Antral function in duodenal ulcer

The gastric secretion of acid, chloride and pepsin in response to antral stimuli and to insulin and maximal histamine stimulation in duodenal ulcer and controls

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Summary

A dye-dilution technique is used to compare the concentration and output of acid, chloride and pepsin in duodenal ulcer patients and controls following stimulation of the antrum with alcohol, sodium bicarbonate and peptone with the response to insulin hypoglycaemia and maximal histamine stimulation.

The mean secretory rate was higher in response to all the stimuli in duodenal ulcer patients except to sodium bicarbonate.

Following antral stimulation by peptone and sodium bicarbonate the acid and chloride concentrations rose to very high levels. The mean outputs were the same as those following maximal histamine stimulation although there were wide individual variations. There was no difference between duodenal ulcer patients and controls. The ‘neutral chloride’ concentration was much less than in the other phases.

The basal secretion and the secretions in response to insulin and histamine stimulation showed higher concentrations, as well as higher outputs of acid and chloride in duodenal ulcer patients.

Pepsin concentration in response to the various stimuli showed no difference between duodenal ulcer patients and controls and the pepsin output reflected the changes in secretory volume.

Introduction

These investigations were carried out on South Indian patients and controls. The aims were as follows:

1. To compare the response to antral stimuli with the maximal histamine response and with the response to insulin hypoglycaemia.
2. To determine whether there was any significant difference in the concentration and output of acid, chloride and pepsin in response to the various stimuli, between duodenal ulcer patients and controls.

Methods

The duodenal ulcer patients were all admitted into hospital and had active ulcers which were confirmed radiologically. The controls were either in-patients admitted for surgical conditions such as herniae or varicose vein operations and who gave no history of any dyspepsia.

It was not possible to use a continuous aspiration technique because, in order to determine the antral response, it was necessary that the antrum should be stimulated by a given amount of the stimulating fluid for a constant time. Instead the dye-dilution technique, as described by Brooks et al. (1950) was used. At the beginning of every 10 min 100 ml of fluid containing phenol red (7 mg/l) were injected into the stomach and the stomach was completely emptied by aspiration at the end of the 10-min period. From the concentration of the dye in the aspirate it was possible to calculate both the volume of juice secreted and also the amount that had emptied from the stomach. It was then possible to calculate the actual concentration and quantity of acid, chloride and pepsin that had been secreted during the period.
Both sodium bicarbonate and peptone were used as antral stimulants because there is evidence that these act on the antral mucosa in different ways (Celestin, 1967b). The effect of peptone is abolished by 3% lignocaine solution whereas the effect of sodium bicarbonate is unaltered, suggesting that peptone acts through neuro-receptors and sodium bicarbonate acts directly on the mucosal cells. For interest, in a further small series of ulcer patients and controls alcohol was used as a stimulus in the same concentration (7%) as in a standard Ehrman meal.

The subject was kept on a liquid diet for 24 hr to avoid blockage of the tube by rice particles. A radio-opaque tube was passed at 07.30 hours and positioned under X-ray control so that the distal end lay in the pyloric antrum. The patient was asked to remain lying on the left side to encourage the gastric secretions to remain in the stomach, but during the aspirations he was turned onto the right side.

(i) Basal secretion

Five runs of 100 ml of N saline with phenol red were injected through the tube and aspirated. The last three aspirates were used for analysis.

(ii) Antral phase

The stomach was washed out twice with 100 ml of water to remove any acid from the antral mucosa and then 100 ml of either 3.5% sodium bicarbonate, or 10% peptone solution or 7% alcohol containing phenol red were instilled. This was aspirated after 10 min. Then 100 ml of N saline with phenol red were injected through the tube and aspirated after a further 10 min.

(iii) Insulin phase

Soluble insulin was given intravenously (15 units for subjects weighing 100–120 lb; 10 units for subjects under 100 lb) and the stomach was emptied after 10 min, the aspirate being discarded. Three runs of 100 ml of N saline with phenol red were injected through the tube and aspirated after 10 min.

