks, and four were ten to 24 weeks old. Five of the six pigs had died suddenly without premonitory clinical signs. The sixth pig was submitted alive because of malaise of several days duration which had worsened noticeably. The major gross necropsy finding was marked fibrinopurulent, pericarditis (Fig. 1). The pericardium was thickened and the pericardial sac was filled and distended with a thick, tenacious fibrinopurulent exudate. Histologically, the exudate varied quantitatively from pig to pig, consisting principally of eosinophilic proteinaceous fluid and large amounts of fibrin mixed with focally

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TABLE I. Type and Annual Occurences of Cardiac *Streptococcus suis* Lesions in 48 Pigs

Year	Fibrinopurulent Pericarditis	Myocarditis	Valvular Endocarditis
1982	1	1	1
1983	2	7	7
1984	1	8	6
1985	2	5	7
Total	6[a]	21[b]	21[c]

[a] *S.suis* type 2 was isolated from five pigs. The *S. suis* isolated from the sixth pig was not typed
[b] *S.suis* type 2 was isolated from all except two hearts. The *S.suis* isolated from those two hearts was not typed
[c] *S.suis* type 2 was isolated from all except three. The *S.suis* isolated from those three valves was not typed

was not *S. suis* type 2. Furthermore, over the four-year period, *S. suis* was cultured from all pigs in which fibrinopurulent pericarditis was the major lesion seen at necropsy.

Hemorrhagic, necrotizing myocarditis was the principal lesion in 21 pigs. Clinically and at necropsy these cases resembled mulberry heart disease (MHD). Affected pigs typically were three to five weeks old, recently weaned, and were the most robust pigs in a litter. Usually one, and seldom

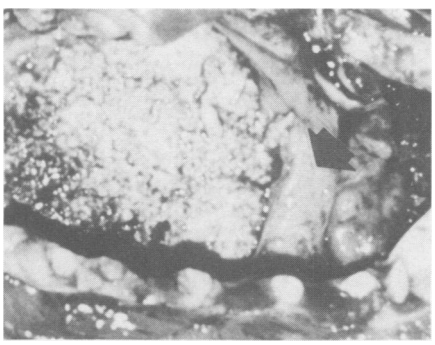

Fig. 1. Thick, tenacious, fibrinopurulent pericarditis caused by *Streptococcus suis* in a pig. Note the markedly thickened pericardium (arrow).

accentuated infiltrates of predominantly neutrophils and smaller numbers of mononuclear cells which filled the entire pericardial sac. In more chronic infections, the organized exudate consisted of large amounts of fibrin with proliferating fibrocytes and collagen which filled the pericardial sac (Fig. 2). The pericardium was thickened by collagen and mononuclear inflammatory cell infiltrates. Dense infiltrates of predominantly mononuclear inflammatory cells and a few neutrophils occupied the subpericardium for approximately the external one-quarter of the pericardial space in two hearts. Inflammatory cells were less numerous and more mixed in composition immediately beneath this. Variations and combinations of these extremes were found in other hearts. Necrotic debris, especially from degenerating pyknotic and karryorrhectic nuclei, was present in varying amounts in all pericardia. In four of the six pigs *S. suis* type 2 was isolated in pure culture, and mixed with *Actinomyces (Corynebacterium) pyogenes* in a fifth. The *S. suis* isolated from the pericardium in the sixth pig

