Prospective Hematologic Evaluation of Gastric Exclusion Surgery for Morbid Obesity

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To evaluate the long-term frequency and severity of anemia and selected vitamin and mineral deficiencies after gastric exclusion surgery for morbid obesity, the authors prospectively examined hematologic and nutritional parameters in 150 consecutive patients. These patients underwent a standardized gastric exclusion procedure during a six-year period (1976–1982) and were closely followed for up to seven years (mean, 33.2 months). Anemia developed in 36.8% of the population at a mean time from operation of 20 months. It was more frequent in women than in men (p < 0.01), and it required transfusions in 3.5% of the population. A low serum iron concentration developed in 48.6%, iron deficiency in 47.2%, a low serum vitamin B_{12} concentration in 70.1%, vitamin B_{12} deficiency in 39.6%, and RBC folate deficiency in 18% of the population. Both iron and folate deficiencies responded to oral replacement. As a result of the high frequency and severity of anemia and nutritional deficiencies noted, all gastric exclusion patients should, as a minimum, be placed on oral multivitamin preparations containing iron, folate and vitamin B_{12}. In addition, it is imperative that these patients be followed closely for the remainder of their lives with appropriate studies and replacement as necessary.

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A NEMIA IS REPORTED to occur in up to 60% of patients after partial gastrectomy.[1-5] Most series reporting results of gastric exclusion surgery for morbid obesity note an incidence of anemia of less than five per cent.[6-16] Iron, vitamin B_{12}, and folate deficiencies, which occur frequently after gastric surgery for ulcer disease,[1-5] are thought to occur infrequently after gastric exclusion surgery.[6-14] However, recent studies suggest that anemia and iron, folate and vitamin B_{12} deficiencies may be considerably more frequent after gastric exclusion surgery than previously recognized. Halverson et al.[15] noted an 18% incidence of anemia and a 20% incidence of low serum iron concentration after gastric exclusion surgery. MacLean et al.[16] reported a low serum vitamin B_{12} concentration in 24% and a low RBC folate concentration in 18% of a small group of patients after gastric surgery for morbid obesity.

Materials and Methods

The patient selection and patient population have been reported.[17] After meeting standard criteria for a gastric exclusion procedure and an initial evaluation and explanation of the procedure by the surgeon, patients were referred to the surgical metabolism clinic for an extensive outpatient examination.[17]

There were 132 women and 18 men, with an average age of 35.8 years (18–55) and 35.0 years (21–48), respectively. The entire population was 103% (SD ± 25.7) above ideal body weight with a mean initial weight of 280 lbs. (SD ± 46.8) and a mean excess weight of 140 lbs. (SD ± 45.5). The mean time from operation was 37.8 months (12–72). The average length of follow-up was 33.2 months (7.72). Three patients have been lost from follow-up, and three patients have died.[17]

All patients underwent a standardized gastric exclusion procedure. Details of perioperative and postoperative management have been described previously.[17] The procedure involved a double staple line application completely across the stomach, resulting in a 30-cc proximal pouch and an antecolic gastrojejunostomy with a 1-cm

stoma. Patients were seen at 3, 6, 12, 18, and 24 months, and then at least yearly for evaluation of metabolic parameters. Multivitamin supplements with iron (12–27 mg), B_{12} (5–9 mcg), folate (0–400 mcg) and copper (2–3 mg) were prescribed for all patients. Although most patients acknowledged taking a daily multivitamin, the specific compliance in this group of patients is unknown.

Weight loss, complications, and metabolic results have been reported.[17,18] Patients in this series, on the average, lost 75% of their excess weight and 38% of their original weight and stabilized at 30% above ideal body weight. Ninety percent of the weight loss occurred in the first 12 months. However, 80% of the patients continued to lose weight at 18 months, and 40% lost weight to 24 months. This weight loss has been maintained from 2–5 years.[17]