(iv) Maximal histamine stimulation

Fifty milligrams of mepyramine maleate were injected intramuscularly. After 30 min the stomach was emptied and histamine (0.04 mg/kg body weight) was given intramuscularly. Then four runs of 100 ml of N saline with phenol red were injected through the tube and aspirated after 10 min each time.

Table 1. Peak concentrations and outputs

<table>
<thead>
<tr>
<th></th>
<th>No. of Cases</th>
<th>Secretory rate (ml/min)</th>
<th>Emptying rate (ml/min)</th>
<th>Total acid Conc. (mEq/l)</th>
<th>Output Conc. (mEq/min)</th>
<th>Total chloride Conc. (mEq/l)</th>
<th>Output Conc. (mEq/min)</th>
<th>Pepsin Conc. (P.U/ml)</th>
<th>Output Conc. (P.U/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal</td>
<td>Controls</td>
<td>64</td>
<td>3.2 ±1.4</td>
<td>7.4 ±2.4</td>
<td>53 ±16</td>
<td>98 ±35</td>
<td>0.30 ±1.2</td>
<td>2.7 ±1.2</td>
<td>7.6 ±3.2</td>
</tr>
<tr>
<td></td>
<td>D. ulcer</td>
<td>97</td>
<td>3.8 ±0.7</td>
<td>7.5 ±2.8</td>
<td>64 ±0.24</td>
<td>116 ±0.42</td>
<td>0.4 ±1.4</td>
<td>9.4 ±3.9</td>
<td></td>
</tr>
<tr>
<td>Antral (NaHCO₃)</td>
<td>Controls</td>
<td>26</td>
<td>3.9 ±1.5</td>
<td>8.4 ±2.8</td>
<td>*190 ±0.74</td>
<td>214 ±0.81</td>
<td>±3.6</td>
<td>†11.05</td>
<td></td>
</tr>
<tr>
<td></td>
<td>D. ulcer</td>
<td>43</td>
<td>3.8 ±0.2</td>
<td>6.5 ±4.0</td>
<td>*196 ±0.74</td>
<td>238 ±0.75</td>
<td>±6.7</td>
<td>†12.8</td>
<td></td>
</tr>
<tr>
<td>Antral (Peptone)</td>
<td>Controls</td>
<td>24</td>
<td>3.1 ±1.2</td>
<td>7.7 ±2.4</td>
<td>165 ±0.60</td>
<td>186 ±0.68</td>
<td>±6.9</td>
<td>13.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>D. ulcer</td>
<td>41</td>
<td>4.8 ±2.3</td>
<td>7.8 ±3.1</td>
<td>159 ±0.72</td>
<td>185 ±0.83</td>
<td>±1.4 ±1.3</td>
<td>5.0 ±15</td>
<td></td>
</tr>
<tr>
<td>Antral (Alcohol)</td>
<td>Controls</td>
<td>14</td>
<td>4.6 ±1.5</td>
<td>7.8 ±2.8</td>
<td>71 ±0.32</td>
<td>127 ±0.57</td>
<td>±3.0 ±1.3</td>
<td>13.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>D. ulcer</td>
<td>13</td>
<td>6.0 ±0.53</td>
<td>8.8 ±3.1</td>
<td>80 ±0.45</td>
<td>140 ±0.81</td>
<td>±3.0 ±1.3</td>
<td>17.3</td>
<td></td>
</tr>
<tr>
<td>Insulin</td>
<td>Controls</td>
<td>63</td>
<td>5.0 ±2.4</td>
<td>8.5 ±3.5</td>
<td>77 ±0.37</td>
<td>134 ±0.70</td>
<td>±2.9 ±1.3</td>
<td>14.0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>D. ulcer</td>
<td>93</td>
<td>5.8 ±2.7</td>
<td>8.4 ±4.1</td>
<td>90 ±0.51</td>
<td>146 ±0.80</td>
<td>±3.1 ±1.3</td>
<td>16.4</td>
<td></td>
</tr>
<tr>
<td>Maximal histamine</td>
<td>Controls</td>
<td>55</td>
<td>6.8 ±2.6</td>
<td>10.9 ±4.2</td>
<td>88 ±0.63</td>
<td>131 ±0.90</td>
<td>±3.1 ±1.3</td>
<td>20.3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>D. ulcer</td>
<td>86</td>
<td>8.1 ±4.2</td>
<td>10.8 ±3.4</td>
<td>104 ±0.85</td>
<td>145 ±1.16</td>
<td>±3.2 ±1.3</td>
<td>25.1</td>
<td></td>
</tr>
</tbody>
</table>

