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The Absorption of Vitamins in Disorders of the Alimentary Tract

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Space does not permit me to deal with the physiology of absorption of vitamins, but I feel that I should make some reference to the action of intestinal bacteria.

The action of intestinal bacteria

It has been shown that bacteria similar to those present in the alimentary tract, when grown under suitable conditions, can synthesize thiamine, riboflavin, nicotinic acid, pantothenic acid, biotin, p-aminobenzoic acid, vitamin B_{12} and vitamin K. By means of depleting organisms such as *Staphylococcus aureus* and *Bacterium coli* of folic acid and growing them under very strict cultural conditions, Lascelles & Woods (1952) have induced them to synthesize folic acid. However, other organisms in culture can destroy or absorb vitamins. Table 1 summarizes an experiment in which the mixed flora from human gastric contents was incubated for 48 h in two media with added cyanocobalamin*. Thereafter the amount of vitamin B_{12}* present was measured microbiologically with *Lactobacillus leichmannii* as the test organism. The two media used were nutrient broth and the medium of Burkholder (1952) which contains only dextrose and asparagine together with salts of potassium, sodium, magnesium and ammonia.

* Throughout this paper the terms cyanocobalamin and pteroylglutamic acid are used to denote the pure chemical substances as employed in therapy. The terms vitamin B_{12} and folic acid refer to the substances present in natural materials; they are not necessarily precisely the same chemical substances.

It will be seen from Table 1, that under these conditions most of the organisms investigated could absorb cyanocobalamin and that the extent of this uptake differed with the two media employed.

Table 1 *Results of experiments to establish whether mixed flora from human gastric juice removes added cyanocobalamin from culture media*

| | | Content of vitamin B_{12} in | | | |
| | | Nutrient broth | | Burkholder's medium | |
Case no.	Organisms identified	Before incubation ($m\mu g/ml.$)	After incubation ($m\mu g/ml.$)	Before incubation ($m\mu g/ml.$)	After incubation ($m\mu g/ml.$)
1	*Bacterium coli* intermediate Type II	30	8	32	24
2	*Bact. coli* Type I	36	12	33	15
3	*Bacterium aerogenes* Type I Staphylococci	32	2	29	30
4	*Bact. coli* intermediate Type II Yeasts	30	4	30	14
5	*Bact. aerogenes* Type I Staphylococci Streptococci	35	0.4	30	15
6	*Bact. coli-mutabile* Staphylococci	29	3	29	22
7	Staphylococci	32	32	28	28
8	*Bact. coli* Type I Staphylococci	32	Trace	28	14
9	Staphylococci Streptococci	31	25	29	28
10	*Bacterium cloacae* Staphylococci Streptococci	32	1	32	3
11	*Bact. coli* intermediate Type I Staphylococci Diphtheroids	28	1	32	2

The difficulty that arises in attempting to decide about the importance of any such test-tube observations is that our knowledge of the bacterial content of the small intestine is very limited. Bean & Spies (1940) suggested that vitamin deficiencies might develop in diarrhoeal states because alterations in intestinal flora would interfere with the synthesis of vitamins by bacteria. Many workers have considered the importance of intestinal bacteria in relation to vitamin-deficiency states, including the megaloblastic anaemias, but most of those who have studied the bacteriology of the alimentary tract have examined the gastric and duodenal contents and the faeces, and from their results have made surmises about the bacteriology of the small intestine. It is very difficult to carry out intubation experiments which do not involve contamination of one level of the alimentary tract with the contents of another; experiments based on sterilization with antibiotics are too indirect to be satisfactory.

