The aim of the study is the examination of the relative portal blood flow, by assessment of the hepatic perfusion index (HPI) in different degrees of hemo-
dynamic alterations related to liver cirrhosis and some focal liver diseases.
Hepatic radionuclide angiography (HRA) was performed with bolus injection of
740 MBq 99m-Tc-pertechnetate, during one minute (1 mtec), using ROTA
scintillation camera and Micro Delta computer (Siemens). HPI was estimated
by Sarper’s method of slope analysis.
In 10 controls, HPI was 0.68 ± 0.06; it was significantly decreased (p < 0.01)
in 5 patients with chronic active hepatitis (HAA, 0.57 ± 0.03), 13 with liver
cirrhosis without (LC, X = 0.49 ± 0.13) and 18 with esophageal varices
(LC/EV, X = 0.32 ± 0.19), as well as in 4 patients with LC and sclerosated
esophageal varices (LCEV, X = 0.16 ± 0.11). Comparing to HAI and LC
(HAHC, p > 0.05), HPI values were significantly lower in LCEV (p < 0.01)
and LCSEV (p = 0.05), while the values between the last two groups didn’t
differ (p > 0.05).
In 22 patients with liver hemangiom (LH, X = 0.64 ± 0.08) HPI values were
physiological (C-LH, p > 0.05). However, in 4 patients with hepatocellular
carcinoma (HC), HPI (X = 0.20), and 8 with liver metastases (LM, X = 0.40
± 0.28), HPI values were significantly decreased (p < 0.01), but they didn’t
differ between themselves (H-LM, p = 0.05).
Portal liver perfusion decreases in respect to the portal hypertension and
collateral circulation development. Thus, significant difference is proved be-
tween HPI values in cirrhotic patients with and without esophageal varices,
while after sclerotherapy, HPI remains very low. Considering that in patients
with hemangiomia, HPI values are normal, which is not the case in those
with primary carcinomas and metastases, HRA is a useful method for the dif-
ferential diagnosis of hemangiomia and primary liver carcinomas, together
with ultrasonography and blood pool scintigrapy.
try) and nutritional status (AC) were evaluated in 15 healthy subjects – control group (7 girls and 8 boys; age 13.33 ± 1.96; mean ± s.d.) and 20 patients (10 girls and 10 boys) with portal hypertension (9 with portal vein occlusion; age 12.56 ± 3.10; 11 with liver cirrhosis, age 14.00 ± 3.26) and history of variceal bleeding. Under nutrition was diagnosed in 3 controls and in 12 patients (p < 0.05). REE (per kg b.m.) in controls and patients were: 121.87 ± 21.50 kJ and 142.50 ± 32.91 kJ respectively (p = 0.043). REE (per 1.73 m²) were: 664.37 kJ and 745.66 kJ resp. (p < 0.0023). Substrate utilization rate for controls and patients were: glycerone: 1.90 g/kg b.m. and 2.23 g/kg b.m. resp. (n.s.); fat: 1.55 and 2.01 g/kg b.m. resp. (p < 0.04); protein: 1.51 g/kg b.m. and 1.23 g/kg b.m. resp. (n.s.). Conclusions: Patients with portal hypertension and history of variceal bleeding are prone to be malnourished as revealed by AC. Increased REE with increased fat utilisation rate can explain that finding.

**1651 A Prospective, Randomized Trial of Injection Sclerotherapy vs. Banding Ligation in the Management of Bleeding Esophageal Varices**


Aims: To compare the efficacy and complications between sclerotherapy and ligation in management of bleeding esophageal varices. Methods: 120 patients admitted to our hospital with acute esophageal variceal bleeding were randomized to receive sclerotherapy (EIS group) or band ligation (ELV group). All of them received treatment within 24 hrs of index bleeding. Sclerosant 1.5% Sotradecol and Steigmann-Goff ligator were used, respectively. Endoscopy treatment was repeated at an interval of 2-3 weeks until varices obliterated.