*Deduced from chloride values. Standard deviations not given.
†Figures for 10-min period after withdrawal of NaHCO₃ solution.
TABLE 2. Statistical analysis of Table 1

<table>
<thead>
<tr>
<th>Controls/D. ulcer</th>
<th>Secretory rate (ml/min)</th>
<th>Total acid</th>
<th>Chloride</th>
<th>Pepsin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal</td>
<td>SED</td>
<td>0.2319</td>
<td>5.02</td>
<td>0.16</td>
</tr>
<tr>
<td>'t'</td>
<td>P</td>
<td>&lt;0.01</td>
<td>2.6</td>
<td>2.01</td>
</tr>
<tr>
<td>Antral (NaHCO₃)</td>
<td>SED</td>
<td>16.13</td>
<td>0.093</td>
<td>0.50</td>
</tr>
<tr>
<td>'t'</td>
<td>N.D.</td>
<td>1.5</td>
<td>0.64</td>
<td>0.4</td>
</tr>
<tr>
<td>P</td>
<td></td>
<td>&gt;0.1</td>
<td>&gt;0.1</td>
<td>&gt;0.5</td>
</tr>
<tr>
<td>Antral (peptone)</td>
<td>SED</td>
<td>0.4347</td>
<td>10.8</td>
<td>0.079</td>
</tr>
<tr>
<td>'t'</td>
<td>3.9</td>
<td>0.55</td>
<td>1.5</td>
<td>N.D.</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.001</td>
<td>&gt;0.5</td>
<td>&gt;0.1</td>
<td>N.D.</td>
</tr>
<tr>
<td>Maximal histamine</td>
<td>SED</td>
<td>0.4243</td>
<td>10.7</td>
<td>0.062</td>
</tr>
<tr>
<td>'t'</td>
<td>3.3</td>
<td>0.84</td>
<td>2.1</td>
<td>1.4</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.001</td>
<td>&gt;0.1</td>
<td>&lt;0.05</td>
<td>&gt;0.1</td>
</tr>
<tr>
<td>Insulin</td>
<td>SED</td>
<td>0.411</td>
<td>6.23</td>
<td>0.044</td>
</tr>
<tr>
<td>'t'</td>
<td>2</td>
<td>2.08</td>
<td>3.2</td>
<td>2.05</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>&lt;0.005</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

N.D. = No difference sufficient for statistical analysis.

Recording of results
For basal secretion the mean of the last three aspirates was taken. Normal saline itself is a mild antral stimulant so the figures obtained were not strictly basal, but were satisfactory for purposes of comparison. The figures recorded for antral stimulation were those obtained from the aspirate taken at the end of the period of instillation of the sodium bicarbonate or peptone or alcohol solution. During the periods of insulin and histamine stimulation the peak 10-min figures were taken and the peak 30-min maximal histamine responses were calculated.

The acid response to peptone stimulation often persisted longer, even lasting into the insulin phase, leading to higher figures for this phase than those obtained after sodium bicarbonate or alcohol stimulation.

The method used for pepsin estimation was that of Anson and Mirsky, using human plasma as the protein substrate in place of ox carboxyhaemoglobin (Sen & Roy, 1963). In the case of sodium bicarbonate stimulation it was not possible to estimate pepsin during the actual period of instillation of the sodium bicarbonate solution because the high pH inactivated the pepsin. The figures for the next 10-min period were used.

Findings

Secretory rate
Duodenal ulcer patients had a slightly but significantly higher secretory rate than the controls in response to all stimuli except sodium bicarbonate.