The Australian workers Cregan & Hayward (1953), and later Cregan, Dunlop & Hayward (1953), have published the results of observations involving the culturing of organisms obtained at operation by inserting needles directly through the wall of the small intestine. Only a transient flora was found in normal persons,
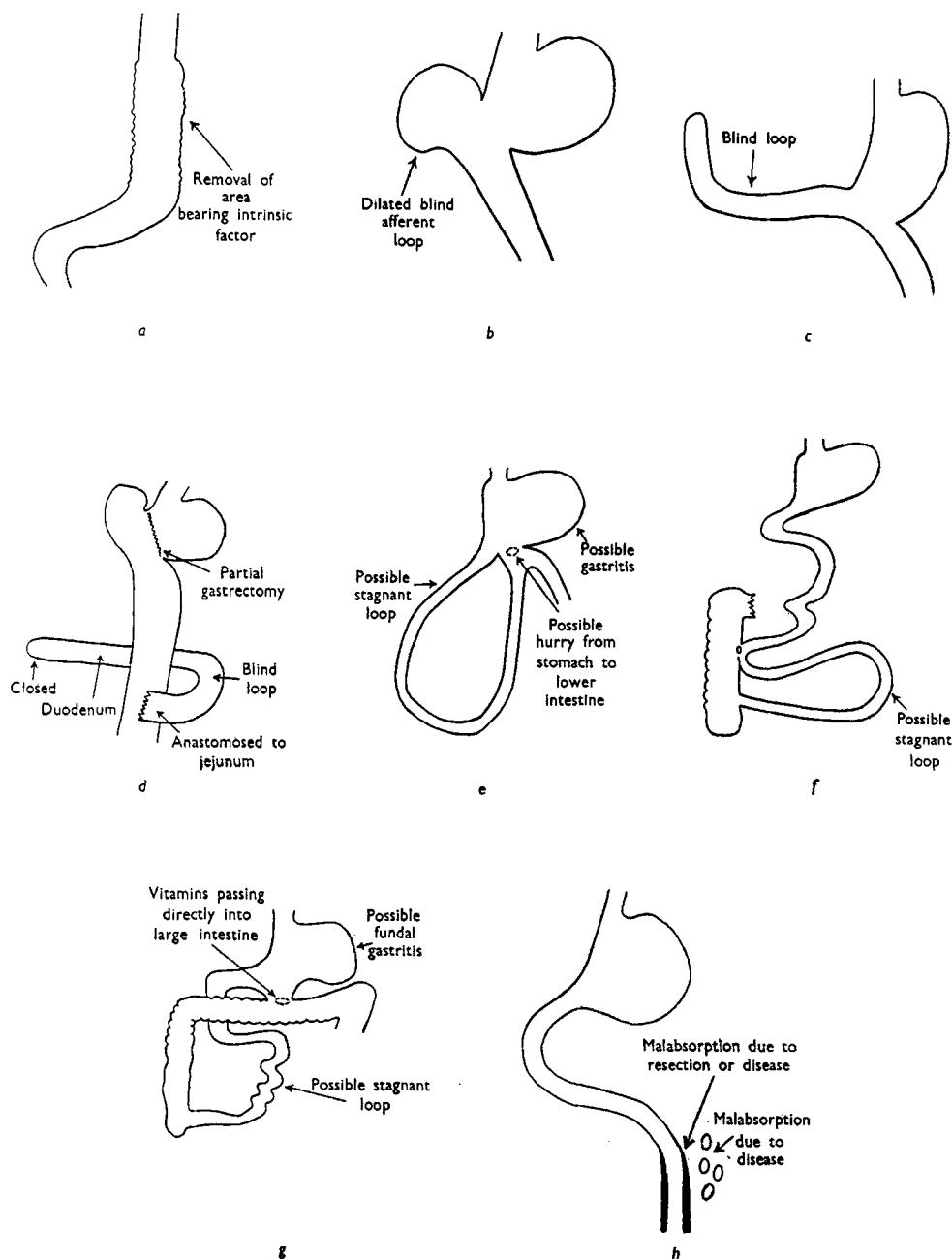
Fig. 1. Commoner mechanisms of production of vitamin deficiency in alimentary diseases. (*a*) Total gastrectomy, extensive partial gastrectomy, neoplasm, or other conditions destroying fundal tissue of the stomach. (*b*) Partial gastrectomy with dilated loop. (*c*) Polya gastrectomy with long afferent loop. (*d*) Y-shaped anastomosis. (*e*) Gastroenterostomy. (*f*) Jejuno-colic fistula. (*g*) Gastro-colic fistula. (*h*) Disease of small intestine or mesenteric glands.

confirming observations made 50 years previously by Cushing & Livingood (1900) and by Hewetson (1904). The Australian workers, as a result of similar observations on patients undergoing gastrectomy operations, pour scorn on the view that vitamin B deficiencies after gastrectomy, in sprue or in nutritional megaloblastic anaemia, are due to bacteria invading the small intestine and depriving the host of vitamins. However, although these workers showed that in a number of patients undergoing operation for gastric or duodenal ulcer or for gastric carcinoma the flora present in the upper jejunum or lower ileum was not associated with a resident flora in the middle section of the small intestine, this does not to my mind entitle them to come to any conclusion about the mechanism in idiopathic steatorrhoea and related diseases which they did not investigate. Their view may be correct but it has yet to be proved.

