Gastric Acidity in the Comatose Patient*

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The occurrence of acute gastric ulceration and bleeding secondary to intracranial disease is well known to all neurosurgeons. Although the syndrome was initially described by Rokitansky, it has borne Harvey Cushing's name since his lucid description in 1932. Cushing suggested the existence of a primary parasympathetic center in the diencephalon with direct influence on the vagal nuclei of the medulla. Stimulation of this center would produce excessive vagal activity leading in turn to increased gastric motility, hypertonus, and hypersecretion of acid and pepsin. Finally, this hereditary state produces ulceration, bleeding, and perforation.

Only a few observations have been made in man to support this concept, or to shed light on the mechanisms responsible for this type of gastric hemorrhage. Davis reported one case with gastrointestinal hemorrhage after craniotomy for a brain tumor. His patient did not have an elevated gastric acid secretion. Dragstedt reported three patients studied after brain tumor surgery. Hyperacidity was present in each case, but the precise degree or duration was not specified. The limited data available seemed to make broader observations worthwhile. Therefore, the gastric acid secretion in patients with severe head injury was studied.

**Material and Method**

Ten comatose patients were studied. There were seven males and three females ranging in age from 13 to 50 years of age. Two of the patients had sustained gunshot wounds of the head; the remaining eight had closed head injuries from automobile accidents. Six of the 10 patients were decerebrate. None of the patients was placed on steroids, and none had pre-existing history of peptic ulcer disease.

The patients became part of the study on the day of injury. A nasogastric tube was inserted, and the stomach was initially lavaged with normal saline. The tube was connected to intermittent suction, and the gastric aspirate collected daily during the 12 hours between 6 p.m. and 6 a.m. The volume of aspirate was measured, and the free acid content determined. The free acid secretion was recorded in "clinical units," or "degrees," which are the ml of 0.1N NaOH required to neutralize 100 ml gastric aspirate with Topfer's reagent as indicator. Total free hydrochloric acid is expressed in mEq and is the product of the "clinical units" or "degrees" and the volume, in liters, secreted over 12 hours. This method of gastric acid analysis is commonly used in gastrointestinal surgical practice. Patients suspected of having peptic ulcers, hiatus hernia, or gastric malignancy, are studied in this way. Thus, since the normal values of acid secretion obtained by this technique are well known, its use without modification seemed appropriate in a preliminary study of this nature. The normal individual secretes an average of 18 mEq per 12 hours. Values above 20 mEq are consistently seen in chronic peptic ulcer disease.

Usually, collection of gastric juice was stopped by the tenth day for various reasons. If the patient began to bleed, treatment obviously interfered with collection. Occasionally, the gastric bleeding was easily controlled, and collection could be continued. The diagnosis of bleeding was made when blood appeared in the nasogastric tube, when a fall in the patient's blood hemoglobin content was seen, or when melena was detected. As soon as bleeding was noted, gastric lavage with cold normal saline was begun. If this did not readily control the hemorrhage, the patient was placed on intramuscular parasympathetic blocking agents and antacid irrigation of the stomach. Blood was given as necessary. Gastric surgery was not performed on any patient in this series.

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TABLE 1

Acid secretion in six decerebrate comatose patients

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<th>Case No.</th>
<th>Day after Admission</th>
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<tbody>
<tr>
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<tr>
<td>1</td>
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<tr>
<td>2</td>
<td>18</td>
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<tr>
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<td>5</td>
<td>29</td>
</tr>
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<td>6</td>
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* Day gastric bleeding first noted.
† Collection discontinued because of treatment.
‡ Collection discontinued because of tube feeding.

Results

Table 1 shows the acid secretion in the decerebrate patients. In all but one patient, the values fell in the hypersecretion range, consistently approaching those seen in chronic peptic ulcer disease. One patient yielded acid levels in the range of the Zollinger-Ellison syndrome.\(^1\) Five of the six patients bled from the gastrointestinal tract. The one patient who did not bleed was achlorhydric. In two patients, collections were discounted because extensive treatment was necessary to control their bleeding (Cases 2 and 5), and in three, tube feedings were begun (Cases 3, 4, and 6). One patient in this group, who had experienced gastric hemorrhage, died; multiple small gastric ulcers and hemorrhages were found at autopsy.

Table 2 summarizes the data obtained in comatose patients who were not decerebrate. Although some values are in the hypersecretor range, most are not. Only one patient bled; this occurred on the eighth day following injury. Two days before the hemorrhage, a cerebral arteriogram was done because of apparent deterioration of her condition. This tendency to bleed develops quite rapidly; five of six patients who bled did so within the first 6 days of injury. Davis, et al.,\(^2\) noted

TABLE 2

Acid secretion in four non-decerebrate comatose patients

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<thead>
<tr>
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</tbody>
</table>

* Day gastric bleeding first noted.
† Collection discontinued because of tube feeding.
Gastric Acidity in the Comatose Patient

this type of bleeding by the sixth to eighth day in 48 patients following intracranial surgery for tumors. French, et al., observed that the bleeding in their 17 cases was manifest by the eighth day following the acute CNS disturbance.

No explanation of the hyperacidity observed is possible from the present data, nor is there any indication that hyperacidity alone is responsible for the gastric bleeding in our patients. However, a definite pattern has emerged. Severe head injury resulting in coma is commonly associated with gastric hyperacidity, particularly if decerebrate rigidity is present. This hyperacidity correlates roughly with the development of gastric hemorrhage.

It is simple to determine gastric acidity in patients with severe head injury. We feel it should be done in all such patients, particularly those who are decerebrate. If the hyperacidity lasts more than 2 or 3 days, treatment with antacids, gastric lavage, and parasympathetic blocking agents seems warranted.

Summary

Gastric hyperacidity has been demonstrated in a series of patients comatose from severe head injury. The greatest secretion of acid occurred in the comatose patients who exhibited decerebrate rigidity. No patient who was treated with antacids and parasympathetic blocking agents required gastric surgery to control the bleeding. It is recommended that gastric acidity be monitored in comatose patients and that, if hyperacidity persists, antacid therapy be instituted.

References

10. Rokitansky, C. (Cited by Cushing, H., see reference 2.)