An Evaluation of Nonsuppurative Joint Disease in Slaughter Pigs

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Abstract

Fifty-two joints from pigs with nonsuppurative joint disease from a local abattoir were examined grossly, histologically, and microbiologically in order to establish macroscopic differences between degenerative arthropathy and arthritis due to an infectious organism. The joints were grouped grossly according to the type and severity of lesions of the synovial membrane and cartilage, and microscopically according to the severity of synovial membrane lesions. Osteochondrosis and *Erysipelothrix rhusiopathiae* were the most common causes of nonsuppurative joint disease in the joints examined. The major macroscopic differences between these two arthropathies were in the nature and severity of the synovial and cartilaginous lesions and involvement of the lymph node draining the diseased joint. Typically, in osteochondrosis, the changes are feathery hypertrophy of villi, focal full-thickness cartilage buckles, ulcers or flaps, and no change in the draining lymph node, whereas in *Erysipelothrix*-caused arthritis, the villous hypertrophy is severe and polypoid in nature, there is diffuse erosion of articular cartilage, and the draining lymph node is consistently hypertrophic and often cystic.

Key words: Porcine, polyarthritis, osteochondrosis, *Erysipelothrix rhusiopathiae.*

Résumé

Une évaluation de l'arthropathie non suppurante, chez des porcs d'abattage

Cette étude portait sur l'examen macroscopique, l'histopathologie et la bactériologie de 52 articulations de porcs envoyés à un abattoir de Saskatoon et atteints d'une condition articulaire non suppurante, afin de préciser les différences macroscopiques entre l'arthropathie dégénérative et l'arthrite septique. Les auteurs regroupèrent les articulations précitées, selon la nature et la sévérité des lésions macroscopiques de la membrane synoviale et du cartilage, ainsi que selon la gravité des lésions microscopiques de la membrane synoviale. L'ostéochondrose et l'infection par *Erysipelothrix rhusiopathiae* s'avérèrent les causes les plus fréquentes des lésions articulaires non suppurantes. Les principales différences macroscopiques entre les deux arthropathies résidaient dans la nature et la gravité des lésions synoviales et cartilagineuses, ainsi que dans l'implication du ganglion lymphatique drainant l'articulation lésée. Dans l'ostéochondrose, les changements se caractérisaient par une hypertrophie plumeuse des villosités, des foyers de bourgeonnement qui impliquaient toute l'épaisseur du cartilage, des ulcères ou des rabats cartilagineux, mais aucune lésion dans le ganglion lymphatique avoisinant. Dans l'arthrite due à *E. rhusiopathiae*, l'hypertrophie villeuse était marquée et polypoïde; elle s'accompagnait d'une érosion diffuse du cartilage articulaire et le ganglion lymphatique avoisinant était toujours hypertrophique et souvent kystique.

Mots clés: porcs, polyarthrite, ostéochondrose, *Erysipelothrix rhusiopathiae.*

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Introduction

Joint disease of swine is responsible for major losses to the agricultural community and the meat industry. Condemnations of whole carcasses due to joint disease are responsible for a significant percentage of total condemnations of swine in Canada (Table I). Nonsuppurative arthropathies may be associated with degenerative or septic processes. In swine the most common degenerative arthropathy is osteochondrosis (OC) (1-5), whereas *Erysipelothrix rhusiopathiae* is the most important infectious agent responsible for nonsuppurative joint disease in pigs throughout the world (6-17), in spite of extensive vaccination programs. At slaughter the veterinary inspector must differentiate rapidly between these arthropathies during postmortem examination. This aspect of inspection is particularly important because of the zoonotic significance *E. rhusiopathiae* (15, 18).

The purpose of this study was to relate histological lesions and cultural results to gross changes in condemned joints in order to establish macroscopic criteria for identifying and differentiating the major conditions associated with nonsuppurative polyarthropathies and to ensure reliable disposition of carcasses affected with polyarthropathy.

Materials and Methods

Fifty-two joints selected by a veterinary inspector at an abattoir in Saskatoon formed the basis of this study. The joints were removed from carcasses judged to have nonsuppurative polyarthropathy. Joints removed from four clinically normal pigs from an unrelated experimental study served as controls. In all cases, pigs were killed by electrocution and exsanguination.