Results: Post-hepatic cirrhosis 43 (73%) 41 (87%) Pugh’s grade A/B/C 12%41%/47% 15%/56%/49% Control of active bleeding 12/21/90% 17/18/93% Recurrent bleeding 26 (44%) 9 (15%)* Ectopic varices bleeding 3 (5%) 8 (13%) Obliteration 37 (63%) 45 (74%) Sessions to obliteration 6.5 ± 1.2 3.8 ± 0.4* Patients with complications 16 (27%) 3 (5%)* Patients with mortality 19 (32%) 10 (16%)* *p < 0.001

Conclusions: Endoscopic variceal ligation causes significantly fewer complications than sclerotherapy. In addition, ETV prevents rebleeding from esophageal varices more effectively than EIS does. However, higher frequency of ectopic varices bleeding may be encountered by ETV.

**1652 The Impact of Endoscopic Variceal Ligation on the Pressure of the Portal System**


Background: Endoscopic variceal ligation (EVL) is a viable substitute for endoscopic injection sclerotherapy. The complications of EVL have been proven to be fewer than those of sclerotherapy. It is still unknown how EVL may influence the pressure of portal venous system.

Methods: 19 patients (18 males, one female, mean age, 60 ± 9yrs) with history of esophageal varices bleeding and without ascites were enrolled. All were cirrhotic patients (63% were post-hepatic). EVL was performed at an interval of 2–3 weeks until varices were obliterated. Measurements of portal pressure: Portal-splenovenogram was performed before EVL and after varices obliteration to assess venographic findings. The pressures of main portal vein (PVP), splenic vein (SVP) and superior mesenteric vein (SMMV) were recorded.

Results: 16 patients completed the study. A mean of 4 sessions of EVL within the duration of 2 months was needed. 11 (68%) patients had an elevated pressure and 5 (31%) patients had a reduced pressure after EVL. Mean portal venous pressure before and after EVL was 26.0 ± 4.4 mmHg and 27.9 ± 6.5 mmHg, respectively (p < 0.05). Among patients with an elevated pressure change, PVP increased by a mean of 24%, SVP increased 18% and SMMV increased 19%. Among patients with a reduced pressure change, PVP decreased 24%, SVP decreased 26% and SMMV decreased 22%. Three patients had rebleeding, all belonged to those with an increased portal pressure change.

Conclusions: 1. EVL induced an increased portal pressure by 24% in 70% patients and reduced portal pressure by 24% in 30% patients. 2. Increased portal pressure induced by EVL may result in the occurrence of rebleeding.

**1653 Postprandial Portal Hypersemia in Cirrhotic Patients as a Marker of Liver Disease and Portal Hypertension**

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Previous studies have shown an increase of portal pressure in cirrhotic patients during postprandial states. This increase is caused by an increase in hepatic blood flow, a phenomenon known as postprandial hyperemia. This study was designed to determine whether postprandial hyperemia might be an indicator of severity of liver disease and portal hypertension.

Materials and Methods: Portal flow was measured in 66 patients during fasting and 30 minutes after a standardized meal with an Acuson 128 Doppler system (Mountain View, CA). The degree of portal hypertensive gastropathy and esophageal varices was evaluated by endoscopy.

Results: After the meal the increase in portal flow was significantly lower in patients with severe gastropathy (+23%) and esophageal varices (+26%) compared to patients without gastric lesions (+39%, p < 0.003 and 0.03) and those without esophageal varices (+45%, p < 0.01). Postprandial portal flow increase was diminished in patients with esophageal bleeding or red spots (+24%) compared to non-bleeders (+37%, p < 0.05). Patients taking vasodilatory drugs had a smaller increase of postprandial portal flow (+21%) than patients without this medication (+37%, p < 0.05). Concerning the CHILD score there was a smaller increase in mean portal flow (PF) and volume flow (VF) in patients with higher grading (CHILD A PF +46%, VP +45%; CHILD B PF +32%, VF +33%; CHILD C PF +25%, VF +25%, p < 0.005). In contrast to postprandial flow, fasting portal venous blood flow did not correlate with bleeding risk or clinical scoring.

Conclusions: The postprandial portal hyperemia measured by doppler ultrasonography is inversely correlated with the severity of liver disease and portal hypertension. Small flow velocity increases may indicate a higher risk of variceal bleeding.