Mechanisms of production of vitamin deficiency in alimentary disorders

The commoner mechanisms that may be involved are shown in Fig. 1a–h. So far as we know, destruction of the area of the stomach which produces intrinsic factor results in impaired absorption of vitamin B_{12} alone as shown particularly in Fig. 1a. After various operations for peptic ulcer a blind or stagnant loop of bowel may be produced. Our knowledge of what occurs in a stagnant area of bowel is inadequate, but it is possible that destruction of vitamins by bacteria, or the production of toxins or antimetabolites may take place. In an extension of the investigations summarized in Table 1 it has been possible to show that the organisms absorbed rather than destroyed cyanocobalamin. The other chief pathological mechanisms operating in the alimentary tract include continued rapid passage of the intestinal contents or, as a result of a fistula, the by-passing of a significant area of the small intestine. Malabsorption may be found where there is disease of the small intestine or of the mesenteric glands.

Diseases and disorders associated with vitamin deficiency

It would serve little purpose in making a list of such diseases to indicate in each instance exactly which vitamin deficiencies have been reported, particularly since the reports are usually based on clinical findings which may be completely misleading. In Table 2 there is given a list of the alimentary conditions in which deficiency of some or all of the vitamins has been reasonably well reported.

In some of the conditions listed in Table 2 the deficiency may be brought about or accentuated by dietetic restrictions or prolonged loss of appetite. Since nicotinic-acid deficiency may give rise to nausea, vomiting and diarrhoea, and prolonged nausea, vomiting and diarrhoea may cause nicotinic-acid deficiency, it is obvious that, at least in this deficiency state, a vicious circle may be produced.

Tests of bodily depletion and malabsorption of vitamins

There is a lack of general agreement about the reliability of the various bio-chemical tests that have been used to measure bodily depletion of vitamins and their

Table 2 *Alimentary diseases and disorders in which vitamin deficiencies have been reported*

Prolonged nausea or vomiting	Tumours of small intestine
Osteomyelitis of the jaw and other chronic oral lesions	Strictures of small intestine
	Chylangioma mesenterica
Oesophageal strictures	Foreign body in abdominal cavity with intestinal anastomosis and blind cul-de-sac
Gastric tumours and gastric syphilis	
Peptic ulcer (ascorbic-acid and nicotinic-acid deficiency)	Chronic dysenteries
	Ulcerative colitis
Gastric neuroses	Idiopathic steatorrhoea and tropical sprue
Partial gastrectomy with long afferent loop or dilated loop	Coeliac disease
	Intestinal parasites
Other causes of blind loops	Chronic intestinal obstruction
Old gastroenterostomy or other short-circuiting operations	Overuse of cathartics, liquid paraffin, antibiotics
Gastro-jejuno-colic and other fistulas	Rectal strictures and other chronic rectal disease (perhaps with associated overuse of cathartics)
Tuberculous enteritis	
Tuberculous adenitis and reticuloses of the mesenteric glands	Chronic pancreatic disease (deficiency of vitamin A and vitamin K)
Regional jejunitis	

absorption from the alimentary tract. In a case of idiopathic steatorrhoea, for example, the tests that might be considered include those listed in Table 3.

Almost all tests of the types given in Table 3 have been criticized. In particular, measurement of the urinary excretion of a vitamin after it had been given in large dosage by mouth may give very misleading results since the output will depend upon many factors. These include the degree of absorption from the alimentary tract which may vary with the size of the dose, the extent of bodily depletion, the relative rates of urinary excretion and of uptake of the substance by the organs and tissues, the effects of renal disease, the rate and extent of metabolism of the substance in the body, and its uptake by any oedema fluid or effusion that may be present.

The differential urinary folic-acid excretion test. I have found a differential urinary folic-acid excretion test to be of great value in the diagnosis of the condition usually referred to as non-tropical sprue or idiopathic steatorrhoea and in the investigation of complex cases of megaloblastic anaemia. I want to deal at some length with the megaloblastic anaemias due to alimentary disturbances because they are in many ways easier to recognize than are other vitamin-deficiency states. The presence of megaloblasts in the bone marrow is a sign of deficiency of vitamin B_{12}, folic acid, or both. It is probable that this change is always an indication of advanced deficiency of these vitamins and possible that in pregnancy they may sometimes be seen not because of true deficiency but because of interference with folic-acid metabolism (Girdwood, 1953). Nevertheless, megaloblastic anaemia is a definite entity that can be readily recognized because megaloblasts are not found in normal marrow.

