RADIOGRAPHIC STUDIES ON THE PATHOGENESIS OF THE WASTING IN PIGS INFECTED WITH HEMAGGLUTINATING ENCEPHALOMYELITIS VIRUS (HEV) .

K. Andries*, M. Pensaert* and F. Verschooten**
*Laboratory of Virology, **Department of Surgery,
Faculty of Veterinary Medicine, University of Gent, B-9000 Gent, Belgium.

Upon experimental inoculation of neonatal pigs with HEV, the affected animals develop either an acute or chronic form of illness. Listlessness, inappetence and vomiting are clinical signs common to both forms. Additional signs of the acute form may be paddling, muscle tremors and hyperesthesia. In the chronic form, the pigs become stunted and emaciated, and usually die of starvation after a few weeks. (1)

Immunofluorescent studies on pigs killed within three days after the appearance of clinical signs showed that viral multiplication also occurred in the intrinsic (neurons of the submucosal and myenteric plexuses) and the extrinsic (neurons of the solar ganglion, the distal vagal ganglion and the vagal nuclei in the brainstem) neural innervation of the stomach and the small intestine.(2) It was the purpose of the present experiment to examine whether the gastro-intestinal transit of a barium meal in pigs which survived the first days of illness was altered compared to the transit in uninfected control pigs. The effect of a total denervation of the parasympathic extrinsic control of the gastro-intestinal tract was studied in vagotomized pigs.

METHODS. Three colostrum deprived pigs were inoculated oronasally at the age of 5 to 7 days using 2 x $10^{6.8}$ $TCID_{50}$ of the VW 572 isolate of HEV earlier described.(2) They became ill after an incubation period of 5 to 6 days. Radiographs were taken 6 to 10 days after the appearance of the clinical signs. Three pigs of the same litter served as non-inoculated controls. In a further three animals, a truncular bilateral vagotomy at the level of the diafragm was done through an abdominal approach. Radiographs were taken 5 to 13 days after the operation. In two control pigs, only a laparatomy was done.

The pigs were reared in Horsfall type units and fed twice daily using bovine milk. For radiography, 30 ml of barium sulphate was brought into the stomach lumen via a gastric tube, because of the anorexia in these animals.

The radiographs were taken at different time intervals after the barium uptake (1/2 h, 5,8,11,14,17,20, 28, 36 etc..) using Medichrome film with a standard screen at a distance of 100 cm, exposure being 60 kV and 7 mAs (0.02 sec.). Lateral views only were taken.

RESULTS. In the five control pigs, the gastric emptying time varied from 5 to 8 hours. The complete transfer of the test meal to the small intestine was not delayed after laparatomy. The contrast medium entered the small intestine within 30 minutes after the administration and reached the large intestine within 8 hours.

In pigs with vagotomy, the stomach was dilated and the stomach emptying time varied from 20 to 35 hours. Movement of gastric contents to the small intestine occurred also within minutes after eating the barium meal, and the head of the meal reached the proximal colon at the same time as in the control animals.

In pigs with HEV, the stomach was clearly dilated and the emptying greatly disturbed. Radiopaque material was retained in the gastric lumen for 49 to 171 hours. The barium entered the small intestine 5 to 13 hours after the administration, but the time taken for the head of the meal to travel from the small intestine to the colon was the same as in the control animals.

DISCUSSION. Based on the present knowledge, the following concepts on the pathogenesis of the wasting in pigs with HEV can be put forward. The gastric emptying is greatly disturbed. This is probably a consequence of the earlier viral multiplication in neurons of the extrinsic and intrinsic innervation of the stomach. The delayed emptying is not only due to the earlier viral replication in the vagal nuclei of the brainstem and in the vagal ganglion, since the stomach emptying was less disturbed in pigs with a bilateral vagotomy. The viral multiplication in the submucosal and myenteric plexuses was probably jointly responsible for the gastric stasis in pigs infected with HEV.

The food stagnation in the stomach can be the cause of gastric dilatation, obstipation and reduced appetite in pigs which are chronically ill. Furthermore, the few swallows of milk which are taken up are retained in the gastric lumen for several days and its nutritive value may have become very low when entering in the intestine. The pigs have to use their own body protein and glycogen to stay alive, and die of emaciation after a few weeks.

REFERENCES

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2) Andries K., Pensaert M.B.: 1980, Am. J. Vet. Res., 41: 1372.

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