Emptying rate
There was no significant difference between duodenal ulcer patients and controls in respect to the emptying of fluid.

Acid secretion
The figures given are for total acid. It was not possible to estimate free acid when using sodium bicarbonate or peptone because of their neutralizing and buffering effects. The values given for total acid during sodium bicarbonate stimulation are only estimations, calculated by subtracting estimated values for 'neutral chloride' from the neutral chloride figures. The concentration of neutral
Antral function in duodenal ulcer

TABLE 3. 'Neutral' chloride concentration (mEq/ml)

<table>
<thead>
<tr>
<th></th>
<th>Basal</th>
<th>Antral Peptone</th>
<th>Alcohol</th>
<th>Insulin</th>
<th>Maximum histamine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>45</td>
<td>±32</td>
<td>15</td>
<td>±15</td>
<td>56</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duodenal ulcer</td>
<td>54</td>
<td>±36</td>
<td>17</td>
<td>±12</td>
<td>60</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SED</td>
<td>5-49</td>
<td>3-61</td>
<td>13-49</td>
<td>5-42</td>
<td>4-5</td>
</tr>
<tr>
<td>'t'</td>
<td>1-63</td>
<td>&gt;0-5</td>
<td>&gt;0-5</td>
<td>&gt;0-1</td>
<td>&gt;0-5</td>
</tr>
<tr>
<td>P</td>
<td>=0-1</td>
<td>&gt;0-5</td>
<td>&gt;0-5</td>
<td>&gt;0-1</td>
<td>&gt;0-5</td>
</tr>
</tbody>
</table>

TABLE 4

<table>
<thead>
<tr>
<th></th>
<th>Controls (56)</th>
<th>Duodenal ulcer (85)</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-min maximal histamine response (mEq HCl)</td>
<td>14-06</td>
<td>21-06</td>
</tr>
<tr>
<td>SD</td>
<td>±8-2</td>
<td>±7-7</td>
</tr>
<tr>
<td>(SED 1-378, t = 5-08, P = &lt;0-001)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

chloride in the basal and histamine-induced secretions remained fairly constant and the mean of those was taken in each case for the above calculations.

The mean total acid concentrations were slightly but significantly higher in duodenal ulcer patients in the basal secretion and following insulin and histamine stimulation. There was no significant difference with peptone or alcohol stimulation (and probably sodium bicarbonate). The mean total acid output was significantly higher in duodenal ulcer patients in all except the peptone (and probably the sodium bicarbonate) group.

The acid concentrations in response to antral stimulation by peptone (and probably sodium bicarbonate) were much higher than in response to other stimuli although the corresponding secretory volumes showed little change from basal.

Total chloride secretion

The values followed the same pattern as those for total acid. In response to sodium bicarbonate the concentration rose to even higher levels than in response to peptone but there is no significant difference between duodenal ulcer patients and controls in response to these two stimuli in either concentration or output.

The 'neutral chloride' is represented by the difference between the total chloride and total acid figures (Table 3). There is no significant difference between duodenal ulcer patients and controls in response to any of the stimuli. The concentration of neutral chloride was much less during peptone stimulation. No inverse relationship was found in individuals between the acid concentration and neutral chloride concentration in response to the various stimuli.

Pepsin secretion

There was much less variation in pepsin concentration as compared with acid concentration. It rose to the highest levels in response to peptone and sodium bicarbonate stimulation but there were no significant differences between duodenal ulcer patients and controls. The output was significantly higher in duodenal ulcer patients in the basal alcohol insulin and histamine series but not in the peptone or sodium bicarbonate series. There was no correlation in individuals between pepsin concentration and acid concentration in response to the various stimuli.

Thirty-minute maximal histamine response

The three consecutive 10-min aspirates following histamine stimulation showing the greatest total acid output were taken in each subject to calculate the mean peak 30-min 'maximal histamine response'. This was significantly higher in the duodenal ulcer patients than in the controls (Table 4).

