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Effects of Direct-interrupting Surgery on Gastric Mucosal Congestion in Patients with Esophageal Varices

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Summary. **The purpose of this study is to clarify the effect of direct-interrupting surgery on gastric mucosal congestion in patients with esophageal varices. Fifteen such patients underwent endoscopy in the preoperative, early postoperative and late postoperative periods. The index of local blood volume (IHb) and that of oxygen saturation (ISO2) were endoscopically measured using a tissue reflectance spectrophotometer at the antrum, the angle, and the corpus of the stomach. The congestion index (CI) was calculated by dividing IHb by ISO2. At the antrum and the angle, none of the mean values of IHb, ISO2, CI, or the incidence of mucosal redness changed significantly. At the corpus, the mean values of IHb and CI in the early postoperative period were significantly lower than those in the preoperative period ($p<0.01$ and $p<0.05$, respectively), but those in the late postoperative period showed no significant differences from preoperative and early postoperative values. Also at the corpus, the mean value of ISO2 and the incidence of mucosal redness showed no significant change in the early and late postoperative periods. These results suggest that gastric mucosal congestion of the corpus of the stomach in patients with esophageal varices can be relieved in the early postoperative period although it may recur in the late postoperative period.**

Key words—congestive gastropathy, direct-interrupting surgery, reflectance spectrophotometry.

INTRODUCTION

Bleeding from the gastric mucosa is one serious complication in patients with esophageal varices, in addition to variceal bleeding itself. In a previous study, we reported that there was mucosal congestion at the upper corpus of the stomach in such patients.[1] Change in the mucosal congestion after medical or surgical treatment for esophageal varices is a further issue for investigation, although the latter has been left largely unstudied. The current study was undertaken to clarify the effect of direct-interrupting surgery on the gastric mucosal congestion using reflectance spectrophotometry.

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PATIENTS AND METHODS

Fifteen patients with esophageal varices (6 men and 9 women) aged 35-71 years (53 ± 11, mean$\pm$SD) considered appropriate for elective direct-interrupting surgery, were studied. Their primary disease, determined by means of a wedge biopsy of the liver at the time of surgery, consisted of 8 cases of liver cirrhosis, 5 of idiopathic portal hypertension, and 2 of primary biliary cirrhosis. Twelve of them belonged to Child's Group A, 2 to Group B, and 1 to Group C. The postoperative follow-up period ranged from 8 to 53 months (19 ± 11). Our direct-interrupting surgery consisted of esophageal transection, esophago-gastric devascularization and splenectmy.[2]

After an overnight fast, all patients were premedicated with an intramuscular injection of butropin bromide (4 mg) before endoscopy.

Following endoscopic observation of gastric mucosal redness, measurements of the index of local mucosal blood volume (IHb) and that of local oxygen saturation (ISO2) were carried out, using a tissue reflectance spectrophotometer (TS-200, Sumitomo-denko, Osaka). The congestion index (CI) was calculated by dividing IHb by ISO2. We hypothesized that the severe the congestion, the higher the IHb and the lower the ISO2, and that CI could thereby show the degree of congestion in one parameter.

Three sites of the stomach were selected for these

endoscopic measurements and observations: the greater curvature of the antrum (antrum), the lesser curvature of the angle (angle), and the greater curvature of the upper corpus (corpus).

These endoscopic examinations were performed three times per each patient in the preoperative period (Phase 0), early postoperative period within three months after surgery (Phase 1), and late postoperative period more than six months after surgery (Phase 2). Seven patients failed to undergo endoscopy in the early postoperative period because of anastomotic stenosis (n=4) or they did not come to the clinic for follow-up evaluation (n=3).

The mean values of blood hemoglobin in the three phases were compared to check the presence of significant changes. Also, the incidence of mucosal redness was compared.

The TS-200 is a tissue spectrum analyzer which calculates IHb and ISO2 every 0.5 sec, showing a positive correlation with local mucosal blood volume and local oxygen saturation, respectively. The depth of measured spectra is 0.3-0.4 mm from the surface of the mucosa, the uppermost layer of the mucosa still containing capillary loops.[3,4] A light probe with a 3 mm diameter was passed along the biopsy channel of the endoscope and contacted the mucosa as much "en face" as possible while applying minimal pressure. Measurements were made five times at each site to obtain mean values. Red spots were avoided as measuring sites, and nearby tissue was alternatively selected.