Analytic Methods

All blood samples were drawn after an 8–12-hour overnight fast. Assays were performed using the following methods: hemoglobin, hematocrit, and red blood cell indices–Coulter Counter (Coulter Diagnostics, Hialeah, FL); iron and unsaturated iron binding capacity—Kinetic Discrete Analyzer (American Monitor Corporation, Indianapolis, IN); vitamin B_{12}—rubertope 57 radioimmunoassay (RIA Products, Micromedics Systems, Inc., Horsham PA); red blood cell folate-^{125}I-folate radioimmunoassay (Becton–Dickenson Immunodiagnostics, Orangeburg, NY); Schilling Test (Amersham; Arlington Heights, IL); PTA—Coagamate Photo-optical Analyzer (General Diagnostics, Morris Plains, NJ); serum copper-modification of protein precipitation method of Meret and Henkin[19] using 353 Atomic Absorption Spectrophotometer (Instrumentation Laboratories, Lexington, MA). Total iron binding capacity was calculated from: unsaturated iron binding capacity (μg/dl) + serum iron (μg/dl). Transferrin concentrations were derived from total iron binding capacity using the relationship (TIBC $\times$ 0.76) + 18 = transferrin, described by Stromberg et al.[20] Iron saturation was calculated using the relationship: Iron saturation (%)

$$= (\text{serum iron } (\mu g/dl) \times 100)/\text{TIBC } (\mu g/dl).^{21}$$

Definitions

Patients with a low serum iron (<40 mcg/dl) and a normal TIBC (180–450 mcg/dl) and iron saturation (>16%) were considered iron depleted. Patients with a low serum iron concentration, elevated TIBC, and a normal hemoglobin (men, 16 ± 29/dl; women, 14 ± 2 g/dl) were defined as iron deficient without anemia. Patients with a reduced serum iron concentration, a reduced iron saturation, and a reduced hemoglobin

concentration with microcytic hypochromic indices were considered to have severe iron deficiency anemia.[20–22]

The indeterminate range for vitamin B_{12} was 150–230 pg/ml. Patients were defined as vitamin B_{12} deficient if their serum vitamin B_{12} was less than 150 pg/ml, or if their serum vitamin B_{12} concentration was less than 230 pg/ml, and they had macrocytic indices without anemia.[23–25] B_{12} deficiency anemia was defined as B_{12} concentration less than 230 pg/ml with a decreased hemoglobin concentration, macrocytic indices and a normal RBC folate concentration.[23–25] Patients with a subnormal RBC folate and normal B_{12} concentration but not anemic were defined as folate deficient. Patients with a low RBC folate and normal B_{12} concentrations with macrocytic anemia were considered to have folate deficiency anemia.

Data Analysis

Data were analyzed using Student's t-test, standard error of percentages, standard error of the mean, and Chi square analysis with Yates correction for significance. The two-tailed probability table for p < 0.05 was used. All values are reported as mean ± standard deviation unless otherwise stated.

Results

The mean preoperative hemoglobin concentration was 14.0 ± 1.0 g/dl in women and 15.6 ± 1.46 g/dl in men. The mean preoperative hematocrit was 40.6 ± 3.16% in women and 45.3 ± 3.84% in men. No patient was anemic before surgery. The mean estimated blood loss during the surgical procedure was 457 cc ± 304 cc. Twenty-two patients (15.7%) received blood transfusions either intraoperatively or after surgery. The mean hemoglobin concentration at the time of discharge was 12.3 ± 1.4 g/dl and the mean hematocrit was 36.5 ± 3.9%.