One joint of one limb from each diseased animal was opened at the packing plant to confirm the diagnosis of nonsuppurative joint disease. The contralateral limb was removed with joints unopened, placed in a plastic bag and examined at the Western College of Veterinary Medicine within five hours of removal. In the case of the pelvic limb, if any abnormality in the external iliac lymph node was found, the node was included with the limb. The axillary lymph node of the thoracic limb is not routinely inspected.

Depending on the limb, either the elbow or the stifle joint was examined in this investigation as these joints are most accessible to the inspector on the production line and are most commonly used when making decisions on disposition of carcasses with polyarthropathy. The skin over the intact joint was cleansed with a surgical iodine scrub (Betadine surgical, Purdue Frederick, Inc., Toronto) followed by a rinse with 70% isopropyl alcohol.

Joint fluid was aspirated percutaneously with an 18 g needle into a sterile 10 mL syringe. The volume and characteristics of fluid in the joint were recorded. Fluid was retained for culture.

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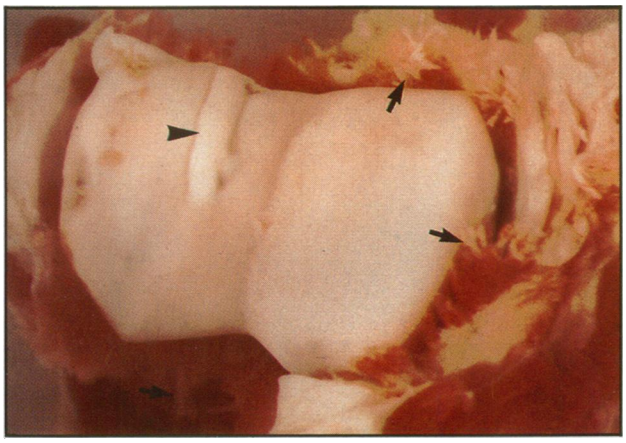

Figure 1. Distal humeral articular surface from a pig with osteochondrosis. Photo taken under water to demonstrate fine, feathery hypertrophy of synovial villi (arrows). Note buckling of the articular cartilage (arrowhead).

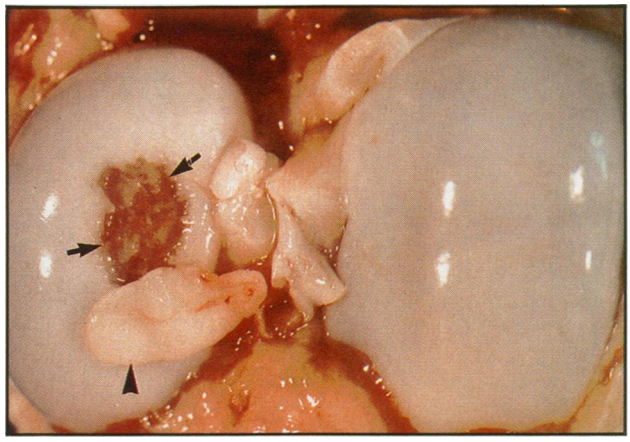

Figure 2. Distal femoral articular surface demonstrating a full thickness cartilage ulcer with sharp borders in a pig with osteochondrosis (arrows). The detached cartilage forms a joint mouse (arrowhead). Note the smooth, shiny appearance of the remaining articular surface.

The joint was then opened and any remaining joint fluid was collected in the process and added to the measured volume. A portion of this fluid was tested with a Chemstrip (Chemstrip 9, Boehringer Mannheim, Dorval, Quebec) reagent strip to assess leukocyte content, pH, protein, and blood.

The synovial membrane and articular cartilage were examined macroscopically and classified according to the presence or absence of lesions (Table II). Any changes in the draining lymph node, if available, were recorded. All joint surfaces and lymph nodes were then photographed.

Samples of synovial membrane were placed in 10% buffered formalin for 24 hours. Tissues were sectioned and processed routinely, stained with hematoxylin and eosin, and examined under a light microscope. The presence or absence of histopathological changes was used to classify the joints according to the number of lesions present (Table III).

