PULMONARY LESIONS IN SWINE IV: MICROBIOLOGIC AND PATHOLOGIC DIAGNOSES IN SWINE ENZOOTIC PNEUMONIA (SEP) IN THE STATE OF MINAS GERAIS, BRAZIL. R.H.G. NOGUEIRA*; M. REZENDE, R. REIS, E.F. NASCIMENTO and M.A.G. CHQUILOFF. VETERINARY SCHOOL, FEDERAL UNIVERSITY OF MINAS GERAIS, BRAZIL. BOX 567.

Among the respiratory infections occuring in swine is swine enzootic pneumonia (SEP) characterized by a chronic course and accompanied by a persistent cough and retarded growth. Although the morbidity high the mortality is low. The etiologic agent of SEP, Mycoplasma hyopneumoniae, was simultaneously reported in the United States of America (Mare and Switzer, 1965) and in Great Britain (Goodwin et all, 1965), and later reported in Canada (L'Ecuyer, 1969), and Japan (Takatori, 1969). The pulmonary lesions consist of well demarcated areas of consolidation varying from plumred to greyish in color situated usually in the antero-ventral areas of the apical and cardiac lobes. Microscopic examination revealed pronounced peribronchiolar and perivascular infiltrates of lymphocytes and thickened alveolar septa owing to proliferation of the septal elements. Other alterations reported were edema in varying degrees and frank obliteration of large areas lymphocytic infiltration (Hodges et all, 1969; Pattison, 1956).

Although studies of the mycoplasmas are rather scanty in Brazil, this disease is considered common based on clinico-anatomic studies. As early as 1939 reports fom São Paulo (Bueno, 1939a; Bueno, 1939b), described pulmonary lesions compatible with those now recognized as the result of SEP. More recently (1974), lesions of SEP were seen in swine from the 3razilian States of Minas Gerais and Rio Grande do Sul (Nogueira et all, 1974; Williams et all, 1977). Based on such evidence as failure to gain body weight, poor feed efficiency, clinical and anatomical studies, and isolation of M. hyopneumoniae we propose that SEP occurs in Brazil.

A total of \$40 swine of various breeds, usually about 6 months of age, were examined in abattoirs of the States of Minas Gerais, São Paulo (Ribeirão Preto) and Parana (Ponta Grossa). Of these, 143 lungs with lesions suggestive of SEP, were selected. Isolation of the organism was done according to the method of Friis (Friis, 1974). Blood agar plates were employed to identify other bacteria. Histologic studies were done on representative sections of affected lungs. Fixation was in 10% buffered formalin, embedding was in paraffin, and hematoxylin eosin was the principal stain.

Gross pathology. In the 840 lungs examined, lesions of pneumonia were noted in 143. These were generally confined to the antero-ventral areas, i.e., apical and cardiac lobes. The dark red to gray lesions were striking that they were sharply delineated from the normal tissue. The bronchioles contained a yellow exudate. The occurrence of pneumonic lesions in swine is not uncommon in our experience, a fact noted by Gordon (1963). Furthermore, the conclusion that SEP was present was expected, as predicted by Switzer (1967) who velieved the disease is world-wide. Histopathology. The histopathologic picture was one of peribronchiolar and perivascular lymphocytic infiltrates with septal thickening owing to lymphocytes and macrophages. In some instances the lymphoid hyperplasia reached a point where it caused compression of the bronchial structures and subsequent obstruction

In addition, edema frequently appeared and was invariably accompanied by round cell types which tended to from a cuffing around the bronchi and bronchioli. The alveoli usually contained aggregates of lymphocytes, lesser numbers of macrophages, and desquamated alveolar epithelial cells. Neutrophils were rarely seen and we felt they were the result of secondary invading bacteria. In some animals there was definite proliferative fibrosis circumscribing the bronchioles, a situation we felt developed only in chronic cases. Frank intra-alveolar and intra-bronchiolar exudate was striking in some of our cases. Bacteriology. Mycoplasma hyopneumoniae was isolated from 31 of the 143 lungs with lesions suggestive of SEP. In two lungs the organism was obtained in pure culture, and in the remaining 29 was found in conjunction with M. hyorhinis (13 animals), Haemophilus sp (10 nyorninis (13 animals), nacemphilities of animals), Pasteurella sp (2 animals), Streptococcus sp (2 animals), and Corynebacterium sp (2 animals). While M. hyopneumoniae is considered to be the etiologic agent of (Goodwin, et all, 1968; L'Ecuyer, 1969, Mare and Switzer, 1965), M. hyorhinis may or may not be important as it has been observed in apparently normal tissues (Estola and Schulman, 1966; Goodwin et all, 1968; Harris et all, 1969; Hartwich and Muller, 1966; L'Ecuyer et all, 1961; Switzer (1955). The lesions observed in swine in Brazil were identical to these reported in the literature (Hodges et all, 1969; Konno et all, 1967; Morales and Roberts, 1969; Nogueira et all, 1974; Pattison, 1956). In fact, similarity was almost in complete accord except for the observation (Roberts et all, 1962) that Pasteurella multocida was the principal contaminat. In Brazil, Haemophilus sp seems to fill that role.

REFERENCES

Estola, T. and S. Schulman, Acta Path Microbiol. Scand.66:113-123, 1966. Friis, N.F. Royal Vet. and Agric. University, Copenhagen, pp. 162. 1974.; Goodwin, R.F.W., A.P. Pomeroy and P. Whittlestone, Vet.Rec. 77:1247-1249, 1965.; Goodwin, R.F.W., A. P. Pomeroy and P. Whittlestone, J.Hyg (London) 66:595-603, 1968. Gordon, W.A.M. Br.Vet.J. 119:307-514, 1963.; Harris, D.L., R.F. Ross and W.P. Switzer, Amer.J.Vet.Res., 30:1621-1624, 1969. Hartwich, J. and U. Muller. Ticrarztl. Umschau 21:103-108, 1966.; Hodges, R.T., A.O. Betts and A.R. Jennings. Vet.Rec. 34:263-273, 1969. L'Ecuyer C., W.P.; Switzer and E.D. Roberts. Amer.J. Vet.Res. 22:1020-1025, 1961. L'Ecuyer, C. Can J.Comp.Med. 33:10-19, 1969; Mare, C.J. and W.P. Switzer, Vet.Med. 60:941-946, 1965. Pattison, I.H. Vet.Rec. 68:490-494, 1956. Roberts, E.D.; W.P. Switzer and C. L'Ecuyer Cornell Vet., 52:306-327. Switzer, W.P. Amer. J.Vet.Res. 16:540-544. 1955. Switzer, W.P. Amer. Vet.Med.Assoc. 151:1656-1661. 1967. Takatori, I. Natl.Inst.Animal Health Quart. (Tokyo) 9:183-184. 1969. Williams, H.A., I.A. Piffer and D.E.S.N. Barcellos. Bol. Inst. Pesq.Vet. 4:87-95. 1977.