There was a significant and sustained reduction in both the mean follow-up hemoglobin concentration and hematocrit for women beginning at 6 months after surgery (Fig. 1A). This resulted in a 39.5% (51 patients) incidence of anemia in women during the follow-up period that was observed at a mean time after operation of 20 months ± 3.5 (range, 3–60 months). Twelve women (9.2%) developed a hemoglobin concentration less than 10 g/dl and five women (3.8%) required hospitalization for workup of the anemia and transfusions. In addition to menstrual blood loss, three women had a gastrointestinal source of blood loss (gastritis).[17] Following treatment, these three patients subsequently redeveloped anemia without an obvious source of blood loss.
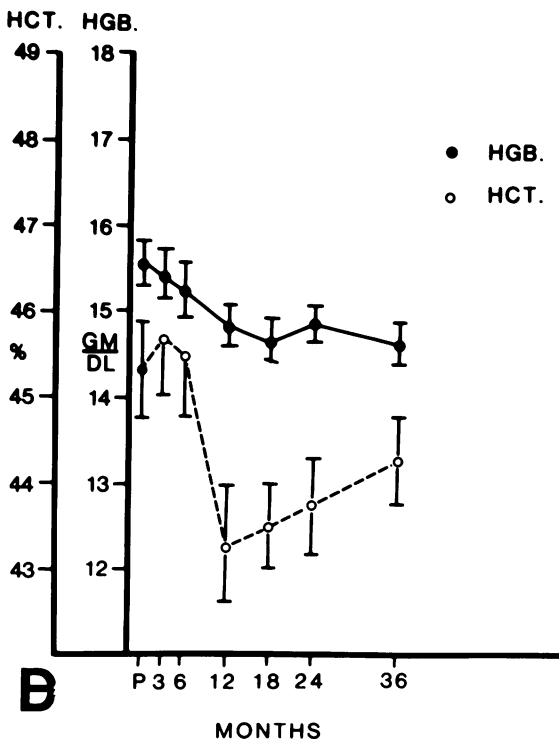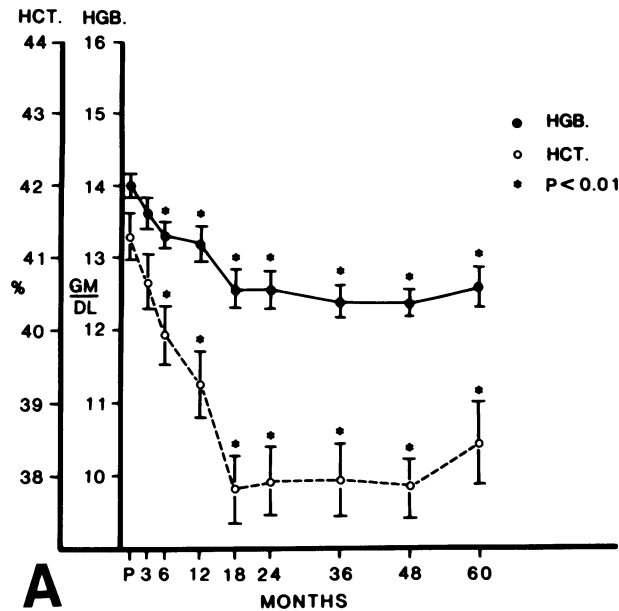
Fig. 1. *A.* The mean hemoglobins and hematocrits in women from preoperative evaluation (P) to 60 months. There was a significant decrease in both hemoglobin and hematocrit noted from 6 months to 60 months when compared with preoperative values. *B.* The mean hemoglobins and hematocrits in men from preoperative evaluation (P) to 36 months. No significant differences were seen.

There was no significant difference in the mean follow-up hemoglobin concentration and mean hematocrit for men compared with preoperative values (Fig 1B). The incidence of anemia in men was 13% (two

patients), which was significantly lower than the incidence of anemia in women (p < 0.01). In addition, one of the anemic men had a gastrointestinal source of blood loss. In total, 36.8% of the population with no abnormal source of blood loss developed anemia.

The mean preoperative serum iron concentration was 94.8 ± 36.7 mcg/dl for the entire population. Twenty-eight patients (19.3%) had a low serum iron concentration before surgery. All patients with a low serum iron concentration preoperatively exhibited a normal serum iron concentration in the early postoperative period as a result of blood transfusion, iron replacement, or dehydration. Eleven of these 28 patients (39.3%) subsequently developed a low serum iron concentration in the postoperative period. An additional 59 patients with normal preoperative serum iron concentrations developed a low concentration after surgery. In total, 70 patients (48.6%) developed a low serum iron concentration after surgery at a mean time from operation of 15.6 months ± 15.4 (range, 3–60 months). A low serum iron was significantly more frequent in women (50.8%) than in men (20%) (p < 0.001). Sixty of the 70 patients (85.7%) with a low serum iron concentration had a concurrent low serum vitamin B_{12} concentration.

