

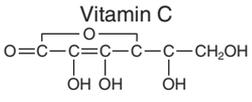
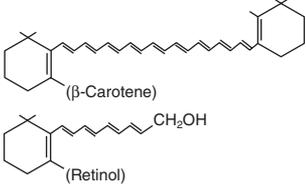
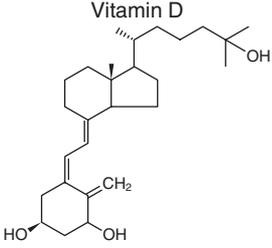
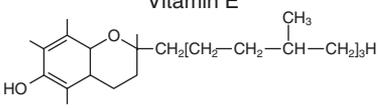
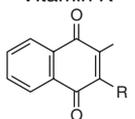
Vitamin	Active derivative or cofactor form	Principal function
<p>Vitamin C</p> 	Ascorbic acid and dehydroascorbic acid	Participation as a redox ion in many biologic oxidation and hydrogen transfer reactions
<p>Vitamin A</p> 	Retinol, retinaldehyde, and retinoic acid	Formation of rhodopsin (vision) and glycoproteins (epithelial cell function); also regulates gene transcription
<p>Vitamin D</p> 	1,25-Dihydroxyvitamin D	Maintenance of blood calcium and phosphorus levels; antiproliferative hormone
<p>Vitamin E</p> 	Tocopherols and tocotrienols	Antioxidants
<p>Vitamin K</p> 	Vitamin K hydroquinone	Cofactor for posttranslation carboxylation of many proteins including essential clotting factors

FIGURE 96e-1 (Continued)

and peripheral neuritis. Patients with *dry beriberi* present with a symmetric peripheral neuropathy of the motor and sensory systems, with diminished reflexes. The neuropathy affects the legs most markedly, and patients have difficulty rising from a squatting position.

Alcoholic patients with chronic thiamine deficiency also may have central nervous system (CNS) manifestations known as *Wernicke's encephalopathy*, which consists of horizontal nystagmus, ophthalmoplegia (due to weakness of one or more extraocular muscles), cerebellar ataxia, and mental impairment (Chap. 467). When there is an additional loss of memory and a confabulatory psychosis, the syndrome is known as *Wernicke-Korsakoff syndrome*. Despite the typical clinical picture and history, Wernicke-Korsakoff syndrome is underdiagnosed.

The laboratory diagnosis of thiamine deficiency usually is made by a functional enzymatic assay of transketolase activity measured before and after the addition of thiamine pyrophosphate. A >25% stimulation in response to the addition of thiamine pyrophosphate (i.e., an activity coefficient of 1.25) is interpreted as abnormal. Thiamine or the phosphorylated esters of thiamine in serum or blood also can be measured by high-performance liquid chromatography to detect deficiency.

TREATMENT THIAMINE DEFICIENCY

In acute thiamine deficiency with either cardiovascular or neurologic signs, 200 mg of thiamine three times daily should be given intravenously until there is no further improvement in acute symptoms; oral thiamine (10 mg/d) should subsequently be given until recovery

is complete. Cardiovascular and ophthalmoplegic improvement occurs within 24 h. Other manifestations gradually clear, although psychosis in Wernicke-Korsakoff syndrome may be permanent or may persist for several months. Other nutrient deficiencies should be corrected concomitantly.

Toxicity Although anaphylaxis has been reported after high intravenous doses of thiamine, no adverse effects have been recorded from either food or supplements at high doses. Thiamine supplements may be bought over the counter in doses of up to 50 mg/d.

RIBOFLAVIN (VITAMIN B₂)

Riboflavin is important for the metabolism of fat, carbohydrate, and protein, acting as a respiratory coenzyme and an electron donor. Enzymes that contain flavin adenine dinucleotide (FAD) or flavin mononucleotide (FMN) as prosthetic groups are known as *flavoenzymes* (e.g., succinic acid dehydrogenase, monoamine oxidase, glutathione reductase). FAD is a cofactor for methyltetrahydrofolate reductase and therefore modulates homocysteine metabolism. The vitamin also plays a role in drug and steroid metabolism, including detoxification reactions.

Although much is known about the chemical and enzymatic reactions of riboflavin, the clinical manifestations of riboflavin deficiency are nonspecific and are similar to those of other deficiencies of B vitamins. Riboflavin deficiency is manifested principally by lesions of the mucocutaneous surfaces of the mouth and skin. In addition, corneal