Further observations on stomach function of scouring piglets

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It has been shown that gastric retention and distension precede clinical scour in piglets (White, Wenham, Sharman, Jones, Rattray & McDonald, 1969). Studies using X-rays of normal piglets showed that radio-opaque material entering the stomach passes into the small intestine within a matter of minutes, with the stomach emptying after approximately 30 min. Barium sulphate-laden material reached the distal colon approximately 12 h after administration and was still evident in pelleted faeces 36 h after.

In the piglets which subsequently scoured, the first indication of abnormality was a decrease in the rate of passage of solid material from the stomach to the small intestine. The piglets continued to eat and gastric hypomotility increased. Retention of solid material and distention followed, and finally gastric stasis. Feeding was often followed by vomiting in piglets with distended stomachs. The distended stomach could reach such a size that it occupied something approaching 50% of the abdominal cavity and caused increasing compression of the thorax with possible cardiac embarrassment. This compression could explain why some piglets dying of scour are cyanosed. About 200 piglets have now been examined with X-rays and in all instances gastric hypomotility has preceded scour. Return of gastric motility has always preceded recovery from the scour syndrome and, if this spontaneous return of gastric motility occurs soon enough, actual scouring may not occur, the only effect being a check in weight gain. Up to three separate episodes of gastric hypomotility have been seen in individual piglets during the first 3 weeks of life. Although all piglets in these studies received colostrum by suckling their dam for the first 2 d of life, the incidence, duration and severity of gastric malfunction was less in piglets left on the sow compared with artificially reared piglets weaned at 2 d old, the incidence being approximately 20%. Periods of gastric malfunction show up on the pattern of weight gain in piglets (Fig. 1).

Death of piglets suffering from the scour syndrome can be attributed to a number of factors—starvation, dehydration and electrolyte loss due to scouring, effects due to compression of the thorax and the eventual invasion of the debilitated piglet by its own bacterial flora.

Since the aetiology of the scour syndrome in piglets is usually attributed to *Escherichia coli* (Sojka, 1965), bacteriological studies were carried out. Barium sulphate was given to the piglets by stomach-tube, and before its administration a sample of stomach contents was withdrawn for bacteriological examination and pH

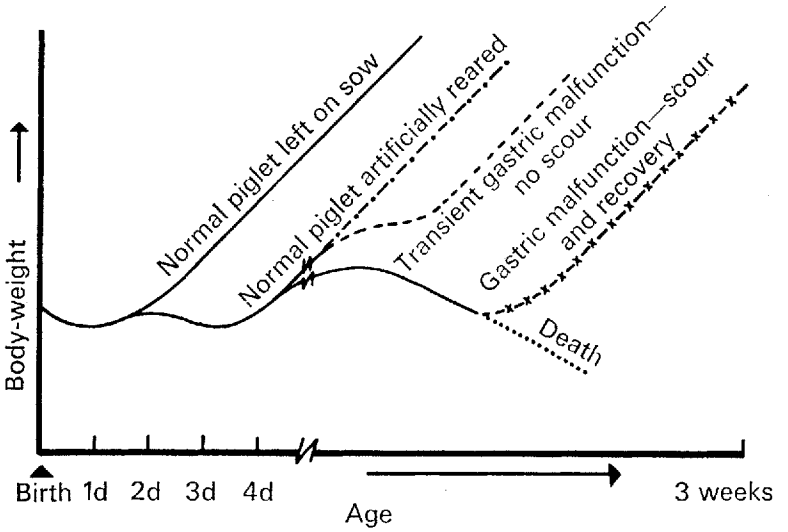

Fig. 1. Pattern of changes in body-weight of normal and artificially reared piglets, and the influence of gastric malfunction and scouring.

measurement. Counts of lactobacilli, total coliforms, haemolytic coliforms and total count of organisms growing on blood agar plates were obtained from naturally reared and artificially reared piglets, both scouring and non-scouring, during the first 3 weeks of life. Similar counts were also obtained at autopsy from stomach contents, anterior and posterior small intestine and colon of normal and scouring piglets. No statistically significant differences were found, except in artificially reared piglets to whose food, lactic acid had been added to reduce its pH to 4·8; counts were approximately one-tenth of those obtained from other piglets. The addition of lactic acid to the food had no influence on the incidence of gastric malfunction. Counts obtained from normal healthy piglets left on the sow were often higher than those obtained from scouring piglets.

The pH changes of stomach contents with age are given in Fig. 2. A suggestion which could account for these changes is that the initial low pH at birth is due to maternal gastrin crossing the placenta and stimulating the foetal stomach. At parturition, this supply of gastrin ceases and the pH rises.