The mean preoperative total iron binding capacity and derived transferrin concentration for the entire population were 370.0 ± 51.0 mcg/dl and 299.3 ± 38.80 mcg/dl, respectively. Both of these normal parameters increased significantly above preoperative values at 24 months. The mean preoperative per cent iron saturation was 25.9% ± 1%. A significant decrease in per cent iron saturation was noted at 24 months that persisted for the remainder of follow-up. Sixty-three of the 70 (90%) postoperative patients with a low serum iron had an elevated TIBC and five of the remaining seven patients with a normal TIBC had an iron saturation of less than 16%. Therefore, 68 (97.1%) of the patients with a low serum iron were considered iron deficient and two patients (2.9%) iron-depleted. Although early in this series iron-deficient patients were treated with parenteral iron, iron-deficient patients later in this series were treated with oral iron with a good response (Fig. 2).

The mean preoperative serum B_{12} concentration for the entire population was 363.1 pcg/ml ± 132.3. Thirteen patients had a low B_{12} concentration before surgery. All but three of these patients exhibited a normal vitamin B_{12} concentration by 3 months after surgery. The three patients who maintained a low serum vitamin B_{12} concentration were excluded from further statistical analysis. In total, 101 patients developed a low serum B_{12} concentration (<230 pcg/ml) during the follow-up period at a mean time from operation of 13.0 months ± 10.1 (range, 3–60 months). Sixty-one patients had a B_{12} concentration in the indeterminant range (150–230

pcg/ml) and 40 patients had a B_{12} concentration in the deficient range (<150 pcg/ml). Among the 40 patients with a B_{12} concentration less than 150 pcg/ml, 6 had microcytic indices, 18 had normocytic indices, and 16 had macrocytic indices. All patients with microcytic or normocytic indices and deficient vitamin B_{12} concentrations were iron-deficient as well. In addition, there were 17 patients with B_{12} concentrations in the indeterminant range that had macrocytic indices. Therefore, of the 101 patients (70.1%) in the entire population with a low serum B_{12} concentration, 57 (39.5%) were vitamin B_{12}-deficient. There was no difference in the frequency of low B_{12} concentrations in men (47%) versus women (72.9%). Most deficient patients were treated with parenteral B_{12}. Preliminary experience in this series with oral B_{12} supplementation with intrinsic factor suggests that it may be adequate to replace and reduce B_{12}

RESPONSE OF FOLATE DEFICIENT PATIENTS TO ORAL FOLATE REPLACEMENT

FIG. 3. The response of 10 folate deficient patients to oral folate replacement. All patients responded to oral folate replacement by 6 months.