Biochemically, too, as I hope to show, there is something very definite to measure by a combination of the differential urinary folic-acid excretion test and a serum-vitamin B_{12} measurement.

Usually, but not always, if a patient is depleted of folic acid his urinary output

Table 3 *List of some biochemical investigations of vitamin absorption and of 'bodily desaturation'*

Vitamin	Test	Reference
Vitamin A	Blood vitamin A absorption tests	Yiengst & Shock (1949); Aron (1949)
Biotin	Urinary output after oral dose	Oppel (1948)
Thiamine	Urinary output after injected dose; blood pyruvic acid and lactic acid after dextrose	Williams, Mason, Power & Wilder (1943)
	Urinary output after oral dose	Friedman, Kmieciak, Keegan & Sheft (1948)
Riboflavin	Urinary output after injected dose	Axelrod, Spies & Elvehjem (1941); Najjar & Holt (1941)
Nicotinic acid	Urinary output after oral or injected dose	Najjar (1944)
	Urinary output after complex 'saturation' test	Ellinger & Hardwick (1947)
p-aminobenzoic acid	Blood levels and urinary output after oral dose	Strauss, Lowell & Finland (1941)
Pteroylglutamic acid	Differential urinary folic-acid excretion test	Girdwood (1953)
Cyanocobalamin in the presence of intrinsic factor	Serum vitamin B_{12} level (*Euglena gracilis*) (*Lb. leichmannii*)	Mollin & Ross (1953); Unglaub, Rosenthal & Goldsmith (1954); Girdwood (1954)
	Faecal excretion of orally administered cyanocobalamin labelled with radioactive cobalt	Welch, Scharf, Heinle & Meacham (1952)
	Urinary output of labelled cyanocobalamin after oral dose	Schilling (1953)
	Measurement of hepatic uptake of labelled cyanocobalamin	Glass (1954)
Ascorbic acid	Urinary output after oral dose	Harris & Abbasy (1937)
	Urinary output after injected dose	Wright (1938)
	Measurement in blood or white cells and platelets	Bessey, Lowry & Brock (1947)
Vitamin D	Serum alkaline phosphatase	
	Serum inorganic phosphorus	
	Serum calcium	
Vitamin E	Plasma-tocopherol tolerance tests	Klatskin & Krehl (1950)
Vitamin K	Determination of so-called prothrombin activity of plasma. This measures other deficiencies, including that of Factor VII	Quick (1940)

of folic acid is low after an injection of 5 mg pteroylglutamic acid. If the patient is 'saturated' with folic acid by, say, 15 mg injections of pteroylglutamic acid given daily for a week, and then the urinary excretion of folic acid after a 5 mg dose given orally is measured, it gives a very satisfactory measurement of the degree of absorption of folic acid from the alimentary tract.

The method is as follows:

(1) 5 mg pteroylglutamic acid given by subcutaneous injection. All urine collected for 24 h and its folic acid content measured microbiologically (A).

(2) 5 mg pterolyglutamic acid from the same batch given by mouth, in water. All urine collected for 24 h and its folic-acid content measured (B).

(3) The results may be expressed as percentages from the calculation $\dfrac{B}{A} \times 100$.

In normal persons the result is 70–140%. In intestinal malabsorption the result may be very low indeed. For example, in one patient who was known to have idio-

pathic steatorrhoea in adult life consequent upon coeliac disease in childhood and who had been receiving folic-acid therapy, the differential urinary folic-acid excretion test showed 0·22% absorption, while a fat balance test showed 87% absorption of fat. Another known case of idiopathic steatorrhoea with tetany but no diarrhoea had 95·8% fat absorption on a 7-day fat balance test carried out in a metabolic ward. The differential urinary folic-acid excretion test showed only 4·9% absorption.

Megaloblastic anaemia need not be present. For example, I have had patients in whom the test showed marked impairment of folic-acid absorption and in whom the presenting features were: spontaneous fractures without anaemia (malabsorption of vitamin D and of calcium), a bleeding tendency and iron-deficiency anaemia (malabsorption of vitamin K and of iron) and iron-deficiency anaemia responding only to intravenous iron (malabsorption of iron); in one there were no symptoms or signs at the age of 21 years but there was a history of coeliac disease in childhood.