Histological studies using routine techniques, and haematoxylin and eosin staining, were carried out on the small intestine, fundus of the stomach and pylorus of scouring pigs; these showed only hyperaemia and slight oedema between the muscle layers as compared with normal healthy controls. No damage suggesting non-contractibility of smooth muscle cells was observed.

As the radiographic appearances and clinical effects were similar to those of human infants with pyloric stenosis (Hamilton & Curtis, 1942; Larsen, 1966; Shuman, Darling & Fisher, 1967), fluoroscopic examinations were carried out on piglets showing gastric malfunction to investigate pyloric function. These studies ruled out true obstructive pyloric stenosis because, when piglets were positioned

vertically behind the fluorescent screen, barium sulphate suspension together with the liquid fraction of the diet were seen to flow passively through the pylorus. It was concluded that gastric retention was due to a lack of muscular propulsion of the solid fraction of the diet through the pylorus.

This led to a consideration of the innervation of the stomach. Because of the dual gastric innervation, one stimulating and the other inhibiting stomach movements, either diminished stimulation or increased inhibition could bring about the observed effects. There is now evidence of a third division of the autonomic nervous system inhibiting the gut (Christensen, 1971) and that adenosine triphosphate or a related nucleotide is the transmitter substance, (Burnstock, Campbell, Satchell & Smythe, 1970) but this knowledge was not available at the time. The possibility of stress having effects on the stomach via the adrenergic sympathetic division of the autonomic nervous system was considered. Stimulating, cholinergic nerves of the stomach are carried by the vagus nerve. The observed syndrome in the piglet was very similar to the sequelae of vagotomy in the human, (Williams & Cox, 1969). The administration of acetylcholine or analogues post vagotomy in humans has been shown to relieve gastric retention (Williams & Cox, 1969). Experiments were carried out, in an endeavour to influence both of these possible factors, namely reduced parasympathetic stimulation or increased sympathetic inhibition, using an acetylcholine analogue bethanochol chloride (Myotonine; Glenwood Laboratories Ltd, London), an α adrenergic blocking agent, phenoxybenzamine HCl (Dibenyline; Smith, Kline & French Laboratories Ltd, Welwyn Garden City, Herts.), and a β adrenergic blocking agent, propanolol (Inderal; Imperial Chemical Industries Ltd). In none of these experiments, using a range of doses, was gastric hypomotility or stasis influenced in any way compared with untreated controls. Repeated injections of adrenalin were administered to normal piglets to precipitate gastric hypomotility but this also failed. The tranquillizer Droleptan (Ortho-Pharmaceutical Ltd, Saunderton, High Wycombe, Bucks.) was used in an endeavour to reduce the

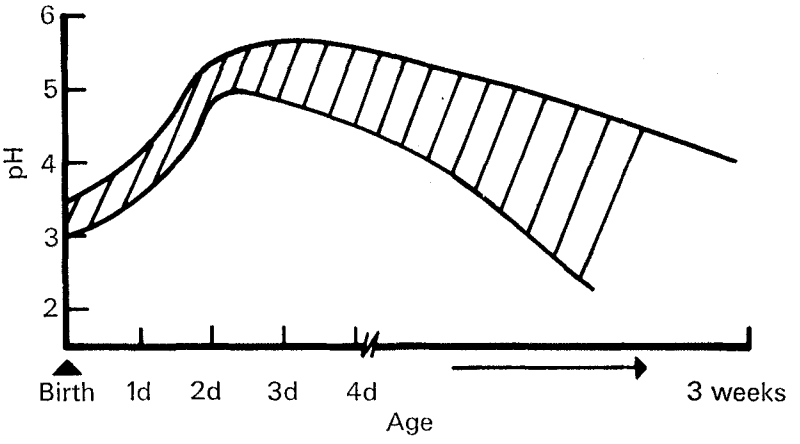

Fig. 2. pH of stomach contents of normal piglets.

https://doi.org/10.1079/PNS19720013 Published online by Cambridge University Press

effects of stress again without any effect on the scour syndrome. The piglet proved to be extremely tolerant of the agents administered, particularly adrenalin.

Adrenal insufficiency in human infants (Jaudon, 1946, 1948; Geppert, Spencer & Richmond, 1950; Morec, 1953; Boyd & MacDonald, 1960) produces symptoms very similar to those of infantile pyloric stenosis. Experiments were conducted to ascertain whether similar adrenal replacement therapy would influence the scour syndrome, again with no effect as compared with untreated controls.