**1654 Clinical and Endoscopic Results in Cirrhotic Patients Submitted to the Transjugular Intra Hepatic Portosystemic Shunt (TIPS)**

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This report describes clinical results in 20 cirrhotic patients (14 M and 6 F mean age 62 ± 6 yrs) undergoing TIPS for variceal bleeding (n 18) or intractable ascites (n 2). Liver disease was postivral in 85%. Child class was A in 14 and B-C in 6 patients. At present the mean follow-up is 6.27 ± 4.27 months range 2-18 months in survivors. The mean portosystemic gradient decreased from 30 ± 5 to 11 ± 4 cmH2O. In one patient bleeding could not be stopped and the patient died after 24 hrs. Another patient died after 30 days due to hepemorropoe. Varices were evaluated by endoscopy using the NIEC score before, at 1 week and at 3 and 6 months after TIPS. A sharp decrease in the severity of esophageal varices was already observed after one week (basal score 3.88 ± 1.21 vs one week 1.36 ± 1.6 p < 0.0001) and was maintained at 6 months (score 1.5 ± 1.4). Bleeding occurred in 1 patient after 60 days from a small residual esophageal varices and was successfully treated with sclerotherapy. Congestive gastropathy (CG) was observed in 8 patients before TIPS. After 3 months CG was unmodifed but at 6 months an improvement in the degree of CG was observed in 5 patients. During follow-up 4 patients manifested a stent stenosis (2, 3, 7, 8 months post TIPS), evidenced by doppler US as a reduction in blood velocity in the stent, which was treated with angioplastic (2 cases) or with a second stent implantation in 2 others. In conclusion TIPS is a feasible procedure for the treatment of variceal hemorrhage in cirrhotic patients, varices almost disappeared in the majority of patients, CG was ameliorated, only 1 bleeding episode was observed within 6 months. Doppler-US is useful in monitoring stent patency.

**1655 Somatostatin as an Adjuvant to Emergency Sclerotherapy of Bleeding Esophageal Varices**

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Introduction: - Acute sclerotherapy (AS) is considered to be an useful and difficult technique because of the critical situation of the patient and the unoptimal viewing condition. Somatostatin reduces splanchic blood flow and portal pressure. Our aim was to evaluate in a double-blind way the usefulness of the simultaneous injection of a bolus of somatostatin as an adjuvant to AS.

Material and Methods: - 63 patients with bleeding esophageal varices were admitted to the trial. 33 received a bolus of 250 mcg of somatostatin. The remaining 30 received a placebo. All of them were actively bleeding. AE was carried out by injecting ethanolamine oleate to near the bleeding point.
The following variables were recorded: liver function, amount of sclerosant, hemodynamic condition, hemorrhagic activity, duration of the procedure, units of transfused blood, hemorrhagic recurrence and mortality. An overall index of technical feasibility (ITF) was designed as the product: (number of injections required x viewing condition (1–3) x subjective feeling of technical difficulty (1–3). Data were compared by t-student and chi-square tests.

**Results** - Both groups were similar on the following parameters: age, hemodynamic condition, degree of patient’s collaboration, activity of hemorrhage and liver function. Although not significantly, more treated patients (14/33) had bled from gastric subcardiac varices than placebo patients did (7/30), (p = 0.1). Once the drug was administered, more treated patients ceased to bleed during the injection (21/33) than control patients (11/30) (p = 0.03). The amount of administered sclerosant was superior in the control group (3.0ml vs 9.4 ml) (p = 0.004). The ITF was significantly better in the treated group (3.5 vs 6.4, p = 0.03). On the contrary, no differences were registered in units of transfused blood, recurrence of hemorrhage, mortality or time spent in the procedure.

**Conclusion** - The administration of a bolus of 25% of somatostatin results in an amelioration of the technical easiness of AS even though it does not seem to substantially change the final result of the procedure in skilled hands. This therapeutic complement could allow AS to be undertaken not only by highly skilled endoscopists, but also by gastroenterologists with a moderate expertise on therapeutic endoscopy.

