

total hemoglobin content, the greater is the tendency toward cyanosis; thus, patients with marked polycythemia tend to be cyanotic at higher levels of  $SaO_2$  than patients with normal hematocrit values. Likewise, local passive congestion, which causes an increase in the total quantity of reduced hemoglobin in the vessels in a given area, may cause cyanosis. Cyanosis is also observed when nonfunctional hemoglobin, such as methemoglobin (consequential or acquired) or sulfhemoglobin (**Chap. 98**), is present in blood.

Cyanosis may be subdivided into central and peripheral types. In *central* cyanosis, the  $SaO_2$  is reduced or an abnormal hemoglobin derivative is present, and the mucous membranes and skin are both affected. *Peripheral* cyanosis is due to a slowing of blood flow and abnormally great extraction of  $O_2$  from normally saturated arterial blood; it results from vasoconstriction and diminished peripheral blood flow, such as occurs in cold exposure, shock, congestive failure, and peripheral vascular disease. Often in these conditions, the mucous membranes of the oral cavity, including the sublingual mucosa, may be spared. Clinical differentiation between central and peripheral cyanosis may not always be straightforward, and in conditions such as cardiogenic shock with pulmonary edema, there may be a mixture of both types.

## ■ DIFFERENTIAL DIAGNOSIS

**Central Cyanosis (**Table 40-1**)** Decreased  $SaO_2$  results from a marked reduction in the  $PaO_2$ . This reduction may be brought about by a decline in the  $FIO_2$  without sufficient compensatory alveolar hyperventilation to maintain alveolar  $PO_2$ . Cyanosis usually becomes manifest in an ascent to an altitude of 4000 m (13,000 ft).

**TABLE 40-1 Causes of Cyanosis**