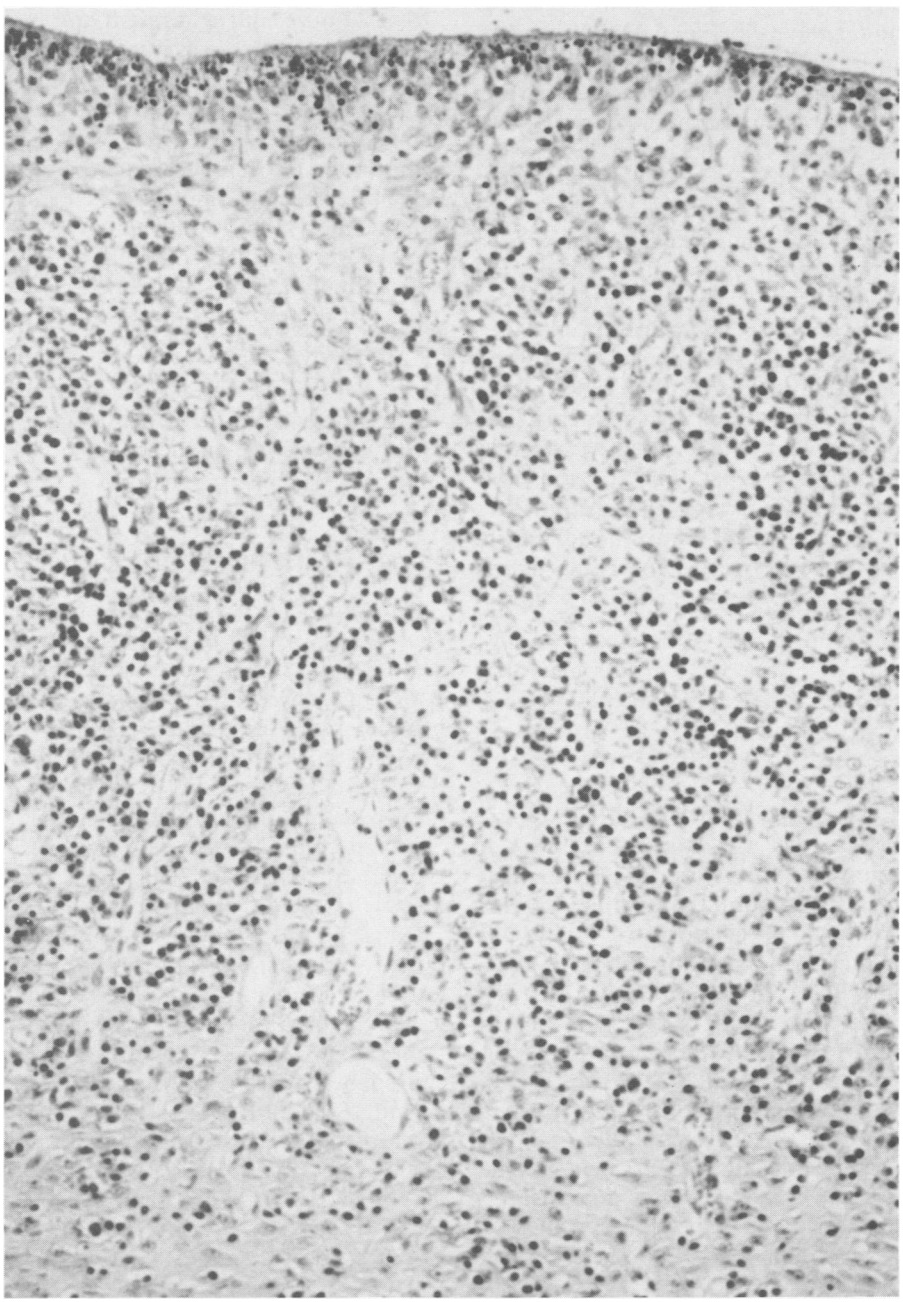

Fig. 2. Organized exudate consisting of fibrin, fibrovascularized tissue and large numbers of predominantly mononuclear inflammatory cells in pericardial sac of pig with *S. suis* fibrinopurulent pericarditis. H & E. X200.

more than three pigs from a single litter died suddenly. Heart lesions consisted of ecchymotic hemorrhages on the epicardium and throughout the ventricular and atrial myocardium (Fig. 3). The pericardial sac was filled with a large amount of serosanguineous fluid mixed with fibrin. Most of the fibrin was adhered to the epicardial surface. Lungs were congested. Smaller amounts of serosanguineous fluid mixed with strands of fibrin were present in the thoracic and, to a lesser extent, the abdominal cavities. *Streptococcus suis* type 2 was cultured from 19 hearts. The *S. suis* cultured from the remaining two hearts was not *S. suis* type 2. No significant bacteria were cultured from a further six hearts with similar histological lesions.