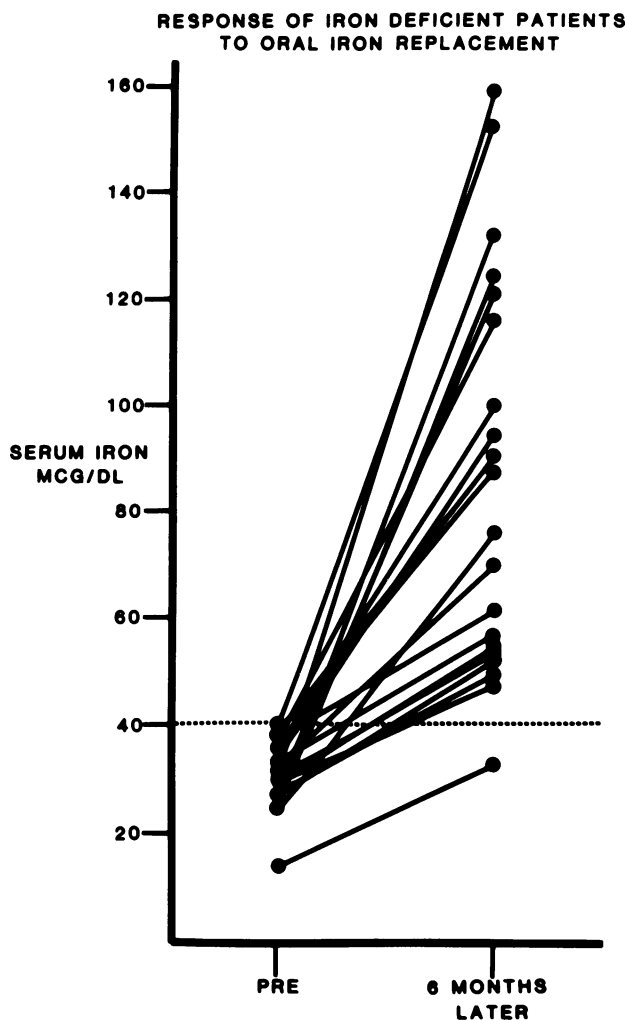## RESPONSE OF IRON DEFICIENT PATIENTS TO ORAL IRON REPLACEMENT

FIG. 2. The response of 20 iron-deficient patients to oral iron replacement. All patients responded to oral iron replacement within 6 months and all but 1 patient had nondeficient concentrations 6 months after the initiation of therapy.
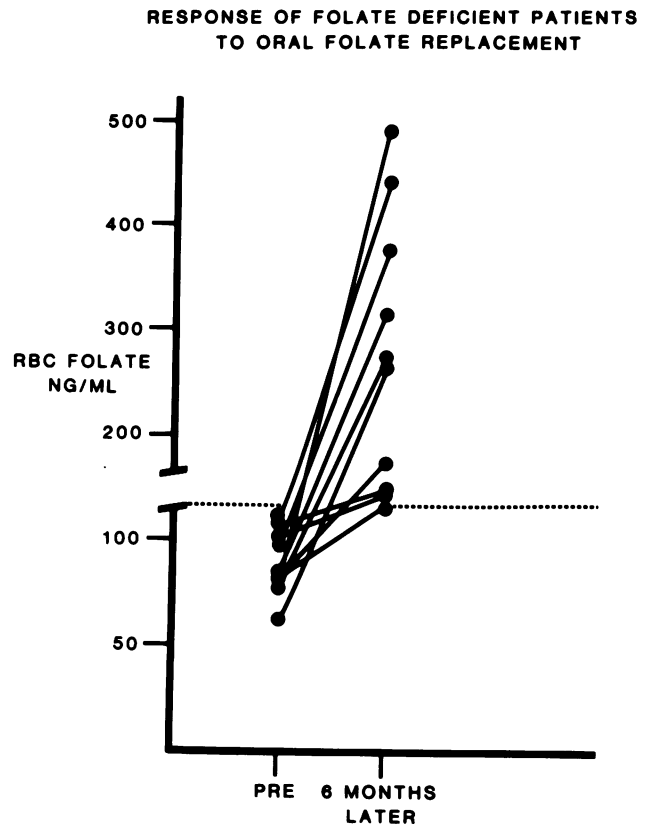
deficiency. This is in agreement with the findings of Schilling et al.[27]

Twenty patients with a low B_{12} concentration underwent a Schilling's test after surgery. The mean ratio of B_{12} absorption with intrinsic factor versus B_{12} absorption without intrinsic factor was 1.1 ± 0.4. One patient was known to have pernicious anemia before surgery. The remaining 19 patients had a normal ratio. In addition, the percentages of absorbed cyanocobalamin with intrinsic factor and without intrinsic factor were in the normal range.

RBC folate concentrations were not measured before surgery. After surgery, 26 patients (18%) were found to have a low red cell folate concentration. All folate deficient patients responded to oral folate supplementation (Fig. 3).