Statistical analysis

The results were expressed as the mean±SD. Statistical comparison between the preoperative and postoperative mean values of blood hemoglobin, IHb, ISO2, and CI were made with Student's paired *t*-test. Comparison of the incidence of mucosal redness was performed with an x^2-test. Differences, recognized at a probability of less than 0.05 were considered statistically significant.

RESULTS

1) Blood hemoglobin (Table 1)

The mean values of blood hemoglobin Phases 0, 1, 2 were respectively 11.9±2.0, 12.6±1.4, and 12.4±1.7, showing no significant change.

Table 1. Blood hemoglobin before and after direct-interrupting surgery, showing no significant changes

Phase	0	1	2
Hemoglobin (g/dl)	11.9±2.0	12.6±1.4	12.4±1.7

Phase 0: preoperative, Phase 1: within three months after surgery, Phase 2: more than six months after surgery.

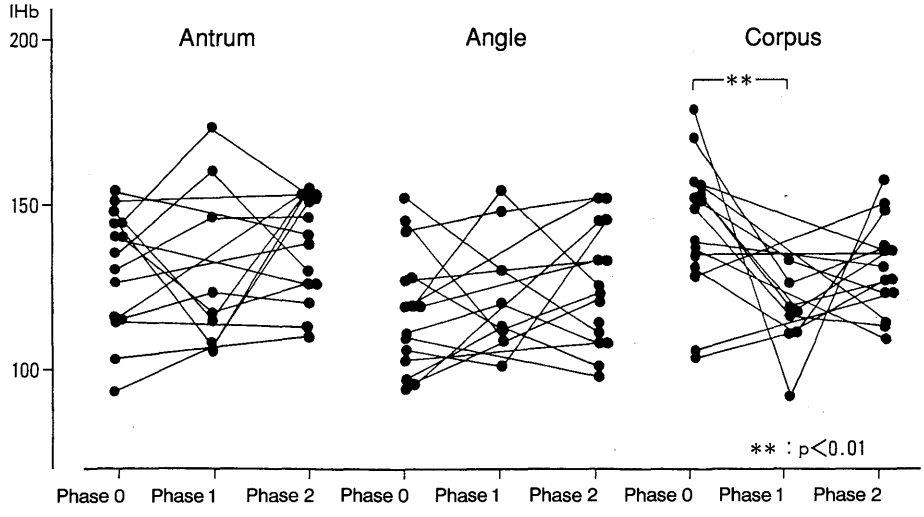

Fig. 1. Changes in the index of local gastric mucosal blood volume (IHb) after the direct-interrupting surgery. At the corpus, IHb decreases significantly in Phase 1, but returns to preoperative values in Phase 2. See Table 1 for the phase numbers.