Total acid output in response to peptone compared with maximal histamine output in the same subjects

In some subjects the total acid output in response to peptone stimulation exceeded the peak histamine output and in others it was less in both the duodenal ulcer and control series. These variations were
considerable but there was no significant difference between the mean ‘peptone’ and ‘histamine outputs’ in either series (Table 5).

Discussion and conclusions

(1) Antral stimulation

The total acid concentration in response to both peptone and probably sodium bicarbonate stimulation in this series is much higher than the response to histamine. The mean total acid outputs, however, are approximately the same. In contrast Celestin (1967a) found that the acid output following stimulation with meat extract (Bovril) was less than that following histamine stimulation, but he used a continuous intravenous histamine infusion (5 mg/hr) and waited for the concentration to reach a steady level. He also estimated the acid output in the 15-min period following the meat-extract stimulation and not during the actual period of stimulation. Giles & Clark (1966) also using intravenous histamine (0.04 mg/kg body weight/hr) and estimating the acid output in the period after meat extract stimulation had similar findings to those of Celestin (1967a).

As judged from the chloride levels, sodium bicarbonate proved to be a stronger stimulus than peptone although the response to peptone lasted for a longer time.

During peptone and sodium bicarbonate stimulation the pH of the gastric contents was above four, which is too high for the mechanism of duodenal inhibition and may have contributed to a higher acid concentration and output. The figures should still be comparable with those obtained during histamine stimulation because duodenal inhibition is no longer operative with maximal doses of histamine (Celestin, 1967b).

(2) Maximal histamine response

Many have stated that the mean 1-hr ‘maximal histamine output’ is lower in Indian duodenal ulcer patients than in Western countries (Sen & Roy, 1963; Vakil & Mulekar, 1965; Krishna Gowda, 1965; Goyal Gupta & Chuttani, 1966; Patel et al., 1967).

The mean peak 30-min ‘maximal histamine response’ in this series is 21.06 mEq HCl (SD±7.7) of total acid which is comparable with Western figures. Bhale Rao (personal communication 1967) also found a comparable mean 30-min ‘maximal output’ of 21.06 mEq HCl (SD±7.7).

Table 5. Peptone series. Comparison of total acid output (mEq/min) following peptone and maximal histamine stimulation

<table>
<thead>
<tr>
<th></th>
<th>Duodenal ulcer (39 cases)</th>
<th>Controls (24 cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peptone stimulation</td>
<td>0.75±0.36</td>
<td>0.60±0.27</td>
</tr>
<tr>
<td>Maximal histamine</td>
<td>0.84±0.22</td>
<td>0.54±0.28</td>
</tr>
<tr>
<td></td>
<td>SED = 0.079</td>
<td>SED = 0.067</td>
</tr>
<tr>
<td></td>
<td>'t' = 1.88</td>
<td>'t' = 4.4</td>
</tr>
<tr>
<td></td>
<td>P = &gt;0.05 &lt;0.1</td>
<td>P = &lt;0.001</td>
</tr>
</tbody>
</table>

Fig. 1. Scattergraph. Total acid concentration and output in controls (●) and duodenal ulcer patients (×). M = Mean; SD = standard deviation.
histamine response' of 22.63 mEq HCl (SD ± 12.3) of free acid, but he found that the 1-hr 'maximal acid output' was 23.49 mEq HCl (SD ± 13) which is lower than in Western countries. Ambasta & Vailly (1967) on the contrary, found a high 1-hr 'maximal acid output' of 41.35 mEq HCl in their cases.

Raghavachari (1959) reviewing 622 Indian patients with duodenal ulcer found that only 10% showed hyperchlorhydria. Grossman, Kirsner & Gillespie (1963), Montague (1966) and Bralow (personal communication 1967) all comment on the wide scatter of values of acid output and the wide overlap between duodenal ulcer cases and controls. Scattergraphs were drawn for all the estimations done in this series and these also showed enormous overlap so that there was no particular level of acid chloride or pepsin that could be regarded as an index of peptic ulceration. This is well illustrated by the scattergraphs for total acid concentration and output shown in Fig. 1.