Microcytic anemia developed in 26 patients (18.0%). All of these patients were iron-deficient. In addition, 16 had a low serum B_{12} concentration alone or a low serum B_{12} and a low RBC folate concentration. Normocytic anemia developed in 17 patients (11.8%). Two patients in this group had normal concentrations of serum iron, B_{12}, and RBC folate, whereas the remaining 15 patients were iron-deficient. In addition, 8 of the 15 patients

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TABLE 1. *Frequency of Anemia and Vitamin Deficiencies after Gastric Surgery for Morbid Obesity*

Author	No. Patients	Maximum and (Mean) Follow-up	Anemia	Low Serum Iron	Low Serum Vitamin B_{12}	Low RBC Folate	Low Thiamine and B Complex	Low Vitamin K
Mason and Printen[6-8]	830	(—)	2.4%	2.4%*	—	—	1.2%	—
Griffen[9]	402	(—)	5%	4.2%*	0.8%*	—	—	—
Buckalter[10]	19	2 yr (—)	Mild and infrequent	—	—	—	—	—
Peltier[11]	400	20 mos (—)	—	—	1%	—	—	—
Linner[12]	227	4 yr (—)	3.1%	—	—	—	—	—
Knecht[13]	171	8 yr (—)	2.9%	—	—	—	1.2%	—
Hartford[14]	50	4 yr (—)	12%	12%*	—	—	—	—
Halverson[15]	69	2 yr (—)	18%	20%	26%	9%	—	20%
MacLean[16]	17	5 yr (28.5 mos)	0	—	24%	18%	50%	—
Present	144	7 yr (23 mos)	36.8%	48.6%	70.1%	18%	—	3.5%

* Nonanemic deficiencies not stated.

with a low serum iron had a low B_{12} concentration or a low serum B_{12} and a low RBC folate concentration.

Macrocytic anemia developed in ten patients (7%). Reticulocyte counts were not obtained routinely, but all patients with macrocytic anemia had either a low B_{12} or low RBC folate. In addition, all of these patients responded to therapy. Four of the patients with macrocytic anemia had a low serum B_{12} concentration, two had a low RBC folate concentration, and four had both a low serum B_{12} concentration and a low RBC folate concentration. Macrocytosis developed in 27 patients without anemia. Twenty patients had a low serum B_{12} and a low RBC folate concentration, four had a low serum vitamin B_{12} concentration alone, and two had a low RBC folate concentration alone.

Other nutritional parameters examined included vitmain K (PTA) and serum copper, zinc, calcium, phosphorous, and magnesium concentrations. Vitamin K deficiency (PTA < 70%) developed in five patients (3.5%) at a mean time from operation of 6 months. No patient experienced any bleeding disorder as a result, and all PTA values returned spontaneously to the normal range. Serum copper and zinc concentrations were not obtained before surgery. After surgery, no patient developed a low serum copper (<85 mcg/dl) or zinc concentration (<110 mcg/dl).

The mean preoperative concentrations of calcium, phosphorous, and magnesium were 9.4 ± 0.38 mg/dl, 3.3 ± 0.5 mg/dl, and 1.6 ± 0.12 Meq/L, respectively.[18] All patients maintained normal serum calcium and magnesium concentrations during the follow-up period, and only 17 (12%) developed transient elevations of serum phosphorous concentrations. Forty-nine patients (34%) developed elevated alkaline phosphatase concentrations after surgery. These elevations have persisted in 22 patients (15.3%); fractionation reveals the primary fraction to be from bone.

Discussion

The incidence of postoperative anemia in the present series (36.8%) is considerably greater than the incidence of anemia reported by other investigators (Table 1). In addition, anemia was significantly more severe than reported previously, and it was associated frequently with combined deficiencies of folate, B_{12}, and iron (61.5%). The incidences of low serum iron concentration (48.6%), iron deficiency (47.2%), low serum B_{12} concentration (70.1%), B_{12} deficiency (39.6%), and RBC folate deficiency (18%) are also considerably greater than reported previously (Table 1). The frequency of RBC folate deficiency was the same as that noted by MacLean et al., but the latter study was composed of a small select group of patients that either had lost weight more rapidly than normal or had been admitted for malnutrition.[16] Although there was no patient selection in the present series, the closeness and length of follow-up may have played a major role in detecting a higher incidence of the deficiencies noted. In addition, the higher incidence of anemia in this series may be specific for gastric exclusion procedures involving complete diversion of food, the "gastric bypass procedures." The incidence of anemia after gastroplasty has not been reported, but Michalek has noted only a 2.6% incidence in 39 patients who underwent a gastroplasty procedure (personal communication).