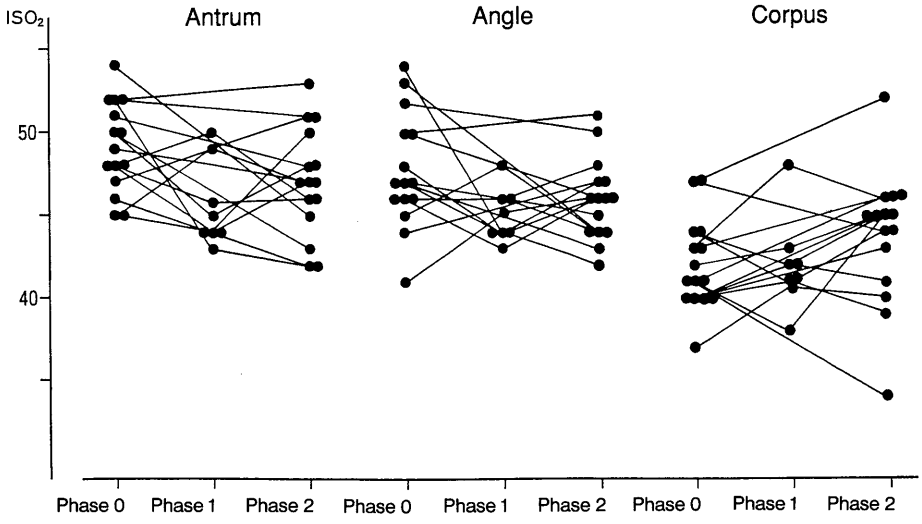

Fig. 2. Changes in the index of local gastric mucosal oxygen saturation (ISO2) after the direct-interrupting surgery. At the antrum, the angle, and at the corpus, ISO2 shows no significant change. See Table 1 for the phase numbers.

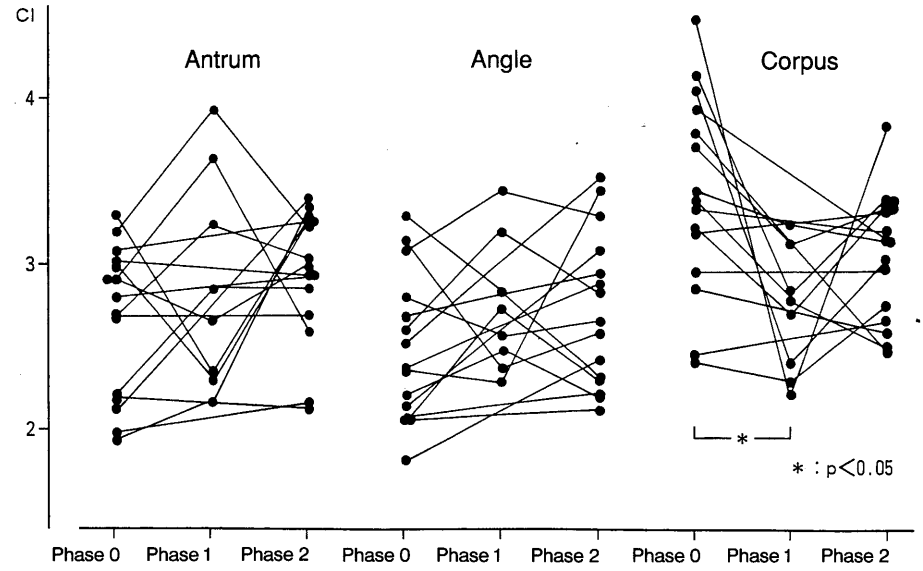

Fig. 3. Changes in the index of local gastric mucosal congestion (CI) after the direct-interrupting surgery. At the corpus, CI decreases significantly in Phase 1 but returns to preoperative values in Phase 2. See Table 1 for the phase numbers.

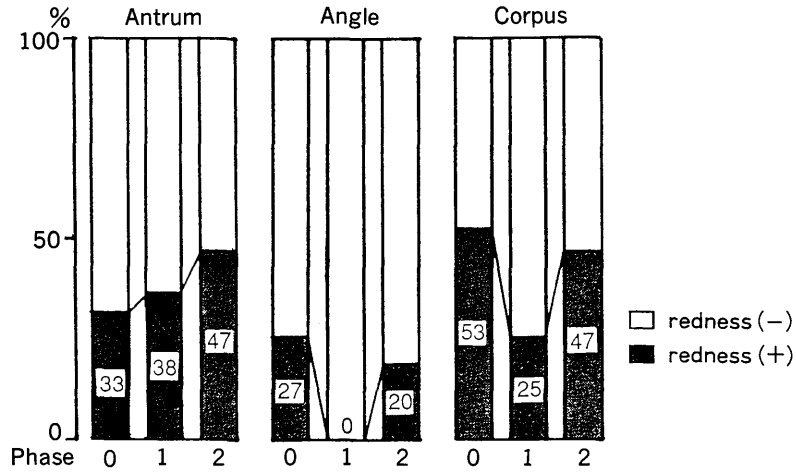

Fig. 4. Changes in the incidence of gastric mucosal redness after the direct-interrupting surgery. See Table 1 for the phase numbers.