1658 | Congestive Gastropathy with Liver Cirrhosis


We have had gastrofibroscope examinations of many patients with congestive gastropathy complicated to liver cirrhosis. However, reddening and edema of gastric mucosa were not found in cases with congestive gastropathy, but chronic gastritis, respectively. Therefore the differential diagnosis as for the two diseases was considerably difficult. We tried to solve this problem and report a considerably successful diagnosis method using endoscopic toluidine blue dye-spraying technique for congestive gastropathy. [Materials and Methods] Endoscopic toluidine blue dye-spraying was carried out in 25 patients with congestive gastropathy complicated in liver cirrhosis and 30 patients with chronic gastritis. In gastrofibroscopy, the change metachromasia of colour after spraying to the mucosa with reddening was observed and biopsy specimens of the same gastric portion were taken. Furthermore, gastric juice was collected by aspiration. Histological diagnosis was done by HE. Histological staining using toluidine blue before and after chondroitin ABC digestion was carried out. The measurement of chondroitin sulfate in gastric juice was carried out using chondroitinase digestion method. [Results] In endoscopic findings, diffuse reddening of mucosa in the greater curvature of body was stronger in congestive gastropathy than in chronic gastritis. In another cases, reticular patterns, erythema, macular reddening, and edema were often found in the greater curvature of body. As for spraying pattern in body of congestive gastropathy, mucosal us blue colour pattern was 35% and the mixed pattern of blue and purple pattern was 60%. Histological staining of toluidine blue in specimens of chronic gastritis revealed staining metachromasia of staining of goblet cells, but after chondroitin ABC digestion, metachromasia staining pattern almost diminished. In congestive gastropathy, metachromasia of goblet cells was very weak. Furthermore the amount of chondroitin sulfate A B C in gastric juice of congestive gastropathy was lower than that chronic gastritis (decrease to about 40%). [Discussion] Toluidine blue is famous for metachromasia staining for chondroitin sulfate. Patients with chronic gastritis more than 50 years old, have almost intestinal metaplasia. Therefore, chondroitin sulfate was secreted from goblet cells into gastric juice. However, in patients with congestive gastropathy the amount of chondroitin sulfate in gastric juice is considerably less in congestive gastropathy than chronic gastritis, because the production of chondroitin sulfate might be inhibited by congestion. [Conclusion] It was strongly suggestive that the gastric production of chondroitin sulfate in congestive gastropathy might be inhibited because of congestion. The fact was also certified using endoscopic toluidine blue spraying and useful for the diagnosis.


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40% of the first VB episodes in cirrhotics occur within the first 6 mths of observation [J. Hepatol. 18 (5) 1993]. This study was aimed to identify the risk factors for early and late occurrence of VB through a multivariate analysis. We studied 155 consecutive cirrhotics with varices and without previous VB. At entry and after 6 months intervals the severity of liver disease and the appearance of varices were assessed according to the Child-Cambridge classification and the JRSHP endoscopic rules. During a mean f u period of 25.0 ( ± 17.5) mths 24% of the patients had VB, 38% did so after within 6 mths. The VB rates were estimated at each 6 mths interval by life tables, Breslow (early differences) and Mantel-Cox test (overall differences). The table shows the relation between VB: varices size (VS), cherry spot (CSS), ascites, tricuspid insufficiency with the early and overall occurrence of VB through the study period. During f u period 28% of F1, in 27% of F2 and in 6% of F3 varices. The actual overall rate of VB for those that ultimately had F1, F2 and F3 was: 3%, 20%, 70%. The cumulative rate of early VB (<6 mths) was 50% in F3 varices present from entry and 16% in F3 varices developed before TIPS Child class was C in 2, B in 4 and A in 14 and no patient had a previous history of HE. All patients received lactulose therapy in the previous week and thereafter. The PSE index according to Conn was assessed before TIPS and after 7, 30, 90 days and then every 3 months. All episodes of HE which occurred at any time were assessed and recorded. 3 patients died before the first month, 17 completed 3 months and 13 completed 6 months of follow up. During the first 3 months after TIPS 1/20 patients (65%) had one or more episodes of HE. HE reached grade III–V in 5 patients. PSE index was higher than 0.25 in at least 2 consecutive evaluations in 17 patients (28%), indicating chronic HE. Several variables were tested but failed to correlate to the development of HE, these were age, etiology of liver disease, and the Child class, Conn index, ammonia levels, galactose elimination capacity and liver volume before TIPS, the stent diameter and the post-TIPS portal systemic gradient. The 2 m index was significantly correlated to the mean velocity of blood flow into the stent at 4 weeks (r = 0.65; p < 0.02). Between 3 and 6 months of follow-up 4/13 patients (31%) had episodes of HE. In 3 of these patients a stable impairment of PSE index was found. In conclusion the incidence of episodic HE is high after TIPS but tends to drop after the first 3 months. A stable chronic elevation in the PSE index is still present in 23% of patients at 6 months although the alteration in mental state never exceeded grade I.
during f1 (P = 0.04); 8% in F2 present from entry and 0% in those developed during f2 (P = ns). The figures for late (>6 mths) V1 were 67% for F3 from entry and 70% for F3 during F1 (P = ns), 50% for F2 at entry and 34% for F2 during F1 (P = ns). Conclusions: (1) Univariate analysis V1 seems to be the most important variable in predicting the first V1. (2) Different treatment strategies should be used for F2 and F3 varices. (3) Regular endoscopic f1 is needed to improve the prediction criteria for the first V1.