Aseptically collected synovial fluid was placed in a 10 × 75 mm glass tube and centrifuged at 1200 g for 20 min in a Clay-Adams centrifuge (Sero-fuge II, Becton, Dickinson and Co., Parsippany, New Jersey 07054). The supernatant fluid was withdrawn and the residual plug plated directly on blood and MacConkey agar, blood agar in a CO_2 atmosphere for *Haemophilus* spp., blood agar in an anaerobic atmosphere, and inoculated into a brain heart infusion broth (BHI). Subcultures were made from the BHI broth at 24 h. Primary and subcultures were incubated for up to 48 h at room temperature. Suspect colonies were Gram-stained and inoculated into triple sugar iron and gelatin for confirmation of *E. rhusiopathiae*. Standard Yeastolate Agar (YA) plates and Horse Serum (HS) broth were inoculated for *Mycoplasma* spp.

A diagnosis of osteochondrosis was based on the presence of sterile synovial fluid and a focal disruption of the articular cartilage at the chondro-osseous junction where a full-thickness cartilaginous buckle, flap, or ulcer was formed.

Joints from which an organism was cultured were grouped and further categorized according to etiology.

If no organism was cultured from the synovial fluid and there were no osteochondrotic cartilaginous lesions, the joint was classified as having no diagnosis. These joints were grouped further according to gross and histopathological changes (Tables II and III).

Results

The gross pathological changes observed in the 52 joints removed from carcasses affected with polyarthropathy are summarized (Table II).

The diagnosis of OC was made for 21 joints with the characteristic macroscopic features of one or more focal full-thickness cartilaginous buckles, ulcers or flaps, mild, feathery hypertrophy of the synovial villi, and the absence of pathological changes in the regional lymph node (Figures 1 and 2).

The macroscopic features of the 18 joints from which *E. rhusiopathiae* was cultured were diffuse, partial thickness erosion of the articular cartilage, often with pannus formation and fibrillation, moderate to severe polypoid hypertrophy of the synovial villi and severe lymphoid hyperplasia, congestion and often cyst formation in the regional lymph node (Figures 3-5). Five joints which were sterile and one joint which was positive for *Streptococcus equisimilis* had similar macroscopic changes. The only gross pathological change

TABLE I
Condemnations for Arthritis in Swine[a]

Fiscal Year	Total Number of Swine Slaughtered	Total Number of Carcass Condemnations	Rate/1000 Slaughtered	Carcasses Condemned for Arthritis	Rate/1000 Slaughtered	Percentage of Total Condemnations	Portions Condemned for Arthritis	Rate/1000 Slaughtered
1969-70	7,168,044	35,826	4.998	8,761	1.222	24.5	49,928	6.965
1975-76	7,387,198	47,309	6.404	11,189	1.515	23.7	59,762	8.090
1982-83	12,695,185	57,223	4.507	16,562	1.345	28.9	119,985	9.451
1983-84	13,146,387	53,497	4.069	15,784	1.201	29.5	139,339	10.559
1984-85	13,138,237	53,210	4.050	16,136	1.228	30.3	119,880	6.096

[a]Agriculture Canada Statistics

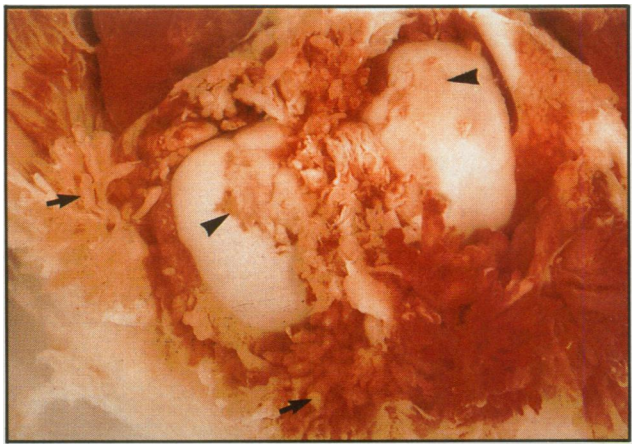

Figure 3. Distal femoral articular surface from a pig with arthritis caused by **Erysipelothrix rhusiopathiae.** *Photo taken under water to demonstrate the severely hypertrophied, elongate polypoid synovial villi (arrows). Note erosion of articular surfaces (arrowheads).*

