occur. Obtundation or coma may be present. At presentation, plasma AVP is usually but not always subnormal relative to the concurrent hypernatremia/hyperosmolemia. DI is usually absent but may develop during rehydration as blood volume, blood pressure, and plasma osmolarity/sodium return toward normal.

Etiology Hypodipsia is usually due to hypogenesis or destruction of the osmoreceptors in the anterior hypothalamus that regulate thirst. The defect can result from various congenital malformations of midline brain structures or may be acquired due to diseases such as surgery or aneurysms of the anterior communicating artery, primary or metastatic tumors in the hypothalamus, head trauma, granulomatous diseases such as sarcoidosis and histiocytosis, AIDS, and cytomegalovirus encephalitis. Adipsic hypernatremia without demonstrable hypothalamic lesions has also been associated with autoantibodies directed against the subfornical organ. Episodes of transient hypodipsic hypernatremia have also been reported in association with depression or other psychological disorders, suggesting that reversible neurochemical defects in the osmoregulation of thirst can also occur.

Pathophysiology A deficiency in osmotically induced thirst results in a failure to drink enough water to replenish obligatory renal and extrarenal losses. Consequently, plasma osmolarity and sodium rise often to extremely high levels before the disorder is recognized. In most cases, plasma AVP is subnormal relative to the hyperosmolarity/hypernatremia, but it is still adequate to prevent DI. However, during rehydration, plasma AVP sometimes falls to levels that result in DI before the dehydration is fully corrected **(Fig. 381-6)**. In other cases, plasma AVP does not decline even when the patient is overhydrated and a hyponatremic syndrome indistinguishable from inappropriate antidiuresis develops. This suggests that the AVP osmoreceptors normally provide inhibitory as well as stimulatory input to the neurohypophysis. In both situations, AVP secretion responds normally to nonosmotic stimuli such as nausea, hypovolemia, or hypotension, indicating that the neurohypophysis is intact. In a few patients, however, the neurohypophysis is also