Focally extensive or diffuse myocardial hemorrhages and necrosis with infiltrations of neutrophils and mononuclear cells were seen histologically in atrial and ventricular myocardium (Figs. 4 and 5). Microvascular thrombosis was common and sometimes large vessels also were thrombosed. A large area of ischemic myocardial necrosis surrounded larger thrombosed vessels. Severe necrotizing vasculitis of myocardial vessels and multifocal myofiber mineralization were seen in several hearts. Small colonies of coccoid bacteria occasionally were present within vessels and the interstitium.

All but two of the 21 pigs with vegetative valvular endocarditis had died suddenly. The two submitted alive were killed *in extremis*. Although pigs with valvular endocarditis ranged

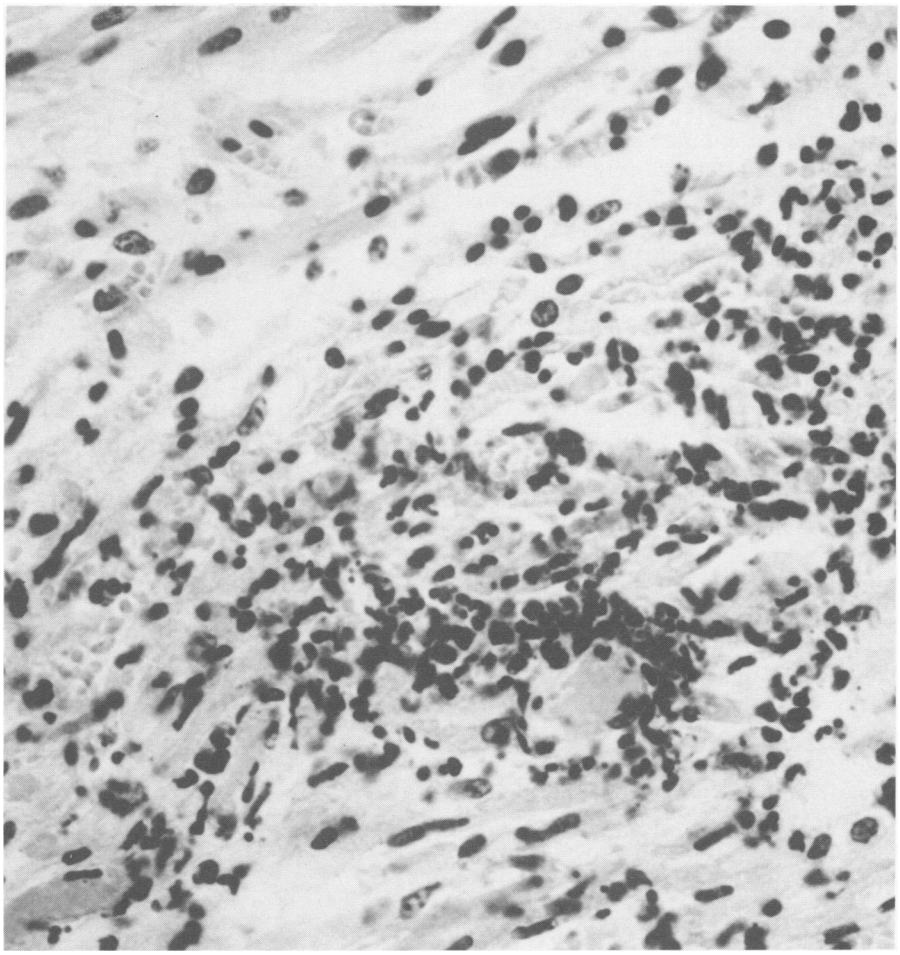

Fig. 4. Focally extensive myocardial hemorrhage and necrosis with necrotizing vasculitis (lower centre), thrombosis and large influx of neutrophils and mononuclear inflammatory cells seen in ventricular myocardium of pig with *S. suis* myocarditis. H & E. X500.