None of twenty-seven adult patients proved by other means to have intestinal malabsorption has had a negative folic-acid absorption test, although I am quite prepared to find that it sometimes occurs. None of seventy control patients showed abnormal results by this test.

It will be noticed that I prefer to use the term 'intestinal malabsorption' rather than idiopathic steatorrhoea, non-tropical sprue, the sprue syndrome or the malabsorption syndrome. There are several reasons for it. When a patient has malabsorption of folic acid and of iron with no diarrhoea and with normal fat absorption, the terms 'steatorrhoea' and 'sprue' are hardly applicable. The term 'syndrome' is usually used where there is a definite pattern of symptoms; in fact here the clinical features are polymorphous. Even the word 'idiopathic' is unsatisfactory—we have had patients believed at first to have idiopathic steatorrhoea who have been shown by laparotomy or post-mortem to have tuberculous adenitis of the mesenteric glands, regional jejunitis or tumours of the small bowel.

At the moment I cannot put forward results of absorption tests for other vitamins, but investigations along similar lines in relation to other vitamins are proceeding.

Recently with the assistance of Dr Malcolm Low, I have done preliminary experiments to extend this folic-acid excretion test by adding the oral dose of pteroylglutamic acid to 50 g glucose in solution and doing a blood-sugar curve and serum folic-acid test over a period of 4 h. This procedure enables us to do two absorption tests at once. The peaks usually come at different times, the folic-acid peak being from $1\frac{1}{2}$ to 3 h after the oral dosing. So far, in three cases of intestinal malabsorption treated for several years, the glucose-tolerance curves were flat and the rises of serum folic acid at 3 h were 7, 8 and 20 mμg/ml. In thirteen controls, including three cases of megaloblastic anaemia of pregnancy and one who had steatorrhoea after partial gastrectomy, the blood-sugar curves showed no evidence of malabsorption and the serum folic-acid rise at 3 h ranged from 90 to 235 mμg/ml.

Method of vitamin therapy. From the therapeutic point of view, it would seem desirable in intestinal malabsorptive diseases to supplement orally administered pteroylglutamic-acid therapy with occasional injections of the substance. The same

Table 4 Some findings in patients who have had operations upon the stomach

Case no.	Age	Sex	Operation	Time since operation	Reason for operation	Haemo-globin (g/100 ml.)	Red blood cell count (10⁶/cu.mm)	Marrow	Serum vitamin B_{12} level (μμg/ml.)	Response to vita-min B_{12} therapy	Folic-acid absorp-tion	Fat absorp-tion (%)	Diarrhoea	Features possibly due to vitamin deficiency
1	66	M	Total gastrectomy	5 months	Gastric ulcer	13·2	4·15	Normoblastic	85	Not given	Normal	—	Absent	None
2	48	F	Total gastrectomy	7 years	Gastric ulcer	7·8	2·01	Megaloblastic	—	Positive	Normal	82	Present	Glossitis
3	51	F	Partial gastrectomy	10 years	Gastric ulcer	8·1	2·32	Intermediate erythroblasts	110	Not given	Normal	90	Absent	Glossitis
4	68	F	Partial gastrectomy	6 years	Gastric ulcer	6·8	1·59	Megaloblastic	—	Positive	—	—	Absent	None
5	50	F	Partial gastrectomy	5 years	Gastric ulcer	11·1	3·04	Megaloblastic	90	Positive	—	—	Absent	Glossitis
6	47	M	Partial gastrectomy	18 months	Duodenal ulcer	15·1	5·22	Normoblastic	250	Not given	—	—	Absent	Glossitis
7	67	F	Partial gastrectomy	4 years	Gastric ulcer	13·6	4·91	Normoblastic	140	Negative	Normal	96	Absent	Glossitis
8	53	F	Partial gastrectomy	3 years	Gastric ulcer	11·8	3·8	—	250	Negative	—	—	Absent	Glossitis Angular stomatitis
9	44	M	Duodeno-jejunostomy Partial gastrectomy	23 years 2 years	Duodenal ulcer	10·2	2·97	Normoblastic	165	Negative	Normal	82	Present	None
10	67	F	Partial gastrectomy	4 years	Gastric ulcer	13·6	4·19	Normoblastic	140	Negative	Normal	78	Present	Glossitis Dysphagia
11	63	M	Gastro-enterostomy	4 years	Duodenal ulcer	6·1	1·60	Megaloblastic	—	Positive	Normal	—	Absent	None
12	73	F	Gastro-enterostomy	8 years	Duodenal ulcer	5·8	1·66	Megaloblastic	75	Positive	Normal	—	Absent	None
13	63	F	Gastro-enterostomy	25 years	Duodenal ulcer	4·4	3·12	Normoblastic	68	Positive	Normal	—	Absent	Glossitis Angular stomatitis