2) IHb (Fig. 1)

The mean values of IHb in Phases 0, 1, 2 were respectively 130±19, 131±25, 138±16 at the antrum, 119±19, 124±19, 125±18 at the angle, and 143±21, 116±12, 131±14 at the corpus.

The mean value of IHb at the antrum and at the angle showed no significant change through the three phases. At the corpus, they decreased significantly in Phase 1 ($p<0.01$) and increased again in Phase 2, showing no significant difference from those in Phase 0 or 1.

3) ISO2 (Fig. 2)

The mean values of ISO2 in Phases 0, 1, 2 were respectively 49±3, 46±3, 47±3 at the antrum, 48±4, 45±2, 46±5 at the angle, and 42±3, 42±3, 44±3 at the corpus.

At the three sites of the stomach, no significant change in the mean values of ISO2 was observed through the three phases.

4) CI (Fig. 3)

The mean values of CI in Phases 0, 1, 2 were respectively 2.7±0.5, 1.9±1.2, 3.4±0.6, at the antrum, 2.5±0.4, 1.8±1.0, 2.9±0.4 at the angle, and 3.4±0.6, 1.8±0.9, 2.7±0.6 at the corpus.

The mean value of CI at the antrum and at the angle showed no significant change through the three phases. At the corpus, they decreased significantly in Phase 1 ($p<0.05$) and increased again in Phase 2, showing no significant difference from those in Phase 0 or 1.

5) Incidence of gastric mucosal redness (Fig. 4)

The incidence of mucosal redness in Phases 0, 1, 2 were respectively 33%, 38%, 47% at the antrum, 27%, 0%, 20% at the angle, and 53%, 25%, 47% at the corpus.

At the antrum, where the surgical procedure of the direct-interrupting surgery seems to have little effect, the incidence of gastric mucosal redness increased as the phase proceeded, showing no significant difference. At the angle and at the corpus, where the surgical procedures have greater effect, the incidence of the mucosal redness was lower in Phase 1 than that in Phase 0, and higher in Phase 2 than that in Phase 1, returning to approximate values in Phase 0. No significant difference was observed among these values, however.

DISCUSSION

Gastric mucosal bleeding in patients with esophageal varies is sometimes massive and life-threatening, accounting for 10-50% of all bleeding in such patients.[5-7] Gastric mucosal red spots, most frequently observed at the fundus and the corpus,[1,5,9-12] are considered to be a subclinical condition for this mucosal bleeding.[8] This mucosal appearance was originally mistakenly named as "hemorrhagic gastritis," but has been lately renamed "congestive gastropathy"[5]

or "portal hypertensive gastropathy"[13] based on its histological findings as characterized by the ectasia of mucosal and submucosal veins with little or no evidence of inflammation.

In our previous study, this mucosal congestion was expressed in terms of increased local blood volume and congestion index (higher IHb and CI), and decreased oxygen saturation (lower ISO2) by reflectance spectrophotometry.[1] By gas clearance technique, some investigators have reported a decreased blood flow of the upper corpus of the stomach.[14,15] However, the use of gas clearance technique does not permit investigators to distinguish congestion from ischemia, because it can express both of these states as the same "decreased blood flow." More recently developed laser velocimetry has the same disadvantage. In contrast to these methods, reflectance spectrophotometry has the advantage of being able to distinguish congestion from ischemia, showing higher IHb with lower ISO2 when congestion is present, and lower IHb with normal or lower ISO2 when ischemia is present.

The incidences of variceal bleeding and gastric bleeding after selective shunting surgery is 7.5% and 5.5%, respectively, and after direct-interrupting surgery are 6.8% and 3.8%, respectively.[16] The question arises as to how the mucosal congestion of the upper stomach changes after the variceal eradication, and there have been very few reports on the role of surgical procedures in this milieu. Mitarai, using gas clearance technique, reported a gastric mucosal blood flow reduced by 20% at four weeks after direct-interrupting surgery, and mucosal injury was observed in most cases that showed over 20% reduction.[17] On the other hand, Satani, also using gas clearance technique, observed moderate reduction and early recovery of gastric mucosal blood flow within one week after surgery, with almost no mucosal hemorrhage and only mind atrophic changes at the corpus at four weeks after the operation.[18] These two papers reported different results on gastric mucosal blood flow and mucosal redness after direct-interrupting surgery. Our endoscopic observation showed a lower incidence of gastric mucosal redness at the corpus in Phase 1 than in Phase 0, which seems to be in agreement with Satani's results at four weeks after surgery.