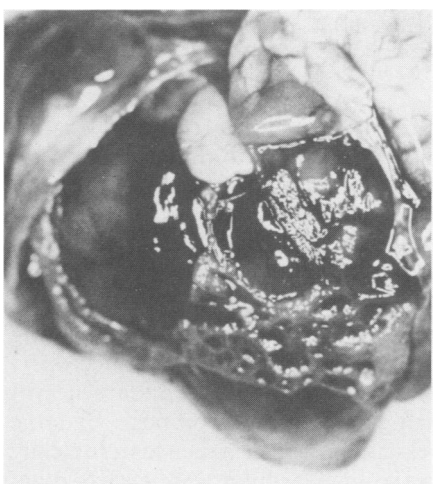

Fig. 3. Severe *S. suis* fibrinohemorrhagic myocarditis resembling mulberry heart disease. The fibrin is adhered to the epicardial surface.

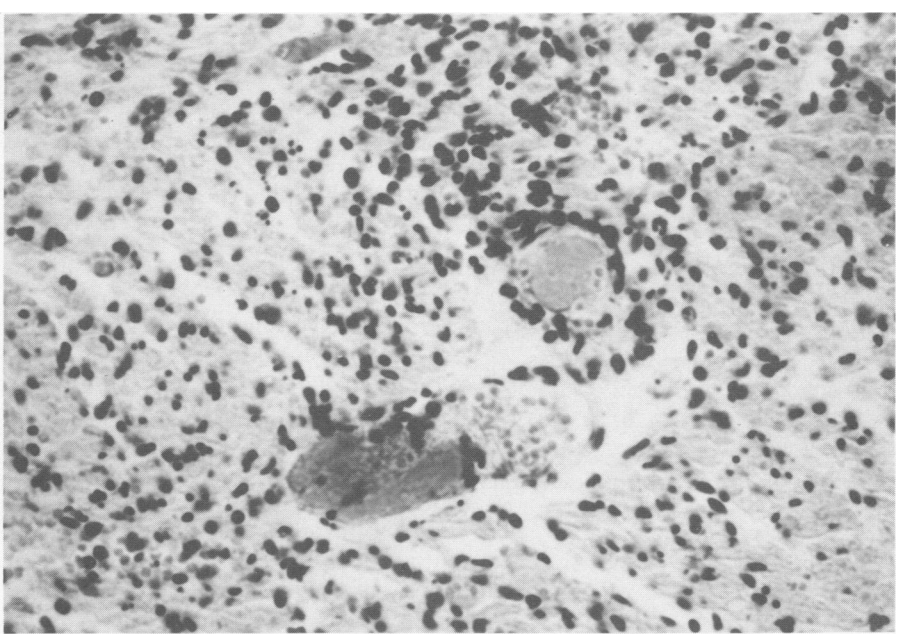

Fig. 5. Large thrombosed vessels surrounded by hemorrhage, necrosis and large numbers of neutrophils and mononuclear inflammatory cells in ventricular myocardium of pig with *S. suis* myocarditis. H & E. X250.

from five to 22 weeks of age, most (13 of 21) were over ten weeks old. The left atrioventricular (AV) valve was involved in 18 hearts, and was the only valve affected in nine hearts. The right AV valve was affected in eight pigs and the only one affected in two. The aortic valve was the sole affected valve in one pig. Combinations of two or more valves were inflamed in nine pigs. All four valves were involved in two pigs; bilateral AV valvular endocarditis occurred in two additional hearts. Affected valves typically were enveloped by dense, yellow or cream-colored proliferating vegetations of septic thrombi (Fig. 6) which sometimes extended to and involved the adjacent mural endocardium. Histologically, an affected valve was partially replaced by a layer of granulation tissue covered by a mixture of fibrin, neutrophils and mononuclear inflammatory cells, necrotic debris, mineralized foci and plaques, and large numbers of coccoid bacteria (Fig. 7). Similar inflamed areas and small perforations were present in the mural endocardium adjacent to affected valves in most hearts. *Streptococcus suis* type 2 was isolated from the valves of all but three hearts. The *S. suis* cultured from those three hearts was not typed. In the four-year period of this study, *S. suis* was isolated from all valvular endocarditis cases in non-adult pigs except two, from one of which no organism was isolated.