is true of other vitamins. My experience, like that of Badenoch & Fourman (1954), has been that large doses of vitamin D are required, and recently Boscott & Cooke (1954) have suggested that there is an abnormal utilization or requirement of ascorbic acid. I am not one of those who believe that almost every disease known to man should be treated with vitamin E, but I wonder whether we should not investigate further the possibility of vitamin E deficiency being of importance in malabsorptive diseases. Deficiency of vitamins of the B complex are frequently seen, and vitamin B_{12} deficiency occurs occasionally.

Vitamin deficiencies after operations upon the stomach

Although steatorrhoea is not uncommon after gastrectomy or gastroenterostomy operations, clinical evidence of vitamin deficiencies is not frequently found. This is shown in a paper by Blake & Rechnitzer (1953) from the Department of Medicine of the University of Edinburgh, dealing with 179 patients.

Again, however, the vitamin deficiency that becomes easily obvious and that can be measured fairly exactly by biochemical methods is one that gives megaloblastic anaemia. This time, however, although steatorrhoea may be present simultaneously, the deficiency in my experience has been of vitamin B_{12} and not of folic acid.

The serum vitamin B_{12} level may be measured microbiologically with *Lb. leich-mannii* as the test organism. The normal range by this method is 130–600 $\mu\mu g$/ml. (Girdwood, 1954). In Table 4 there are given the findings in a series of patients referred after gastric operations because they were believed to be anaemic. They are not included in Blake & Rechnitzer's paper. A few were seen before vitamin B_{12} assays were possible.

It will be seen from Table 4 that megaloblastic anaemia after such operations is associated with a low serum level of vitamin B_{12} and responds to cyanocobalamin therapy. The lowering of the serum level of vitamin B_{12} may precede the appearance of megaloblasts in the marrow. Indeed case 3 has been maintained on pteroylglu-tamic-acid therapy for 18 months without further haematological relapse or neuro-logical complications, and her vitamin B_{12} level is now 50 $\mu\mu g$/ml. In cases 2, 7 and 8 the glossitis has not responded to therapy with multivitamin tablets, cyanoco-balamin or pteroylglutamic acid. In cases 8 and 9 the anaemia has not responded to these measures, but the latter patient had a dilated stagnant loop. In most instances loss of intrinsic factor with resultant vitamin B_{12} deficiency appears to be the primary cause of the anaemia, but other factors as shown in Fig. 1*a–h* may some-times be involved.

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Mineral Metabolism in Alimentary Disease

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Diseases of the alimentary tract affect mineral metabolism in two main ways—by deprivation or malabsorption of dietary minerals, and by loss in vomitus or faeces of the minerals contained in alimentary-tract secretions. The interference with absorption which must underlie both these mechanisms may be mechanical, as in pyloric stenosis; or may be functional, as in idiopathic steatorrhoea. The two mechanisms may indeed both be operative, as in intestinal obstruction, where food intake is valueless and at the same time large amounts of minerals are lost by vomiting and by continuous aspiration of alimentary secretions. In general, chronic alimentary disease causes mineral malnutrition mainly by deprivation and malabsorption of food minerals, whereas in acute catastrophes the effects of loss of alimentary secretions predominate. Since the mineral composition of alimentary secretions is much more akin to that of extracellular fluid than to that of cellular and skeletal tissue, the acute catastrophes cause the syndrome of dehydration, whereas chronic illness leads to a bewildering variety of mineral disturbances involving intracellular ions such as potassium and phosphate, and bone minerals such as calcium and phosphorus. Again, the distinction is not absolute, for potassium depletion is demonstrable both in acute and chronic alimentary-tract disease. Besides straightforward deprivation, malabsorption, or loss of minerals, alimentary-tract disease can influence mineral metabolism indirectly by way of general nutritional disturbance, as in the sequence: anorexia—> deficient intake of calories and proteins—> hypoproteinaemia—> low plasma volume—> renal retention of salt and water—> oedema.