We wish to thank Dr C. Vivier, Chief Medical Superintendent of Tygerberg Hospital, for permission to publish.

REFERENCES

1. Driscoll, S. G. (1977): Hum. Path., **8**, 529.
2. Jones, W. B. (1975): Clin. Obstet. Gynec., **18**, 247.
3. Hilgers, R. D. and Lewis, J. L. (1974): Gynec. Oncol., **2**, 460.
4. Ober, W. B., Edgcomb, J. H. and Price, E. B. (1971): Ann. N.Y. Acad. Sci., **172**, 299.
5. Ancel Blaustein (1977): *Pathology of the Female Genital Tract*, pp. 704 - 709. New York: Springer-Verlag.
6. Brewer, J. I. and Gerbie, A. B. (1966): Amer. J. Obstet. Gynec., **94**, 692.

# Lesser Curve Necrosis Following Proximal Cell Vagotomy for Gastric Ulcer

## A Case Report

D. F. DU TOIT, C. B. KEMP

## SUMMARY

**A case of avascular necrosis of the lesser curve of the stomach following a highly selective vagotomy (HSV) for a gastric ulcer is reported. It is fortunately rare, but is a frightening complication. The necrosis, which is presumably ischaemic in origin, is caused by total devascularization of the lesser curvature of the stomach. The complication is usually diagnosed late and has a considerable mortality.**

*S. Afr. med. J.*, **55**, 98 (1979).

With highly selective vagotomy (HSV) the integrity of the alimentary tract is not breached and there is no anastomosis, thus minimizing the complication of leakage and bleeding. Other case reports describing necrosis of the lesser curvature following HSV for duodenal ulceration have been reported in recent literature.[1-7] No cases of lesser curvature necrosis following HSV for gastric ulcer have been recorded.

An HSV can be recommended for combined gastric and duodenal ulceration, or for gastric ulceration alone.[8] HSV for gastric ulcers was found to be effective, but the final place of this procedure in the treatment of patients with both duodenal and gastric ulceration has still to be defined. Only long-term follow-up will clarify this.

The operative mortality rate was approximately 0,3% for 5 539 cases reviewed when HSV was performed for duodenal ulceration.[5] Necrosis of the lesser curvature occurred in 0,2% of the abovementioned series.

**Department of Surgery, University of Stellenbosch and Tygerberg Hospital, Parowvallei, CP**

D. F. DU TOIT, M.B. CH.B., F.C.S. (S.A.), F.R.C.S.
C. B. KEMP, M.B. CH.B., M.MED. (SURG.)

Date received: 14 July 1978.

## CASE REPORT

A 72-year-old Coloured woman was admitted to the surgical unit with severe haematemesis and melaena. No history of salicylate ingestion could be obtained, and she had had no previous episodes of gastro-intestinal haemorrhage.

On examination the blood pressure was 140/90 mmHg, pulse rate 110/min and the haematocrit 29%. She was of small stature with a mass of 46 kg. Cardiovascular, respiratory and abdominal examinations were within normal limits. Melaena was present on rectal examination. The urine analysis was normal and a chest radiograph was within normal limits.

A gastric ulcer measuring 1 cm in diameter on the lesser curvature of the stomach was demonstrated on gastroscopy and on a barium study. The appearance was that of a benign ulcer.

A laparotomy was warranted after a second major haemorrhage had occurred. At operation the stomach appeared normal. No ulcer could be seen or felt in the duodenum or along the lesser curvature. No perigastric fibrosis was present in the lesser omentum. The small and large bowel contained blood but because the haemorrhage had ceased, the proximal jejunum was empty.

In view of the patient's age and stature, an HSV was performed using the technique described by Amdrup.[9] The dissection commenced at the pyloric region after the nerves of Laterjat had been identified, and was performed from below upwards. The left index finger was passed

through an avascular hole made in the lesser omentum, behind the stomach, so that the structures were lifted anteriorly. The vascular branches to the stomach were isolated and divided between metal clips. First the anterior and then the posterior vessels to the lesser curve were divided. The dissection continued above the cardia so that the oesophagus could be encircled with the left index finger. The distal 5 - 6 cm of the oesophagus were cleared of all nervous connections. A circumferential search for nerves descending along the oesophagus concluded this part of the procedure.

During the procedure no diathermy was used on the stomach and no gastrotomy was performed as the ulcer could not be seen or palpated. The short gastric vessels were left intact.