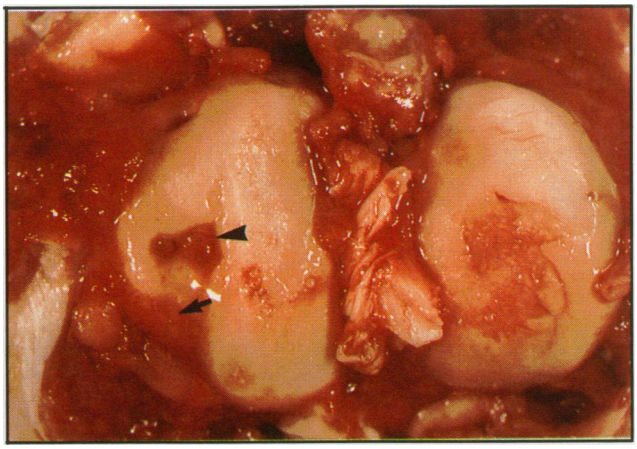

Figure 4. Distal femoral articular surface showing diffuse erosion of the articular cartilage in a pig with arthritis caused by **Erysipelothrix rhusiopathiae.** *Pannus encroaches from the periphery of a condyle (arrow) and from the subchondral area (arrowhead).*

noted in five culturally sterile joints and one yielding *Mycoplasma hyosynoviae* was very mild hypertrophy of synovial villi. The second joint from which *Streptococcus equisimilis* was cultured had only mild, polypoid hypertrophy of synovial villi.

The Chemstrip test strip from all joints examined indicated a pH of 8, a protein value of 500 mg/dL, 250 erythrocytes/μL of blood, and leukocyte values varying from 10-75 WBC/μL. There was wide variation in appearance and volume of synovial fluid but it was generally increased in volume and serosanguineous, and contained clumps and strands of fibrin (Figure 6).

The histological changes (Table III) were not as specific as the gross lesions.

Five joints affected with OC, and five which were culturally sterile and had mild feathery synovial hypertrophy grossly, had no histological change. Ten osteochondrotic joints had only fibrin overlying the synovial lining, whereas six had fibrin and perivascular lymphoid nodules. The infiltration of plasma cells and lymphocytes was mild ($\leq$ 10 cells per high power field) (Figure 7).

The synovia of joints from which a pathogenic agent was cultured, and of five which had severe macroscopic changes but were culturally sterile, had consistently more severe histological changes than those described previously. The inflammatory infiltrate was most often severe (10-100 cells/high power field) and there were always perivascular lymphoid nodules present (Figure 8). In 14 of 23 of these joints there was fibrinoid necrosis of the tips of the large, polypoid villi (Figure 9). Histologically, the cysts in the lymph nodes consisted of dilated medullary sinusoids, having no wall or lining membrane. These dilated spaces were filled with pale, eosinophilic fibrillar material.

Giant cells and cartilaginous fragments were seen occasionally in the synovial lining of the joints examined but were not diagnostic for any one type of joint disease (Figures 10 and 11).

Erysipelothrix rhusiopathiae was the cause of severe arthritis in 18 of 52 porcine joints in this study. The major gross pathological differences between these joints and those with lesions of osteochondrosis are summarized (Table IV).

Discussion

The two major causes of nonsuppurative polyarthropathy in swine in this study were OC and *E. rhusiopathiae.*

Ostechondrosis is a generalized disease with high morbidity and is characterized by focal disruption of normal differentiation of chondrocytes in a metaphyseal or articular growth plate (5, 19). The earliest lesions consist of foci of hypertrophied chondrocytes in which maturation is arrested concurrent with failure of subchondral vascular penetration and provisional calcification of the matrix resulting in retention of cartilage (1). It is not known if the primary defect is in the vasculature or in the chondrocytes (20). Derangement of vascular supply results in impairment of the transportation of metabolites to and from the chondrocytes and interference with their ability to secrete and maintain matrix (21). In turn, these events predispose to structural weakness in the cartilage (1, 22). Degeneration of the deepest layers, aided by shearing forces generated through the chondro-osseous interface consequent to faulty conformation and joint instability, results in the formation of fibrin-filled cleft-like structures subjacent to the developing articular cartilage (23, 24). This results eventually in buckling of the cartilage. Fissures extending to the surface give rise to the flap-like lesions termed osteochondrosis dissecans (22).