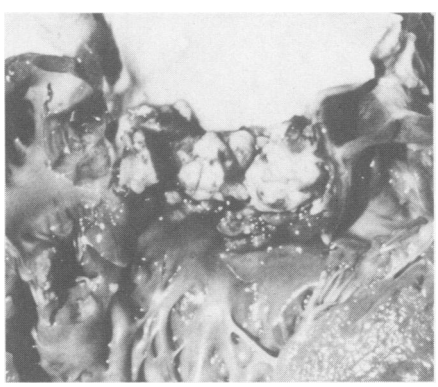

Fig. 6. *Streptococcus suis* valvular endocarditis in a pig.

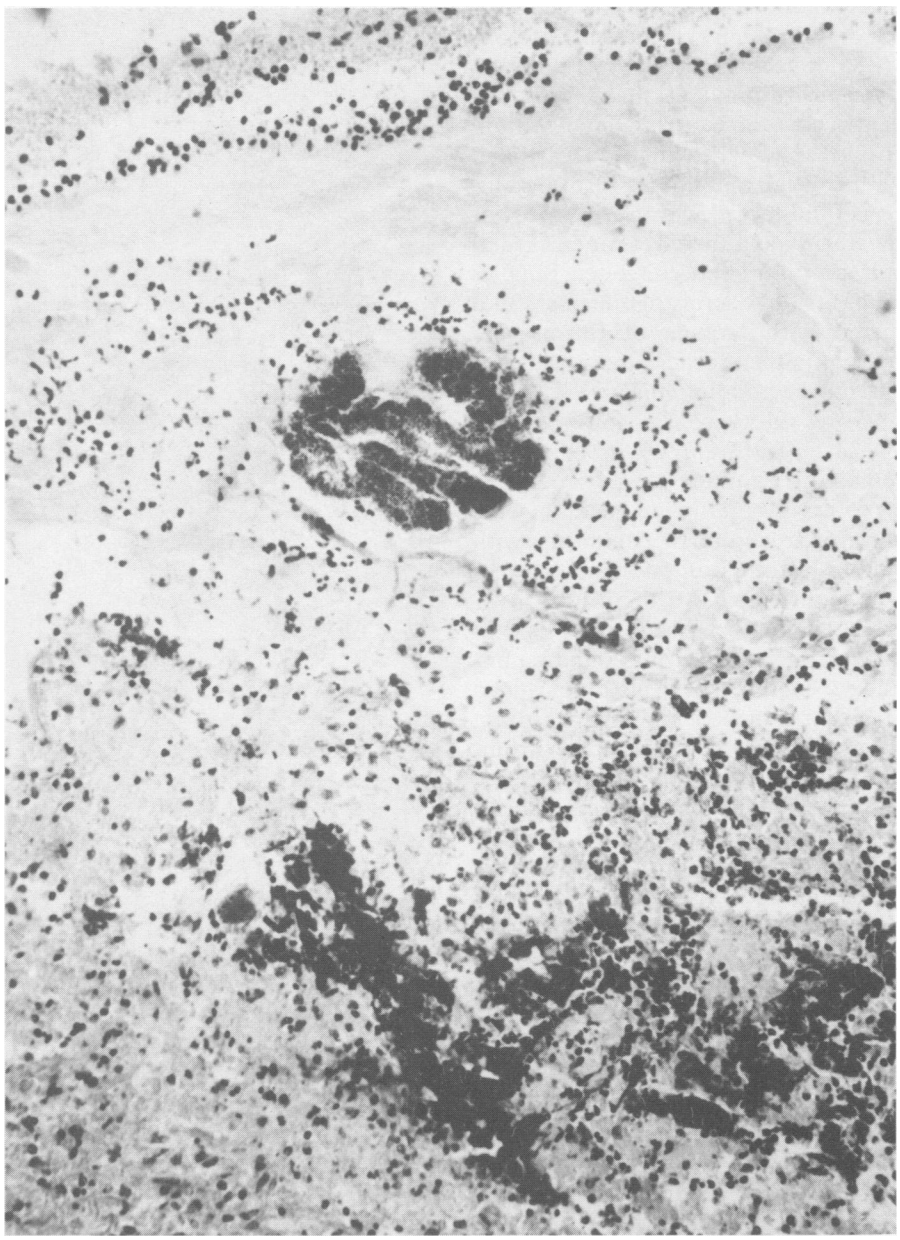

Fig. 7. Fibrinocellular exudate, a bacterial clump, necrotic debris and mineralized foci and plaques enveloping leaflet in a pig with *S. suis* valvular endocarditis. H & E. X200.

DISCUSSION

The heart as a major organ site for *S. suis* lesions has not been previously reported. The affinity of *S. suis* type 2 for serous membranes is well documented (1,4), but the singling out of the pericardium as a sole target site with the production specifically of fibrinopurulent pericarditis is not. Pigs with this lesion usually were in the high risk, postweaned age group for *S. suis* disease but were otherwise not noticeably different from other non-affected pigs, or pigs with previously reported *S. suis* lesions.

Hearts with hemorrhagic myocarditis were difficult to differentiate from MHD. Sudden death of one or two of the fastest growing pigs in a litter is clinically characteristic of MHD. The adherence of fibrin to the epicardium in pigs with hemorrhagic myocarditis was a helpful feature in differentiating between the two diseases at necropsy. The necrosis, purulent exudate, necrotizing vasculitis, thrombosis of large vessels and bacteria seen histologically in the myocardium were clearly diagnostic for myocarditis, and distinctly different from the noninflammatory myocardial degeneration of MHD.

Culture of *S. suis* was confirmatory. Jones (8) found focal and diffuse myocarditis as an accompanying lesion in seven of 15 pigs' hearts with endocarditis caused by β-hemolytic *Streptococcus*. He postulated that the myocarditis in those hearts resulted either from vascular