(3) Total chlorides and 'neutral' chlorides

Brooks et al. (1950) felt that total chloride estimations were a valuable guide to total gastric activity. Gamble & McIver (1928) claim that the chloride concentration fluctuates within narrower limits than total acid. Toby (1936) using histamine test meals and Semb, Myers & Foss (1966) using gastrin stimulation showed that total chloride concentrations were increased in duodenal ulcer patients and reduced in gastritis and gastric ulcer. Gupta (1964) using alcohol test meals found no difference between duodenal ulcer patients and controls. The present series showed no difference in concentration of total chloride between duodenal ulcer patients and controls during antral stimulation (peptone, sodium bicarbonate or alcohol) but did show a significant difference in basal concentrations and following insulin and maximal histamine stimulation. The scattergraphs also showed noticeably less range of fluctuation for total chloride than for total acid or pepsin following maximal histamine stimulation, although not with the other stimuli.

The term 'neutral' chloride is used to describe the difference between the figures for total chloride and total acid and refers to the number of chloride ions which are in excess of hydrogen ions. The neutral chloride concentration is said to closely follow the sodium ion concentration (Gamble & McIver, 1928; Glass et al., 1952), the concentrations of potassium, calcium and magnesium ions showing but little variation. Several investigators claim that the neutral chloride and hydrochloric acid concentration vary inversely (Gamble & McIver, 1928; McLean, Griffiths & Williams, 1928; Hollander, 1932, 1938). Toby (1936) found that although the total chloride concentration was less in patients with gastric ulcer or gastritis, the 'neutral' chloride concentration was greater. In duodenal ulcer patients both were increased, but the ratio of total chloride to 'neutral' chloride remained unchanged. Glass et al. (1952) found that subjects with an acid response to insulin hypoglycaemia had an accompanying rise in total chloride concentration with a fall in 'neutral' chloride and sodium ion concentration, whereas those with no acid response showed an increase in both total chloride and 'neutral' chloride. Semb et al. (1966) showed that although the output of sodium ions does not change with gastrin and histamine stimulation, the increased volume is accompanied by a decreased concentration of sodium ions. Yamagata (1966) also found a lower concentration of sodium ions with the increased secretory volume of duodenal ulcer patients.

In the present series there was no significant difference in 'neutral chloride' concentration between duodenal ulcer patients and controls. The ratio of 'neutral' chloride concentration to total chloride concentration showed a marked fall with peptone stimulation and a lesser fall with maximal histamine stimulation (Table 3). It is interesting to note that after vagotomy the fall in acid concentration was accompanied by a big rise in 'neutral' chloride concentration (Tovey, 1968).

(4) Pepsin

Gupta (1964) claimed that raised pepsin concentrations were a more valuable index in the diagnosis of duodenal ulceration than raised acid levels. Vanzant and co-workers (Vanzant, 1933; Vanzant et al., 1936) presented similar findings. The present series, however, shows that the increased output of pepsin in duodenal ulcer patients is due to increased secretory volume and not increased concentration. This agrees with the findings of Hirschowitz (1957), Chinn, Book & Beams (1951), Chinn (1953), Book, Chinn & Beams (1952) and Yamagata (1966). The difference in concentration found by the other workers is probably due to the fact that they overlooked the greater volume of gastric juice in duodenal ulcer patients which when added to the fixed volume of a test meal would result in apparently higher concentrations than in controls.

Hirschowitz (1957) and Vibeke Bitsch et al. (1966) said that vagal stimulation gives rise to a higher concentration and output of pepsin than histamine stimulation but this was not found by Book et al. (1952) or in this present series.

Janowitz, Hollander & Winkelstein (1953) found that antral stimulation by distension did not affect pepsin secretion. It was found in this series that antral stimulation by sodium bicarbonate and peptone caused an increase in both pepsin concentration
and output, although the increase in output was less than with maximal histamine stimulation.

The present series agreed with the results of Chinn et al. (1951) in that the relationship of pepsin secretion to acid secretion is inconstant—low pepsin levels may occur with high acid levels and vice versa.

Acknowledgments

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