The incidence of anemia and iron, B_{12}, and folate deficiencies noted in the present series are very similar to those noted after partial gastrectomy for ulcer disease. For example, Hines et al.[1] noted a 52% incidence of anemia, a 47.6% incidence of iron deficiency, a 20.2% incidence of B_{12} deficiency, and a 25.7% incidence of folate deficiency in 292 patients after partial gastrectomy. Mahmud et al.[27,28] noted a 75% incidence of low serum B_{12} concentration after partial gastrectomy, and Tovey

and Clark[2] noted a 57% incidence of iron deficiency in men and a 72.5% incidence of iron deficiency in women after partial gastrectomy. The predominance of iron deficiency anemia noted after partial gastrectomy was also detected in our series after gastric exclusion. Forty-one patients (28.5%) had either a microcytic or normocytic anemia that appeared to be the result of iron deficiency alone or iron deficiency in combination with B_{12} and folate deficiencies. Additional similarities between postgastrectomy anemia and postgastric exclusion anemia include: the lower but significant incidence of macrocytic anemia (6.9%); the more frequent occurrence of iron deficiency and anemia in women[1-3]; and the difficulty in establishing B_{12} deficiency anemia because of concurrent deficiencies of iron, B_{12}, and/or folate.[1,21]

In the present series, the frequencies of B_{12} and folate deficiencies may have been underestimated because of the use of serum B_{12} concentrations and RBC folate concentrations. Under most circumstances, serum B_{12} concentrations and RBC folate concentrations are sensitive and accurate indicators of B_{12} and folate deficiencies. However, after gastrectomy, the RBC vitamin B_{12} concentration appears to be a more sensitive index of B_{12} deficiency than the serum B_{12} concentration and in the presence of iron deficiency, the serum folate concentration appears to be a more sensitive index of folate deficiency than the RBC folate concentration.[27] The latter results from an impaired utilization of folate in iron deficient erythrocytes, leading to elevated RBC folate concentrations even in the presence of folate deficiency.[29] The incidence of iron depletion (1.4%) in this series has probably been underestimated as well, since serum ferritin concentrations were not routinely obtained. Therefore, the frequency of depleted iron stores in the presence of a normal serum iron concentration is unknown in this series.

In this series, deficient nutrition intake has undoubtedly exerted a major role in the development of iron, B_{12}, and folate deficiencies as well as in the development of anemia. Marked reductions of both iron and folate intake after gastric exclusion surgery have been documented.[30,31] The reductions in intakes of vitamins and minerals appear to result from frequent intolerance to meats and dairy products as well as from a marked reduction in meal size.

The normal Schilling tests in 95% of the B_{12} deficient patients examined and the adequate restoration of iron and folate concentrations with oral supplementation suggest that malabsorption and maldigestion are not prominent after gastric exclusion surgery. However, they may, in fact, exert a significant role in the development of the nutritional deficiencies noted. For example, despite normal Schilling tests and enhanced absorption of me-

dicinal oral iron in postgastrectomy patients, the absorption of food bound B_{12} and iron is impaired.[27] This appears to result from an absence of gastric acidity. In addition, the primary source of iron absorption, the duodenum, is bypassed by gastric exclusion surgery. This has been implicated as an important factor in the development of postgastrectomy iron deficiency. Patients with a partial gastrectomy and a Billroth I anastamosis demonstrate better iron absorption and a lower incidence of iron deficiency than patients with a Billroth II anastamosis.[3,5,32]

Blood loss also may have contributed to the development of anemia and vitamin and iron deficiencies noted in this series. The increased incidence of iron deficiency and anemia in women suggests that menstrual blood loss exerted a significant role. An abnormal blood loss was documented in only four of the anemic patients, three of whom subsequently redeveloped anemia without a source of blood loss. Therefore, a significant abnormal loss of blood did not appear to be important in the development of anemia. Similarly, since mean hemoglobin concentrations and hematocrits were unchanged from preoperative concentrations at 3 months of follow-up, it appears that operative blood loss was not a significant factor in the subsequent development of iron deficiency and anemia.

