Gastric potential difference and pH in ulcer patients and normal volunteers during Stroop’s colour word conflict test

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SUMMARY Whether mental stress is important in the pathogenesis of gastric mucosal disorders is not clearly established. This study investigated the relationship between sympathetic activation caused by the Stroop’s colour word conflict test and gastric mucosal function, monitored by measuring the gastric mucosal electrical potential difference (PD). In 13 healthy volunteers and 12 duodenal ulcer patients gastric PD, pH, and heart rate were measured continuously during basal conditions, during mental stress evoked by the Stroop’s colour word conflict test, and after return to basal conditions. The volunteers fell into two groups: In seven no sympathetic activation was elicited as no changes in heart rate were demonstrated. Gastric pH was unchanged, and PD increased slightly. Sympathetic activation was elicited in the other six with increased heart rate by 18 (6) beats per min. Potential difference declined significantly during sympathetic activation (ΔPD = −5 (2) mV, p < 0.05). Gastric pH increased. Eleven of 12 ulcer patients had sympathetic activation accompanied by a decline in PD, and an increased pH. Sympathetic activation in ulcer patients and volunteers impaired gastric mucosal function, as shown by a decline in gastric PD.

The pathogenesis of chronic gastric mucosal diseases is still under debate, but its relationship to mental stress has gained support. Several studies have shown that duodenal ulcer patients do not experience more severe or more stressful life events than controls, but the ulcer patients may have a lower threshold for perceiving situations as stressful. A relationship has been found between experimentally induced centrally mediated autonomic stimulation ('stress') and changed gastrointestinal functions in terms of motility and intestinal transit. The aim of this study is to investigate whether mental activation induced by the Stroop’s colour word conflict test had a direct effect on the gastric mucosal function evaluated by measuring the gastric potential difference (PD) and pH.

Methods

PATIENTS AND VOLUNTEERS Thirteen healthy volunteers, six women, with a mean age of 27 years (range 22 to 47) served as controls. None had a history of gastrointestinal disease or dyspeptic symptoms. Twelve patients with chronic duodenal ulcer disease, four women, with a mean age of 36 years (range 21 to 54) were also examined. Duodenal ulcer disease had been diagnosed by upper gastrointestinal endoscopy. The stress test was carried out within one week after endoscopy had proved that the ulcer had healed. The patients were not receiving any medication. The volunteers and ulcer patients were untrained in laboratory stress, and the two groups were comparable in this respect. The study protocol was approved by the local ethical committee.
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The test was performed, and after a rest period of further 60 minutes the investigation was terminated.

Statistical Analysis

Results are presented as mean (SD). The Wilcoxon's non-parametric pair analysis and the Friedman test were used to evaluate statistical hypothesis. p<0.05 was considered significant.

Results

Normal volunteers with 'stress response'

Six of the normal volunteers showed a response with sympathetic activation and a significant increase in the heart frequency (Δ HF=18 (6) beats/min, p<0.05). They all reported a subjective feeling of
stress. Their gastric PD and pH responses are shown in Figure 1. Potential differences declined significantly during the stress test (Δ PD = -5 (2) mV, n=6, p<0.05), and recovered after the resting period. (A change – for example, from -40 mV to -20 mV is described as a reduction in PD). The gastric pH increased during the stress test, and only partly recovered during the rest period. The magnitude of the change in PD and pH did not correlate.

NORMAL VOLUNTEERS WITHOUT ‘STRESS RESPONSE’

In seven of the normal volunteers Stroop’s test did not elicit sympathetic activation. The heart frequency was unchanged, and they reported no subjective feeling of ‘stress’. The gastric PD and pH responses are shown in Figure 2. Potential differences increased (Δ PD = 4 (1) mV, n=7, p<0.05), and remained at this level throughout the rest of the study period. The gastric pH values did not change.

For all the normal volunteers the baseline PD values recorded 30 minutes after the PD probe had been placed was -45 (6) mV. This value is within the normal range for a comparable age matched population.7

ULCER PATIENTS

The gastric PD and pH responses for the ulcer patients are shown in Figure 3. Sympathetic activation was elicited in all but one patient. Heart frequency increased (Δ HF = 16 (6) beats/min, n=11, p<0.05), and they reported either ‘stress’ or ‘severe stress’. The one patient with unchanged heart frequency reported a feeling of disorientation. As seen in Figure 3 the 11 patients showed a significant reduction in gastric PD during Stroop’s test (Δ PD = -6 (2) mV, n=11, p<0.05). Potential differences recovered during the rest period. For some patients the gastric pH increased during the ‘stress period’, and recovered partly after rest (n=6). For seven patients no changes in the intragastric pH were observed.

The initial basal PD values: -35 (10) mV were significantly lower than that of a matched normal population: PD = -45 (8) mV.

Discussion

The Stroop’s colour word conflict test caused sympathetic activation in 11 of 12 ulcer patients and in six of 13 healthy volunteers. A significantly reduced gastric PD was found during sympathetic activation often together with an increase in gastric pH. Gastric PD measurements describe the gastric mucosal integrity and function, and a decline in PD indicates a hampered gastric mucosal ‘health state’; either because of a low resistance or a low active ion transport.7,8,10 Thus, mental stress with sympathetic activation caused a direct change in the gastric mucosal function.

This gastric PD and pH response with a decline in PD and an inhibited acid secretion is similar to the response found during gastric hypoxia and ischaemia12 in animals. The possible relationship between mental stress and gastric mucosal ischaemia was described in 1833 by William Beaumont, who observed that Alexis Sct Martin had a pale and dry stomach during ‘fear, anger, or whatever depresses or disturbs the nervous system’.13

The ulcer patients in this study had a low gastric PD value compared with controls despite a normal upper gastrointestinal endoscopy. Ulcer patients were previously reported to have a significantly increased resting plasma catecholamine level,14 and in another study ulcer patients were reported to have a low gastric mucosal blood flow.15 Thus, during stress episodes blood flow may further decline, and this would enhance the effects of the noxious substances usually present in the stomach (acid, bile, pepsin, etc). It has been claimed that ulcer patients do not experience an increased number of stressful life situations compared with controls, but without being more stressful everyday life in these patients may provoke an increased number of stress episodes.16 These frequent stress episodes could be a cofactor in the pathogenesis of gastric mucosal disease.

It can only be hypothesised that the gastric PD and pH changes during mental stress are caused by ischaemia. Unfortunately, only a few suitable methods for gastric mucosal blood flow determinations are available for in vivo human studies, as the
measurements are often influenced by changes in the acid secretory state. Duodenal bile reflux during mental stress would cause the same PD and pH response, and it cannot be excluded that the PD and pH response could be the result of bile reflux.

Sympathetic activation in patients and volunteers was assessed by the increase in heart frequency, as a relation between arterial plasma noradrenaline and heart rate was previously shown. We did not measure the arterial plasma catecholamine concentrations, as we thought it unjustified to perform arterial catheterisations in this study. Unfortunately, venous blood sampling is only misleading in this context.

In some of the normal volunteers Stroop's test did not cause sympathetic activation. The increase in PD found during Stroop's test might be caused by an initially reduced PD in the study period because the intubation procedure may have been unpleasant. Such a stress related initial PD reduction has been previously described in healthy volunteers.

In summary, sympathetic activation evoked by Stroop's colour word conflict test caused a changed gastric mucosal function in terms of a reduced gastric PD. This stress response could be elicited in nearly all the ulcer patients and in half the volunteers.

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References