From the viewpoint of mucosal microcirculation, some new aspects have been revealed by the current results with the use of reflectance spectophotometry. At the corpus, the values of IHb and CI decreased significantly in Phase 1, and appeared to return to preoperative levels in Phase 2, although the blood hemoglobin did not change significantly. Also, the values of ISO2 and the incidence of mucosal redness at the corpus did not show any significant change through the three phases, but the incidence of mucosal redness decreased from 53% in Phase 0 to 25% in Phase 1. These results suggest that, after direct-interrupting surgery, the mucosal congestion of the corpus is relieved in Phase 1 and recurs in Phase 2, whereas at the angle, and at the antrum, none of the mean values of IHb, ISO2, CI, nor the incidence of mucosal redness changes significantly.

The mechanism of improvement of gastric mucoal congestion in Phase 1 needs further investigation. Devascularization probably plays an important role; it blocks not only collateral veins but also inflowing arteries of the corpus, thereby interrupting the regional hyperdynamic state. Portosystemic shunting operations are known to stop bleeding from portal hypertensive gastric mucosa.[19] If a direct-interrupting operation also accompanies portal decompression, as Kawasaki reported,[20] it could also contribute to relieving gastric mucosal congestion.

The mechanism of the recurrence of mucosal congestion of the corpus in the late postoperative period is another question to be answered. The same mechanism of recurrence of esophageal varices might be attributed to it; firstly, incomplete interruption of the local circulation from the portal vein and secondly, increased blood supply from the non-devascularized part of the stomach or the esophagus.[21]

The risk of gastric mucosal bleeding may increase after successful endoscopic injection sclerotherapy (EIS) for esophageal varices.[5,22-24] Some investigators have reported, using gas clearance technique, that the gastric mucosal blood flow is reduced after EIS[15,25] with exacerbation of gastric mucosal redness.[14] Although their follow-up intervals differ, EIS may have an adverse effect on congestive gastropathy, at least temporarily after the procedure.

Our results suggest that gastric mucosal congestion of the corpus in patients with esophageal varices is relieved in the early postoperative period, although this beneficial effect may diminish in the late postoperative period.

REFERENCES

1) Nakamura S, Tamiya Y, Matsubara Y, Tsukada K, Yoshida K, Muto T: Blood distribution and redness of the gastric mucosa in patients with esophageal varices. *J Gastroenterol Hepatol* (**Suppl 1**) 161-163, 1989.

2) Takano Y, Yoshida K, Honma K, Abe Y, Muto T: Esophageal transection combined with splenectomy and paraesophago-gastric devascularization for eso-