The first 4 postoperative days were uneventful with the return of normal bowel sounds on the third day and passage of flatus on the fourth day. On the fifth day, a melaena stool was passed, the cause of which was attributed to a second haemorrhage from the ulcer since it had not previously been excised. On the sixth day, the abdomen was distended and became progressively worse with disappearance of bowel sounds. On the eighth day, the patient had a further severe upper gastro-intestinal haemorrhage associated with a serosanguineous discharge from the operation wound. She responded poorly to resuscitative measures and had persistent hypotension and anaemia. A provisional diagnosis of wound dehiscence and intra-abdominal haemorrhage originating from the stomach was made. Prior to surgery she had a cardiac arrest but recovered sufficiently after resuscitative measures to be able to undergo a further procedure. An emergency laparotomy was performed on the same day because of the persistent hypotension, abdominal distension, wound dehiscence and frank peritonitis.

At laparotomy, dehiscence of the wound and gangrene of the entire lesser curvature were confirmed. The process had extended from the pyloric region onto the lower 2 cm of the oesophagus. Free perforation on the lesser curvature had occurred. The size of the defect was 8 × 2,5 cm. The edge of the perforation was marked by green necrotic tissue. Thrombosed vessels in the edge were present in association with active bleeding from small vessels. A large clot was present in the lumen of the stomach immediately adjacent to the perforation and extending up to the wound. A pungent odour was present and the adjacent organs showed signs of frank peritonitis. After removal of the clot, the interior part of the stomach, anterior and posterior surfaces, and the pyloric region were found to be within normal limits. No stenosis or further ulceration was present. The initial ulcer could not be seen and it was assumed that this and the entire surrounding lesser curvature had undergone necrosis.

Because the patient was a poor operative risk, the lesser curvature was reconstructed after debridement, and omentum was used to cover the suture line.

The condition of the patient continued to deteriorate in the postoperative phase, with renal failure, a bleeding diathesis, and the adult respiratory distress syndrome. She died 2 weeks later after the second laparotomy and as a result of a further severe upper gastro-intestinal haemorrhage in association with cardiac arrhythmia and myocardial infarction. It was postulated that the upper gastro-intestinal haemorrhage was caused by diffuse gastric mucosal bleeding associated with the peritonitis, or due to breakdown of the sutured lesser curvature after acceleration of the necrotic process. Permission for autopsy was refused by the family.

## DISCUSSION

The technique of HSV with excision of the ulcer for gastric ulceration has been described by Johnston[8] but the exact place of HSV in the management of gastric ulceration is still uncertain.[8] The literature indicates that HSV is a safe procedure for duodenal ulceration.[5,10,11] HSV is indicated in undernourished or frail, elderly patients to avoid the untoward effects of gastric resection.[8]

The ulcer is palpated and excised by means of diathermy and sharp dissection through an anterior gastrotomy as near to the greater curvature as possible. The ulcer is shaved off the fibrotic base and the defect is closed with interrupted chromic sutures.[8] At times the ulcer may be very large and inaccessible, especially if it is near the cardia or pyloric region. Excision occasionally leads to free perforation, and it is suggested that representative biopsies alone (of the ulcer) should be taken and the ulcer left *in situ*.

Dissection of gastric ulcers may be difficult because of distortion of the lesser curvature due to perigastric fibrosis. Depending on the site of the ulcer, a limited HSV may be performed.[8] Lesser curvature necrosis is a complication of HSV.[1-4,6,7] The true pathogenesis is not clear, but the presently accepted theory is that of ischaemic necrosis due to devascularization of the lesser curvature.[1,5,6] The anterior and posterior walls have a high vascular anastomotic network. The lesser curvature has a relatively poor submucosal blood flow and a different vascular pattern.[1,5] The lesser curvature is mainly supplied by small vessels arising from the right and left gastric arteries and therefore may have potential areas of ischaemia. Vagotomy alone reduces gastric mucosal blood flow and this may be due to the opening up of submucosal arteriovenous shunts in and around the lesser curvature where the submucous vascular plexus is lacking.[1] This mechanism may produce ischaemic necrosis. Patients with gastric ulcers of the lesser curvature have low mucosal flow rates, and devascularization during HSV may precipitate this complication. HSV has been thought to reduce the blood supply to the lesser curvature.

The condition can present in an insidious fashion such as hydrothorax or as a fulminating condition with haemorrhage, free perforation or frank peritonitis.[1] Abdominal parasynthesis may confirm a frank haemoperitoneum due to perforation. Avascular necrosis of the lesser curvature should be suspected in patients who suddenly show signs of peritonitis in the first postoperative week after HSV. The diagnosis is usually made with difficulty in this rare condition, but should prompt surgeons to re-explore the abdomen in any patient who develops signs of intra-

abdominal complications within 10 days after operation.