Deficiencies of iron, folate, and B_{12} developed in postgastric exclusion patients who were apparently taking multivitamin preparations containing these compounds. However, daily oral supplementation with ferrous sulfate (325 mg), folate (1 mg), and possibly B_{12} (100–500 mcg) are sufficient for reduction and replacements of these deficiencies. This discrepancy may be the result of poor patient compliance with multivitamin preparations or the result of inadequate amounts of vitamins and minerals in these preparations. Alternatively, gastric exclusion patients may be unable to absorb these cofactors when administered as a multivitamin preparation.

A striking feature of this study is the rapid onset after operation of anemia and iron, B_{12}, and folate deficiencies. Anemia developed at a mean time after operation of 20 months $\pm$ 13.5, the low serum iron concentrations developed at a mean time after operation of 15.6 months $\pm$ 15.4, and low serum B_{12} concentrations developed at a mean time after operation of 13.0 months $\pm$ 10.1. Halverson et al.[15] have noted a similar rapid onset of low serum B_{12} concentrations (20 $\pm$ 11 months) and low RBC folate concentrations (13.6 $\pm$ 6 months) after gastric exclusion surgery. In contrast, postgastrectomy deficiencies usually take years to develop. The mean time for postgastrectomy B_{12} deficiency to develop is 5 years (range, 2–10 years),[33] and the mean time for postgastrectomy iron deficiency to develop is 3 years.[3]

The reasons for the rapid onset of these deficiencies after gastric exclusion surgery are not clear, but the marked differences in the rates of development of iron, B_{12}, and folate deficiencies between gastric exclusion surgery and gastrectomy suggest that in addition to nutritional insufficiency, malabsorption, maldigestion, and blood loss, other unidentified factors may be operative in obese patients who undergo gastric exclusion surgery. Possibilities include reduced tissue stores of vitamins and minerals, impaired transport of these cofactors from storage sites to the plasma, or an absorptive defect involving these cofactors in obese patients. Alternatively, the total nutritional intake of gastric exclusion patients may be considerably less than that of partial gastrectomy patients.

In addition to the nutritional and hematologic abnormalities noted, reported similarities between gastric exclusion surgery and partial gastrectomy include early and late dumping syndrome,[14,18] afferent and efferent loop obstruction,[11,14,18] bile gastritis,[11,14,18] and an increased incidence of gallbladder disease.[18] Bone disease, a well-recognized complication of postgastrectomy, has not been evaluated to date after gastric exclusion surgery. As a result of a high frequency of subnormal vitamin D concentrations, morbidly obese individuals may be at an increased risk of developing metabolic bone disease.[35,36] In this series, all patients maintained a normal serum calcium concentration during follow-up evaluations, and only 12% developed transient elevations of serum phosphorous concentrations. However, 34% of patients developed elevated alkaline phosphatase concentrations postoperatively that have persisted in 15.3%. Fractionation reveals the primary fraction to be from bone. Since elevated alkaline phosphatase concentrations are important indicators of postgastrectomy bone disease,[37,38] these data suggest that subclinical bone disease may occur after gastric exclusion surgery and that calcium and vitamin D supplements may be beneficial postoperatively.

Conclusion

It is apparent from this study that anemia, iron deficiency, B_{12} deficiency, and folate deficiency are considerably more frequent and severe after gastric exclusion surgery than previously recognized. The incidence of these deficiencies appears to be reduced with oral supplementation. Therefore, as a minimum, all gastric exclusion patients should be placed on oral multivitamin preparations that contain iron, folate, and B_{12}. In the course of follow-up, additional oral iron, B_{12}, and folate supplementation may be necessary. In addition, oral calcium and vitamin D supplements may be needed. As a result of the interference of food with the absorption

of these cofactors in postgastrectomy patients, postgastric exclusion patients should take these preparations on an empty stomach. Finally, because of the high frequency and severity of anemia and nutritional deficiencies after gastric exclusion surgery, it is imperative that these patients be followed closely for the remainder of their lives, with appropriate studies and replacement as necessary.

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