Inflammation of the synovial membrane may be induced by substances released from damaged articular cartilage. Breakdown of cartilage exposes chondromucoproteins, collagen, and enzymes not normally in contact with the environment outside the avascular cartilage. Type II collagen of hyaline cartilage, in particular, is known to be immunogenic, initiating an inflammatory reaction in the synovial membrane (25).

The histological changes in the synovia of osteochondrotic swine in this study (hemosiderin-laden macrophages and a mild infiltration of plasma cells with a few perivascular lymphoid aggregates) agree with those described in horses (26).

Thus, the lesion in OC is initiated focally at the chondro-osseous junction and results in full-thickness cartilage buckles or flaps surrounded by normal cartilage. The articular surface otherwise is smooth, and synovitis is mild and secondary to cartilaginous lesions and joint instability.

In infectious arthritis, organisms arrive in the synovial membrane during bacteremia (27) and incite both a humoral and a cell-mediated immunological response. Antigen is pre-

TABLE II
Gross Pathological Features of Diseased Swine Joints

	Synovial Fluid			Synovial Villi				Articular Cartilage				
					Characteristics							
Number of Cases and Diagnosis	Volume (mL)	Characteristics	Colour	Degree of Hypertrophy	Feathery	Polyroid	NC*	Erosion	Buckle	Ulcer or Flap	NC	Hyperplasia of Draining Lymph Node
21 OC[a]	6 (1-35)	serosanguineous with fibrin occasionally tan and serous	tan ocassionally red	very mild to mild	+	−	−	−	+(10)	+(19)	−	−
18 ER[b] 5 ND[c] 1 SE[d]	4.5 (0-38)	serosanguineous with fabrin occasionally cloudy	red	moderate to severe	−	+	−	+ often with pannus and fibrillation	−	−	−	+ often cystic
5 ND 1 MH[e]	4 (2-14)	tan, mucinous occasionally clotted occasionally fibrin	tan	very mild	−	−	+	−	−	−	+	−
1 SE	1.5	serous cloudy clumped fibrin	tan	mild	−	+	−	−	−	−	+	−

[a]OC = osteochondrosis [b]ER = *Erysipelothrix rhusiopathiae* [c]ND = no diagnosis [d]SE = *Streptococcus equisimilis*
[e]MH = *Mycoplasma hyosynoviae* * No change

sented to helper/inducer T4 cells which become activated and interact with B cells which undergo transformation to antibody-secreting plasma cells. The combination of the organism with its specific antibody activates the complement sequence, generating a variety of biologically active materials, some with potent chemotactic properties. These substances attract polymorphonuclear leukocytes (PMNs) into the joint to ingest the complexes, with release from lysosomal vacuoles of a variety of hydrolytic enzymes capable of degrading the chondromucoprotein matrix and collagen fibrils of articular cartilage (28-31).

Reaction of T lymphocytes with bacterial antigen causes release of an assortment of lymphokines including lymphocyte blastogenic factor, lymphotoxin, migration inhibition factor and macrophage-activation factor. Interleukin 1 is released from activated macrophages in the synovial membrane and from type A synovial lining cells, stimulating the release of collagenase from fibroblastic synovial cells. Bacterial collagenases readily destroy collagen as well. The proteoglycan matrix of the cartilage is digested by cathepsins from the lysosomes of PMNs and type A synovial lining cells (32).

Thus, in septic arthritis, synovitis is the initial lesion. Immunological reactions within the synovium and joint space cause an influx of inflammatory cells with consequent release of enzymes responsible for the degradation of cartilaginous components. Destruction of cartilage begins at the articular surface with diffuse erosions, fibrillation, and pannus formation.