It has been shown that the stomach has an unrivalled resistance to ischaemic damage, and Wilson-Hey advocated a 'four-point gastric ligation' for duodenal ulceration 40 years ago without having encountered ischaemic necrosis as a complication.[6] This may not be the case when an HSV is performed for gastric ulcer, when the dissection is performed through fibrous tissue adjacent to a relatively avascular zone.[5] Excessive diathermy along the lesser curvature might be responsible for oedema, ensuing ischaemic necrosis, and may present with perforation or haemorrhage.[6]

Preventive measures should include the following: a technically skilled surgeon should perform the procedure and the short gastric vessels should at all times be preserved if possible. Devascularization must be limited to a minimum except for the essential vessels which should be removed. Splenectomy during HSV may be indicated because of trauma, but it should be remembered that splenectomy may further impair the blood supply to the lesser curvature.[5] Ligation of small areas of serosa, including submucosal vessels, should be avoided.[6] HSV should be avoided in patients who undergo renal transplantation until further information is available as the mortality seems higher in these patients.[5] An omental patch, infolding of the lesser curvature or reperitonizing the lesser curvature after dissection may be of added value.[1,5] Any surgeon who has experienced this complication may feel sceptical about the value of reperitonization of the lesser curvature which may seal off small perforations. Because necrosis is usually extensive, reperitonization of the lesser curvature may therefore be of doubtful value.

The best management of necrosis of the lesser curvature is uncertain, as only a small number of cases have been reported. In the case under discussion, the necrosis extended from the pyloro-antral segment onto the lower end of the oesophagus. Debridement of the necrotic edges and peritoneal toilet may be all that are required to repair the defect. As these patients are usually extremely ill, a debridement and suture of the stomach are possibly all that is indicated since gastric resection may contribute to a higher mortality. We feel that the dissection along the lesser curve should be performed with sufficient care and skill, avoiding the use of diathermy, and that the lesser omentum defect should be carefully repaired.

The view that HSV for uncomplicated duodenal and gastric ulcers is free from both serious or minor complications should be qualified. This serious complication demands careful consideration. The incidence of this pathological process can be expected to increase as more and more cases are subjected to HSV.

REFERENCES

1. Halvorsen, J. F., Heimann, P., Solhaug, J. H. *et al.* (1975): Brit. med. J., **2**, 590.
2. Brodie, S. W. (1975): *Ibid.*, **3**, 229.
3. Kalsbeek, H. L. (1977): Arch. chir. Neerl., **29**, 101.
4. Lambrecht, W. (1977): Chirurg, **48**, 742.
5. Johnston, D. (1975): Brit. med. J., **4**, 545.
6. Taylor, T. V. and Torrance, B. (1976): *Ibid.*, **1**, 222.
7. Moore, F. P. and Wyllie, J. H. (1976): *Ibid.*, **4**, 328.
8. Johnston, D. *in* Rob, C. and Smith, R., eds (1977): *Operative Surgery*, 3rd ed., pp. 142 - 149. London: Butterworths.
9. Amdrup, E. *in* Rob, C. and Smith, R., eds (1977): *Ibid.*, pp. 137 - 141.
10. Goligher, J. C. (1974): Brit. J. Surg., **61**, 337.
11. Johnston, D. (1974): Gut, **15**, 748.
12. Heffernan, S. J. (1976): *Ibid.*, **1**, 398.

# Fetal Malformation due to Phenobarbitone

## A Case Report

F. E. BERKOWITZ

### SUMMARY

**A child with facial and digital anomalies and mental retardation after exposure to phenobarbitone during gestation is presented. The teratogenicity of anticonvulsants, including phenobarbitone, is discussed, and guidelines for anticonvulsant therapy during pregnancy are suggested.**

*S. Afr. med. J.*, **55**, 100 (1979).

**Department of Paediatrics, Baragwanath Hospital, Johannesburg**

F. E. BERKOWITZ, B.SC., M.B. B.CH., D.C.H., F.C.P. (S.A.)

Date received: 2 August 1978.

The possibility that anticonvulsant drugs are teratogenic was suggested by Janz and Fuchs[1] in 1964. Since then there have been many reports of fetal malformations associated with maternal ingestion of anticonvulsants during pregnancy.[2-8] In many of these cases diphenylhydantoin or combinations of anticonvulsants, including phenobarbitone, were used. Although phenobarbitone, used alone, appears to have been teratogenic in a few cases,[3,8,9-11] these teratogenic effects have not been emphasized. The present case provides further evidence of the teratogenic effect of phenobarbitone.