Bachmann (1968) in the course of veterinary practice in Denmark found a similar scouring condition in piglets; the symptoms included vomiting, distension of the abdomen and dehydration. At autopsy, atony and marked dilation of the stomach were found. The administration of ascorbic acid produced an immediate and complete cure. Controlled experiments, in which varying amounts of ascorbic acid were administered to piglets, showed no effects on this syndrome compared with untreated controls.

The effects of pentagastrin (Peptavlon; Imperial Chemical Industries Ltd) on gastric motility (Thompson, 1969) were then considered, in view of the pH curve of stomach contents (Fig. 2). In the normal piglet, fluoroscopy showed that injections of small doses of pentagastrin increased the strength and frequency of stomach contractions; larger doses inhibited this activity. In the piglet with hypomotility of the stomach, pentagastrin, at any of the doses that were tried, failed to influence gastric motility.

Degeneration of intramuscular ganglion cells in Auerbach's plexus has been found in the pyloric region of children with infantile pyloric stenosis (Herbst, 1934; Raia, 1943; Alarotu & Christensen, 1949; Belding & Kernohan, 1953; Steinicke Nielsen, 1956*a*). Steinicke Nielsen (1956*b*) examined the pylorus from three groups of baby pigs: (1) piglets dying within the first few days of life after becoming apathetic and weak with occasional vomiting, and showing a distended stomach at autopsy; (2) pigs found dead and regarded as overlaid by their dam, also showing distended stomachs; (3) perfectly normal piglets killed for use as controls. In the first two groups of pigs he found irreversible ganglion cell damage in the pyloric part of the stomach identical to that found in infantile pyloric stenosis. In view of the findings of Steinicke Nielsen (1956*a*,*b*), ganglion cells in the pyloric region of scouring piglets were examined histologically. Preliminary results indicate identical neurone damage to that described by Steinicke Nielsen (1956*a*,*b*). The clinical symptoms observed in infantile pyloric stenosis in human babies were considered to be a consequence of the described nerve-cell damage (Alarotu, 1956). Similar neurone damage in the scouring piglet could account for the scouring syndrome. Spontaneous recovery could be explained by further neurone differentiation in the young animal and recovery of the less severely affected neurones.

REFERENCES

Alarotu, H. (1956). *Acta paediat., Stockh.* **45**, 579.
Alarotu, H. & Christensen, E. (1949). *Acta paediat., Stockh.*, Suppl. **77**, 250.
Bachmann, P. (1968). *Medlemsbl. danske Dyrlaegeforen.* **51**, 38.

Belding, H. H. & Kernohan, J. W. (1953). *Surgery Gynec. Obstet.* **97**, 322.
Boyd, J. F. & MacDonald, A. M. (1960). *Archs Dis. Childh.* **35**, 561.
Burnstock, G., Campbell, G., Satchell, D. & Smythe, A. (1970). *Br. J. Pharmac. Chemother.* **40**, 668.
Christensen, J. (1971). *Gasteroenterology* **60**, 1130. [Editorial.]
Geppert, L. J., Spencer, W. A. & Richmond, A. M (1950). *J. Pediat.* **37**, 1.
Herbst, C. (1934). *Z. Kinderheilk.* **56**, 122.
Hamilton, F. E. & Curtis, G. M. (1942). *Rev. Gasteroent.* **9**, 176.
Jaudon, J. C. (1946). *J. Pediat.* **29**, 696.
Jaudon, J. C. (1948). *J. Pediat.* **32**, 641.
Larsen, G. (1966). *Surgery, St Louis* **60**, 768.
Morec, G. J. (1953). *J. Pediat.* **42**, 359.
Raia, A. (1943). Quoted by Alverez, W. C. (1950). *Gasteroenterology* **16**, 787.
Shuman, F. T., Darling, D. B. & Fisher, J. H. (1967). *J. Pediat.* **71**, 70.
Steinicke Nielsen, O. (1956*a*). *Acta paediat., Stockh.* **45**, 636.
Steinicke Nielsen, O. (1956*b*). *Acta paediat., Stockh.* **45**, 444.
Sojka, W. J. (1965). *Rev. Ser. Commonw. Bur. Anim. Hlth* no. 7.
Thompson, J. C. (1969). *A. Rev. Med.* **20**, 291.
White, F., Wenham, G., Sharman, G. A. M., Jones, A. S., Rattray, E. A. S. & McDonald, I. (1969). *Br. J. Nutr.* **23**, 847.
Williams, J. A. & Cox, A. G. (editors) (1969). *After Vagotomy*. London: Butterworths.

Printed in Great Britain