Experimental induction of chronic arthritis with *E. rhusiopathiae* is well

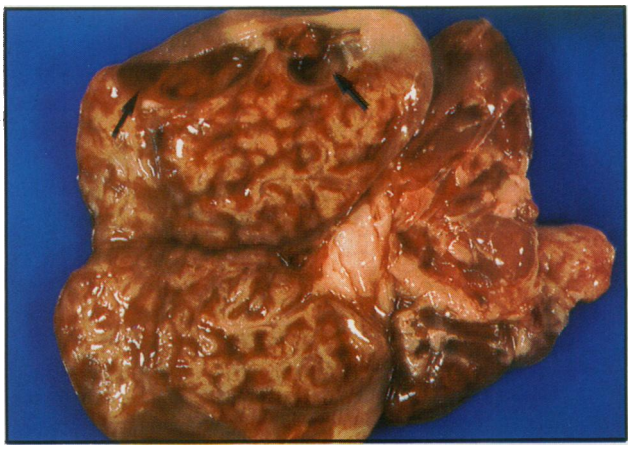

Figure 5. Bisected enlarged external iliac lymph node showing congestion and lymphoid hyperplasia with cyst formation in a pig with arthritis caused by* Erysipelothrix rhusiopathiae *(arrows).

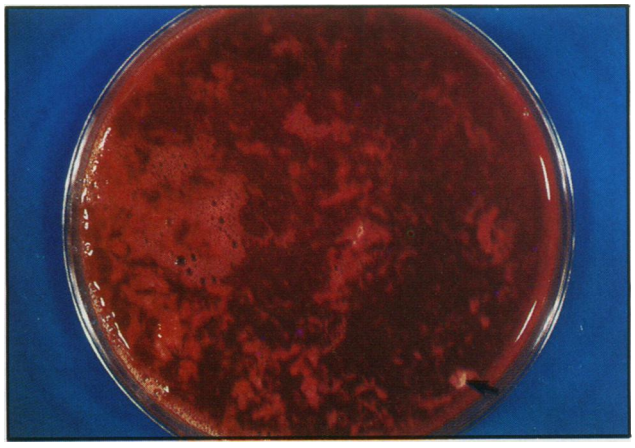

Figure 6. Characteristic serosanguineous appearance of synovial fluid from joint of a pig with either osteochondrosis or arthritis caused by* Erysipelothrix rhusiopathiae. *Note the fibrin present (arrow).

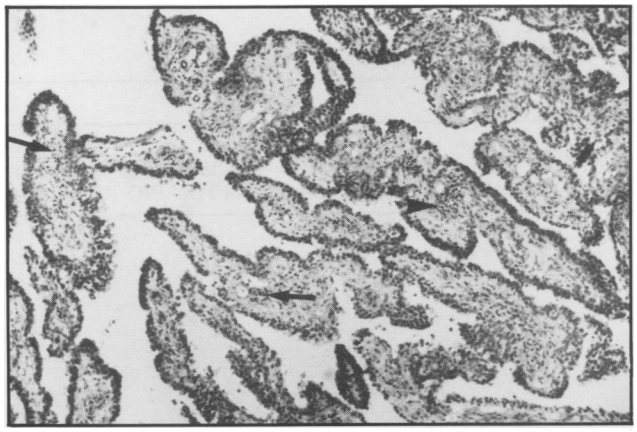

Figure 7. Synovial villi from a pig with osteochondrosis. Villi are slim with very mild infiltration of plasma cells (arrows) and a few perivascular lymphoid aggregates (arrowhead). H&E.

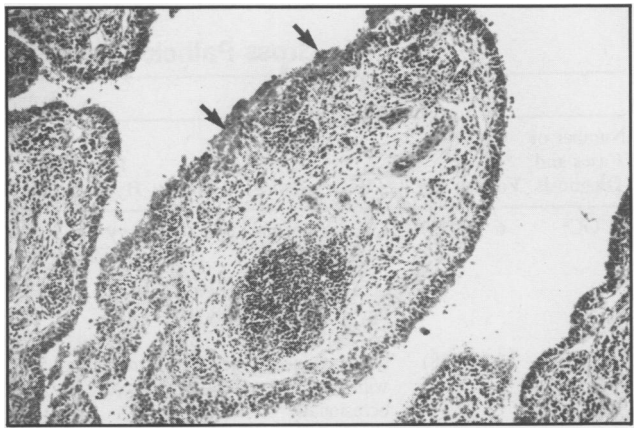

Figure 8. Synovial villi from a pig with athritis caused by **Erysipelothrix rhusiopathiae.** *Villi are plump with moderate infiltration of plasma cells and a very prominent aggregate of perivascular lymphocytese. Fibrin overlies the synovial lining (arrows). H&E.*

documented (33-35). The gross and histopathological changes in the joints in this study are in agreement with those of previous reports (36-38). Cysts have been described in the lymph nodes draining chronically affected joints (37). The cause of these structures is unknown but they might result from partial blockage of efferent lymph channels due to fibrosis resulting from chronic inflammation within the node.