### Long Description:

#### 1660 Non-Shunt Surgery for Bleeding Varices. 12 Years Experience

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During the period from 1979 to 1991, 596 cases of splenectomy and devascularisation were done electively for portal hypertension patients with history of moderate, massive or repeated attacks of haematemesis and or melena (elective group). 41 cases of the same procedure were done for patients with acute variceal hemorrhage which failed to be controlled with emergency sclerotherapy (emergency group). All patients were Child A & B. The mean age of the patients was 34.8 ± 10.5. Male to female ratio was 9:1. Complete liver functions, kidney functions, complete blood picture and upper gastro intestinal endoscopy were done for all patients. Needle liver biopsy was done for 541 patients from the elective group. The pathology was schistosomal in 36.8%, mixed schistosomal and cirrhosis in 33.4% and non-schistosomal in 29.8%. The operation was done by the technique of Hassab (1962). Splenectomy & devascularisation gives immediate control of bleeding in 92.7% of emergency group. Hospital Mortality was 3.8% ± 2.2% in both groups respectively. All patients were followed up for 50 ± 31 month. Recurrent rebleeding was 15.3% ± 17% in both groups respectively. Encephalopathy developed in 25% in both groups. Late mortality was 8.65% ± 9.7% in both groups respectively. We concluded that splenectomy & devascularisation was able to control bleeding both in emergency and elective cases with acceptable recurrence rate of rebleeding and low rate of encephalopathy.

#### 1661 Portal Haemodynamic Response to a Very Low-Dose Nitroglycerin in Cirrhosis


A low dose of nitroglycerin (NTG) predominantly dilates the venous system. Portal haemodynamic responses to a very low dose of NTG were studied in patients with portal hypertension and cirrhosis, compared with those to an acute dose of coronary heart disease. A 0.15 mg of NTG was sublingually given to 10 patients (LDG) and a 0.3 mg to another 10 patients (UDG). Haemodynamic measurements under the hepatic and right cardiac catherization were carried out before and 5 min after NTG administration. Wedged hepatic venous pressure (WHVP) reduced after NTG by 8%; P < 0.01 in LDG, and by 15%; P < 0.01 in UDG. Hepatic blood flow with ICG did not change in both groups. In LDG, axysos blood flow (AZF) did not change in contrast to a significant decrease by 11%; P < 0.05 in UDG. Mean arterial pressure fell by 4%; P < 0.05 in LDG and by 18%; P < 0.05 in UDG. Cardiac index did not change in LDG, but it decreased by 12%; P < 0.05 in UDG. In LDG as well as UDG, mean pulmonary arterial pressure and pulmonary capillary wedge pressure significantly fell and the magnitude of these falls in both groups were same. In UDG, a correlation between changes in WHVP and AZF (r = 0.7, P < 0.05) was observed. This suggested that splanchic vasoconstriction mediated by high-pressure, rather than low-pressure, baroreceptor reflex mainly contributed to a decrease in portal venous blood flow, resulting in a WHVP reduction. Whereas, a slight but significant fall of WHVP induced by a very low dose of nitroglycerin might be due to venodilation including hepatic vascular bed.

#### 1662 Flow and Intestinal Transport in Chronic Biliary Anomalostases


**Aim of the study:** Disadvantages of Roux-Y biliary anomostases are duodenal bypass of bile and the lack of an endoscopic access to the biliary anomostos. Recently hepatico-jejunoduodenal interposition (HJD-IP) has been propagated to overcome these problems. The aim of our study was to investigate the changes in bile flow in these procedures.

