Comment

THE STOMACH IN IRON-DEFICIENCY ANAEMIA

We would like to comment on the interesting papers by Desai, Mehta, Borkar, and Jeejeebhoy (1968) and by Stone (1968) which study gastric acid secretion in iron-deficiency anaemia. In the paper by Desai et al there was no rise in acid secretion 72 hours after an infusion of iron, although the authors state that in patients under the age of 30, and in those patients with an acid output between 5 and 15 m-equiv/hr there was some increase but this did not reach significant levels. Of six patients retested six to eight weeks later, three showed a significant rise in acid output. In the paper by Stone (1968) nine out of 21 patients showed an increase in acid secretion on retesting at five to seven months. The findings in these papers confirm and extend previous work on the subject (Delamore and Shearman, 1965; Shearman, Delamore, and Gardner, 1966; Jacobs, Lawrie, Entwistle, and Campbell, 1966). However, the conclusions reached in the two papers are open to other interpretations.

One of the more constant findings in the many studies on iron-deficient patients is the irreversibility of the gastric mucosal lesion. Thus the increased acid secretion in some patients must be due to an improved secretory performance by the remaining parietal cells, and the mechanism for this may be repletion of iron-containing enzyme systems (Delamore and Shearman, 1965). The depletion of these systems has not been demonstrated in human gastric mucosa but it undoubtedly occurs in the severely iron-deficient rat (Shearman, Floch, Herskovic, Levine, and Spiro, 1967). In the paper by Desai et al (1968), the absence of acid increase at 72 hours is in itself no evidence for the failure of enzyme repletion. The authors fail to point out that repletion is likely to depend upon the replacement of parietal cells, the turnover of which is thought to be extremely slow (Lipkin, Sherlock, and Bell, 1963; MacDonald, Trier, and Everett, 1964). It is of importance that repletion of cytochromes in the intestinal mucosa of iron-deficient rats (Dallman and Schwartz, 1965) and children (Dallman, Sunshine, and Leonard, 1967) depends on cell renewal. On the other hand, improvement in acid secretion at a later time obscures the answer to whether the anaemia or the iron deficiency was the operative factor. In this respect the effects of other forms of anaemia on acid secretion may be important. Table I illustrates the gastric secretory findings on two patients who had anaemia and were deficient in folic acid.

PATIENT 1 Male aged 59 at the time of diagnosis. For many years his epilepsy was treated with phenobarbital 30 mg, tid, and phenytoin 100 mg, tid; Hb was 4.6 g% and MCHC 32%. Marrow was megaloblastic with abundant iron and serum vitamin B<sub>12</sub> was at the lower limit of normal for our laboratory. Schilling test showed 13% recovery in 24 hours. Serum folate was 1.9 mµg/ml (normal 4.5 to 18.0 mµg/ml). There was a full haematological response to folic acid. The improvement in acid and intrinsic factor output is shown in Table I. Anti-convulsant treatment has continued.

PATIENT 2 Male aged 43 at the time of diagnosis of sideroblastic anaemia. Hb was 63 g%. Serum folate was 2.8 mµg/ml. He was treated with folic acid and pyridoxine and Hb at the time of the second gastric function test was 10.6 g%.

In both patients improvement in acid secretion has occurred after correction of the anaemia, and in case 1 there has been an increased pepsin and intrinsic factor output. In addition, apart from these anaemic states, other patients may show recovery in acid secretion, for example, after treatment in thyrotoxicosis (Bock and Witts, 1963) and tropical sprue (Vaish, Sampathkumar, Jacob, and Baker, 1965) and also the apparently normal patient (Weir, 1967). Thus it is possible that many factors are involved in any one case.

The paper by Stone (1968) again encounters the main

<table>
<thead>
<tr>
<th>TABLE I</th>
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<tbody>
<tr>
<td>GASTRIC FUNCTION TESTS ON TWO PATIENTS WITH ANAEMIA AND FOLIC ACID DEFICIENCY</td>
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<tr>
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<tr>
<td><strong>Patient 1</strong></td>
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<tr>
<td><strong>Date</strong></td>
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<tr>
<td>15.12.65</td>
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<tr>
<td><strong>Stimulant</strong></td>
</tr>
<tr>
<td>Volume in post-histamine or post-gastrin hour (ml)</td>
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<tr>
<td>Minimum pH in any 10-minute sample</td>
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<tr>
<td>Total acid (m-equiv)</td>
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<tr>
<td>Maximum concentration (m-equiv/l)</td>
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<tr>
<td>Total intrinsic factor in post-histamine or post-gastrin hour (mg)</td>
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<tr>
<td>Total pepsin in post-histamine or post-gastrin hour (mg)</td>
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1Titration to pH 7
2By the method of Ardeman and Chanarin (1963).
3By the method of Hunt (1948) as modified by Bitsch (1966).
difficulty that has arisen in many previous communications, namely, the definition of ‘idiopathic’ because in retrospect it is impossible to assess menstrual loss or dietary deficiency of iron. Many patients who are classified as having primary achlorhydria (Delamore and Shearman, 1965) may have had blood loss, eg, menstruation, or may develop a source of blood loss as a secondary factor. Nevertheless, the study of many differing groups of patients by several groups of workers (Coghill, 1960; Shearman et al, 1966; Wright, Whitehead, Wangel, Salem, and Schiller, 1966; Cowan, Joseph, and Satija, 1966) lends support to the idea that gastritis may cause the iron deficiency in some cases. Such difficulties in classification make family studies important and these tend to associate pernicious anaemia with idiopathic iron-deficiency anaemia. (MacLachlan and Kline, 1926; Heath, 1933; McFadyen, Goldberg, Dagg, and Anderson, 1967; Wangel, Callender, Spray, and Wright, 1968). It should be noted that iron is less well absorbed in achlorhydria (Goldberg, Lochhead, and Dagg, 1963) as is reflected in the iron deficiency that can accompany pernicious anaemia (Gibson, Kelly, and Wang, 1963).

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REFERENCES