Many workers have experienced difficulty in culturing the *Erysipelothrix* organism from arthritic joints following experimental infection of swine, an observation that has given rise to the suggestion that there is a hypersensitivity component to the lesion (39-42). Culture of small numbers of *E. rhusiopathiae* from the cellular residue from 18 of 23 (78%) of joints with typical gross lesions in this study suggested that centrifugation of joint fluid to concentrate organisms may be helpful in making a microbiological diagnosis of infection caused by *E. rhusiopathiae.*

Streptococcus equisimilis was cultured from one joint with chronic arthritis and one joint with much milder changes in this study. *Streptococcus* spp. are regarded as the second most common cause of arthritis in pigs (15, 43, 44).

Mycoplasma hyosynoviae causes mild nonsuppurative arthritis with an increased volume of serofibrinous synovial fluid and no cartilaginous change (9, 13, 45) which is consistent with the lesions in the joint from which this organism was isolated in this study.

The five joints from which no organism was cultured, and in which the only pathological change was an increase in volume of synovial fluid, may have been normal joints with a mild reaction to the trauma induced during transit of the hogs.

Testing of synovial fluid with Chemstrip reagent strips was not useful in differentiating septic from degenerative joint disease as the leukocyte and RBC values as well as the pH and protein content were similar in all joints tested.

The results of this study indicated that using the criteria outlined in Table IV, the veterinary inspector, on macroscopic examination of the articular cartilage, synovial membrane, and draining lymph node of the stifle or elbow joint of pigs with polyarthropathy may distinguish with confidence, osteochondrosis and arthritis caused by *E. rhusiopathiae*, assuring correct disposition of carcasses affected with polyarthropathy. Examination of the draining lymph node of limbs with joint disease should become an integral part of postmortem examination.

TABLE III
Histopathological Features of Synovia of Diseased Swine Joints

Number of Cases and Diagnosis	Number of Inflammatory Cells*	Number of Layers of Synovial Lining Cells	Overlying Fibrin	Perivascular Lymphoid Nodules	Macrophages with Hemosiderin	Necrotic Villous Tips
4 control	0	0-2	−	−	−	−
5 OC[a]	0	0-2	−	−	−	−
5 ND[b]	0	0-2	−	−	−	−
10 OC	0-1	0-3	+	−	−	−
6 OC	1	0-2	+	+	−	−
5 ER[c]	2-3	0-2	+	+	−	−
1 MH[d]	1†	0-1	+	+	−	−
1 SE[e]	2†	0-1	+	+	−	−
2 ND	3	0-3	+	+	−	−
2 ER	1-2	0-2	+	+	+	−
1 ND	2	0-2	+	+	+	−
10 ER	1-3	0-3	+	+	+	+
3 ND	2-3	0-3	+	+	+	+
1 SE	3	0-2	+	+	+	+

*0 = 1 - 10/higher power field
1 = 11 - 40/higher power field
2 = 41 - 70/higher power field
3 = 71-100/higher power field
predominantly lymphocytic/plasmacytic infiltrate
† − predominantly neutrophils

[a]OC = osteochondrosis
[b]ND = no diagnosis
[c]ER = *Erysipelothrix rhusiopathiae*
[d]MH = *Mycoplasma hyosynoviae*
[e]SE = *Streptococcus equisimilis*