phageal varices. *Chir Gastroenterol* **13:** 21-27, 1979.
3) Sato N, Kamada T, Shichiri M, Kawano S, Abe H, Hagihara B: Measurement of hemoperfusion and oxygen sufficiency in gastric mucosa *in vivo*. *Gastroenterology* **76:** 814-819, 1979.
4) Taniguchi K, Yoshida K, Ohkawa S, Kitazaki T, Ono K, Hiramoto J, Matsumoto Y, Hirano K, Sunago K, Yotsuya K, Kanda M: The development of the tissue spectrum analyzer, TS-200. *Sumitomo Electric Technical Review* **25:** 127-132, 1986.
5) McCormak TT, Sims J, Eyre-Brook I, Kennedy H, Goepel J, Johnson AG, Triger DR: Gastric lesions in portal hypertension: inflammatory gastritis or congestive gastropathy? *Gut* **26:** 1226-1232, 1985.
6) Tres J, Bordas JM, Bru C, Diaz F, Brugiuera M, Rodes J: Upper gastrointestinal bleeding in cirrhosis: clinical and endoscopic correlations. *Gut* **17:** 37-40, 1976.
7) Nagao F, Inagaki Y: Portal hypertension and gastric bleeding. *Geka MOOK* **29:** 178-187, 1983. (in Japanese)
8) Iwao T, Toyonaga A, Tanikawa K: Gastric red spots in patients with cirrhosis: Subclinical condition of gastric mucosal hemorrhage? *Gastroenterol Jap* **25(6):** 685-692, 1990.
9) Papazian A, Braillon A, Dupas J, Sevenet F, Capron J: Portal hypertensive gastric mucosa: an endoscopic study. *Gut* **27:** 1199-1203, 1986.
10) Smart HL, Triger DR: Clinical features, pathophysiology and relevance of portal hypertensive gastropathy. *Endoscopy* **23:** 224-228, 1991.
11) Adachi H: The study of gastric mucosal changes and its mechanism in patients with liver cirrhosis. *Gastroenterol Endosc* **29:** 455-471, 1987. (in Japanese with English abstract)
12) Tsukada Y: Studies on the red spots on gastric mucosa in patients with chronic liver disease. *Niigata Igakukaizasshi* **101:** 797-803, 1988. (in Japanese with English abstract)
13) Tarnawski AS, Sarfeh IJ, Stachura J, Hajuduczek A, Bui HX, Dabros W, Gergely H: Microvasculature abnormalities of the portal hypertensive gastric mucosa. *Hepatology* **8(6):** 1488-1494, 1988.
14) Nishida H, Kodama T, Okano H, Satoh T, Tuji H, Mitsuhuji S, Takamasu M, Huruya S, Maruyama K, Fuse Y, Fukuda S, Takino T: Change of gastric mucosal lesions after endoscopic injection sclerotherapy (E.I.S.) *Jpn J Gastroenterol* **84:** 1037-1044, 1987. (in Japanese with English abstract)
15) Nishiwaki H, Asai T, Sowa M, Umeyama K: Endoscopic measurement of gastric mucosal blood flow with special reference to the effect of sclerotherapy in patients with liver cirrhosis. *Am J Gastroenterol* **85:** 34-37, 1990.
16) Kobayashi M: Present status and perspectives of surgical treatment for esophageal varices. *Gekachiryo* **47(3):** 323-331, 1982. (in Japanese with English abstract)
17) Mitarai Y, Kobayashi M: Correlation between gastric microcirculation and mucosal injury after surgical therapy for esophageal varices. *J Jpn Surg Soc* **91:** 101-113, 1990. (in Japanese with English abstract)
18) Satani H: Etiopathogenesis of hemorrhagic gastritis after esophageal transection with extended devascularization for variceal patients. *J Jpn Surg Soc* **87:** 29-43, 1986. (in Japanese with English abstract)
19) Sarfeh IJ, Juler GL, Stemmer EA, Mason GR: Results of surgical management of hemorrhagic gastritis in patients with gastro-esophageal varices. *Surg Gynecol Obstet* **155:** 167-170, 1982.
20) Kawasaki S, Kidokoro A, Sugiura M, Sanjo K, Idezuki Y: Effects of nonshunting operations on portal venous pressure and hepatic blood flow. *Am J Surg* **153:** 295-298, 1987.
21) Aoki H, Hasumi A, Yoshimatsu Y, Hasimura K, Ishida T, Hayashi O, Kaneta T, Yasuda Y, Oda N: Recurrence and remnants of esophago-gastric varices after non-shunting operation. *Jpn Gastroenterol* **24:** 201-208, 1991. (in Japanese with English abstract)
22) D'Amico G, Montalbano L, Traina M, Pisa R, Menozzi M, Spano C, Pagliaro L: Natural history of congestive gastropathy in cirrhosis. *Gastroenterology* **99:** 1558-1564, 1990.
23) Sarin SK, Sreenivas DV, Lahoti D, Saraya A: Factors influencing development of portal hypertensive gastropathy in patients with portal hypertension. *Gastroenterology* **102:** 994-999, 1992.
24) Portal hypertensive gastropathy (Editorials). *Lancet* **338:** 1045-1046, 1991.
25) Eleftheriadis E, Kotzampassi K, Aletras H: The influence of sclerotherapy on gastric mucosal blood flow distribution. *Am Surg* **56:** 593-595, 1990.