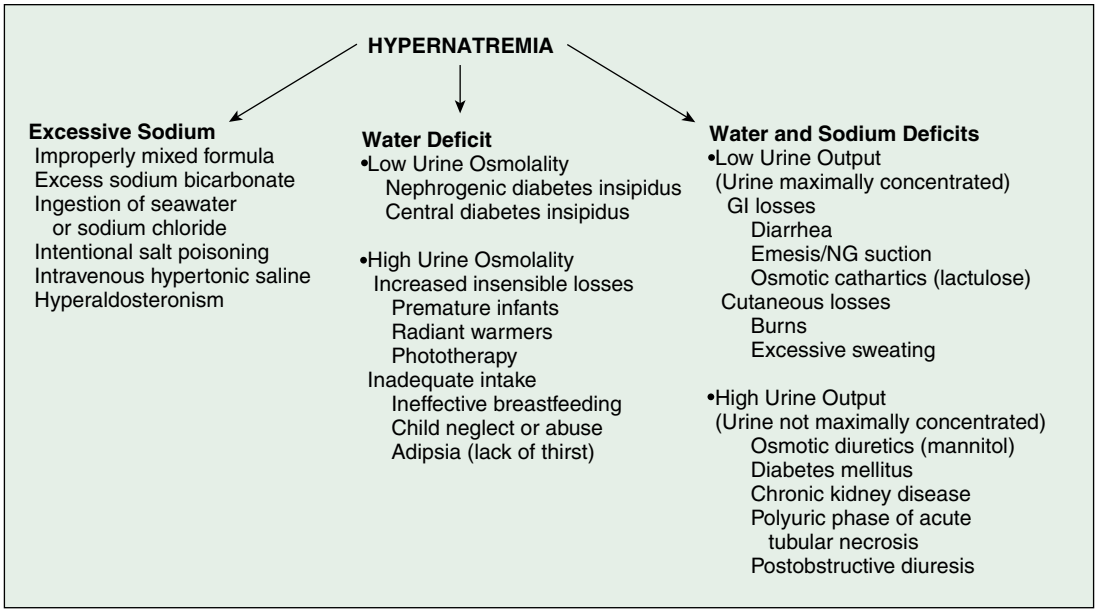

Figure 35-2 Differential diagnosis of hypernatremia by mechanism. *GI*, Gastrointestinal; *NG*, nasogastric.

Clinical Manifestations

Most children with hypernatremia are dehydrated and have the typical signs and symptoms of dehydration (see Chapter 33). Children with hypernatremic dehydration tend to have better preservation of intravascular volume owing to the shift of water from the intracellular space to the extracellular space. Hypernatremic infants potentially become more dehydrated before seeking medical attention. Probably because of intracellular water loss, the pinched abdominal skin of a dehydrated, hypernatremic infant has a *doughy* feel.

Hypernatremia, even without dehydration, causes central nervous system symptoms that tend to parallel the degree of sodium elevation and the acuity of the increase. Patients are irritable, restless, weak, and lethargic. Some infants have a high-pitched cry and hyperpnea. Alert patients are very thirsty, although nausea may be present. Hypernatremia causes fever, although many patients have an underlying process that contributes to the fever.

Brain hemorrhage is the most devastating consequence of hypernatremia. As the extracellular osmolality increases, water moves out of brain cells, resulting in a decrease in brain volume. This decrease in volume can result in tearing of intracerebral veins and bridging blood vessels as the brain moves away from the skull and the meninges. Patients may have subarachnoid, subdural, and parenchymal hemorrhage. Seizures and coma are possible sequelae of the hemorrhage.

Treatment

As hypernatremia develops, the brain generates idiogenic osmoles to increase the intracellular osmolality and prevent the loss of brain water. This mechanism is not instantaneous and is most prominent when hypernatremia has developed gradually. If the serum sodium concentration is lowered rapidly, there is movement of water from the serum into the brain cells to equalize the osmolality in the two compartments. The resultant brain swelling manifests as seizures or coma. Because of these dangers, corrected hypernatremia should be treated gradually. The goal is to decrease the serum sodium by less than 12 mEq/L every 24 hours (see Fig. 33-1). The most important component of correcting moderate or severe hypernatremia is frequent monitoring of the serum sodium to allow adjustment of fluid therapy and provide adequate correction that is neither too slow nor too fast.

In a child with hypernatremic dehydration, as in any child with dehydration, the first priority is restoration of intravascular volume with isotonic fluid. Figure 33-1 outlines a general approach for correcting hypernatremic dehydration secondary to gastroenteritis. If the hypernatremia and dehydration are secondary to water loss, as occurs with diabetes insipidus, a more hypotonic IV fluid is appropriate. A child with central diabetes insipidus should receive an ADH analog to prevent further excessive water loss. A child with nephrogenic diabetes insipidus requires a urine replacement solution to offset ongoing water losses. Chronically, reduced sodium intake, thiazide diuretics, and nonsteroidal anti-inflammatory drugs can decrease water losses in nephrogenic diabetes insipidus.

Acute, severe hypernatremia, usually secondary to sodium administration, can be corrected more rapidly because idiogenic osmoles have not had time to accumulate; this balances the high morbidity and mortality from severe, acute hypernatremia with the dangers of overly rapid correction. When hypernatremia is due to sodium intoxication, and the hypernatremia is severe, it may be impossible to administer enough water to correct the hypernatremia rapidly without worsening volume overload. Some patients require use of a loop diuretic or dialysis.