Acknowledgments

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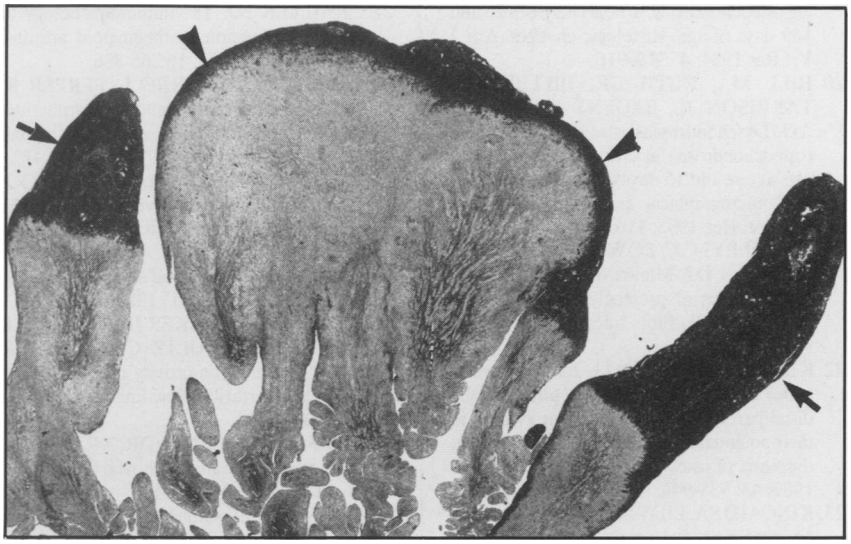

Figure 9. Synovial villi showing villous hypertrophy and fibrinoid necrosis of the tips of the large polypoid villi in a pig with arthritis caused by* Erysipelothrix rhusiopathiae *(arrows). There is fusion of villi (arrowheads). PTAH.

TABLE IV
A Comparison Between Macroscopic Changes in Joints and Regional Lymph Nodes with Osteochondrosis and *Erysipelothrix rhusiopathiae*-Induced Arthritis

Gross Pathological Characteristic	Osteochondrosis	*Erysipelothrix rhusiopathiae* Arthritis
Cartilaginous lesion	Full thickness, focal articular cartilage buckle, ulcer or flap	Partial thickness, diffuse articular erosion ± fibrillation and pannus formation
Synovial membrane	Mild, feathery villous hypertrophy	Moderate to severe, polypoid villous hypertrophy
Draining lymph node	No change	Severe hypertrophy with lymphoid hyperplasia, congestion and often cyst formation

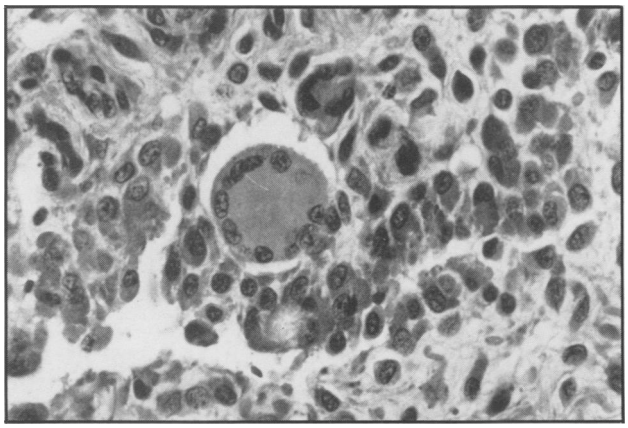

Figure 10. Giant cell within the synovial lining of a pig with arthritis caused by* Erysipelothrix rhusiopathiae. *H&E.

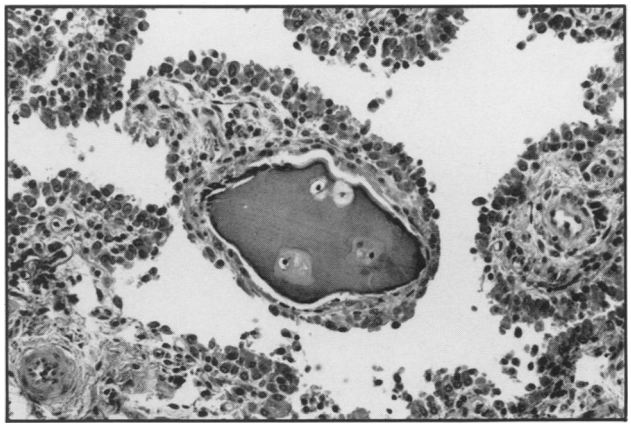

Figure 11. Cartilage fragment surrounded by synovium in a villus from a pig with osteochondrosis. H&E.

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