**Methods:** 15 cholecystectomized mongrel dogs were operated as follows: RY-BA using a 30 cm jejunal loop (n = 5), HJD-IP with a 15 cm jejunal segment (n = 5), no additional procedure (control-group, n = 5). Four months postoperatively all animals underwent a hepato-biliary scintigraphy using 99m Tc Hepatobid® and a Picker Dyna-Camera-4.

**Results:** Region of interest (ROI) were liver, bile ducts, anastomotic side of the jejunal loop, distal part of the ROY-loop, Treitz ligament, gastric antrum and duodenum.

**Conclusion:** Hepatic uptake of the tracer, that means time of max. activity (Tm) in the liver field was delayed in RY-BA (13.2 min) in comparison to the controls (9.0 min). This delay was even more pronounced in HJD-IP (23.0 min, P < 0.05). Trm in the extrahepatic bile ducts showed a similar delay in RY-BA and HJD-IP (40.0-42.6 min versus 20.0 min for the controls). Initial evacuation into the loop (T1) was also similar in both biliary anomostoses (33.2-33.5 min) but maximum of activity (Tm) appeared in the proximal part of RY-BA more late (71.2 min) than in the HJD-IP group (58.5 min). On the other hand percentage of applied activity in RY-BA loops (50.1%) exceeded the value in the HJD-IP loop (41.8%) indicating cumulative excretion of bile in the RY-BA loop. Transport through the Roy-Y loop lasted as long as bile flow from the HJD-IP loop to Treitz ligament. Ti at the ileo-cecal region showed a similar delay in both groups (123.4-127.0 min) compared with the controls (95.0 min). Bile reflux into the gastric antrum was a regular phenomenon in HJD-IP but not in RY-BA animals.

**Conclusion:** Consequences of chronic HJD-IP in dogs are a significant disturbance of hepatic bile uptake and recurrent duodeno-gastric bile reflux. RY-BA show a more normal liver function but a pronounced stasis of bile in the Roy-Y loop. Chronic hepatic inflammation following HJD-IP and motility disorders in the Roy-Y loop in RY-BA may be the underlying pathophysiological mechanisms.

**1663 The Mechanism of Pentagastrin Related Inhibition of Small Bowel Bioelectric Activity – An Experimental Study on the Rat**

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It has been shown that large doses of Pentagastrin (PG, 0.06–0.10 mg/kg BW) brings about the inhibition of small bowel bioelectric activity (SBEA) which is suspected to be caused by a decrease in blood flow due to vasoconstriction. Our aim was to investigate the mechanism of action of PG and the correlation of its effects with the O2 balance in the bowel, which can be of value as an important prognostic sign in various surgical conditions of the intestine, especially in small bowel transplantation.

**Methods:** The study was conducted in 15 murine small bowels in vivo and in vitro. The latter in conditions of bowel perfusion in oxygenated (pO2 = 450 mm Hg) and anoxogenated (pO2 = 100 mm Hg) Krebs solution (KES) at 37°C. Under Topiental narcosis in 7 rats (control group) the native SBEA and its reaction to intravenous PG administration (0.06 mg/kg BW) were registered with silver bipolar electrodes of the clip type. Then 5 cm of the central ileum was isolated and placed in unoxogenated KES. The SBEA and the PG effect were recorded. Afterwards the bowel was transferred to oxygenated KES and the same procedure was repeated. 8 rats (test group) were saturated with an antihypoxant substance (carotene like substance, 5 mg/kg BW) by intraperitoneal administration 30 min prior to laparotomy and procedures were reported as mentioned above.

**Results:** In vivo the SBEA readily vanished to a straight line on PG administration in the control group. This effect was absent in the test group. In hypoxia in vitro the control group again showed a vanishing tendency of the SBEA on PG, while the test group documented no change. In normoxia in vitro the control group showed a decrease in the amplitude and the frequency parameters of the SBEA on PG, but not to the extent of vanishing. The test group, here, recorded an increase in the SBEA values on PG.

**Conclusion:** We conclude that the reaction of the SBEA to high doses of PG is related to the direct action of PG on the smooth musculature and activation of the intramural neural elements through the decrease in the oxygen uptake by the tissue. We suppose that a "high-dose PG test" may serve as an intraoperative test of the bowel in surgical interventions and transplantation.