occlusion or from direct bacterial action on the myocardium (8). Only one report of *S. suis* type 2 myocarditis was found in the literature (4). There were no indications of possible confusion with MHD in that report, suggesting that it may not have been as severe as the hemorrhagic, necrotizing lesions seen in the cases reported here.

The predilection of the left AV valve for endocarditis in pigs has been previously noted (8), although not as strongly as in this study. Valvular endocarditis was initially not included among the diseases caused by *S. suis* type 2 (1,2). However, in a 1976 publication, *S. suis* type 2 (identified as deMoor's group R *Streptococcus*), was listed as one (one case) of 125 lesions caused by various streptococci isolated from diseased pigs in the United Kingdom (9). Since that time, however, valvular endocarditis has been an increasingly frequent lesion associated with *S. suis* type 2 infection (3,4). In fact, this is now the agent most frequently isolated from valvular endocarditis cases in growing pigs in Ontario, and has been reported in Europe as well (10,11,12).

In conclusion, with the multiple lesions of: i) fibrinopurulent, pericarditis, ii) hemorrhagic necrotizing myocarditis and iii) vegetative valvular endocarditis, identified in this study, the heart should be considered another target organ of *S. suis*. In the accompanying publication, distinctive lesions of subacute meningoencephalomyelitis and segmental cortical necrosis of cerebellar folia with degeneration and drop out of Purkinje cells in antibiotic treated *S. suis* infected pigs are reported (7), further extending the range of lesions attributable to *S. suis*.

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