premonitory phase of migraine using functional imaging, and this may hold a key to understanding some part of the role of dopamine in the disorder.

Migraine genes identified by studying families with familial hemiplegic migraine (FHM) reveal involvement of ion channels, suggesting that alterations in membrane excitability can predispose to migraine. Mutations involving the $Ca_v2.1$ (P/Q)–type voltage-gated calcium channel *CACNA1A* gene are now known to cause FHM 1; this mutation is responsible for about 50% of FHMs. Mutations in the Na^+-K^+ATPase *ATP1A2* gene, designated FHM 2, are responsible for about 20% of FHMs. Mutations in the neuronal voltage-gated sodium channel *SCN1A* cause FHM 3. Functional neuroimaging has suggested that brainstem regions in migraine (Fig. 447-2) and the posterior hypothalamic gray matter region close to the human circadian pacemaker cells of the suprachiasmatic nucleus in cluster headache (Fig. 447-3) are good candidates for specific involvement in primary headache.

Diagnosis and Clinical Features Diagnostic criteria for migraine headache are listed in Table 447-3. A high index of suspicion is required to diagnose migraine: the migraine aura, consisting of visual disturbances with flashing lights or zigzag lines moving across the visual field or of other neurologic symptoms, is reported in only 20–25% of patients. A headache diary can often be helpful in making the diagnosis; this is also helpful in assessing disability and the frequency of treatment for acute attacks. Patients with episodes of migraine that occur daily or near-daily are considered to have chronic migraine (see "Chronic Daily Headache" in Chap. 21). Migraine must be differentiated from tension-type headache (discussed below), the most common primary headache syndrome seen in the population. Migraine has several forms that have been defined (Table 447-1): migraine with and without aura and chronic migraine, the latter occurring 15 days or more a month, as the most important. *Migraine at its most basic level is headache with associated features, and tension-type headache is headache that is featureless. Most patients with disabling headache probably have migraine.*

Patients with acephalgic migraine (typical aura without headache, 1.2.1.2 in Table 447-1) experience recurrent neurologic symptoms, often with nausea or vomiting, but with little or no headache. Vertigo can be prominent; it has been estimated that one-third of patients referred for vertigo or dizziness have a primary diagnosis of migraine. Migraine aura can have prominent brainstem symptoms, and the terms *basilar artery* and *basilar-type migraine* have now been replaced by *migraine with brainstem aura* (Table 447-1).

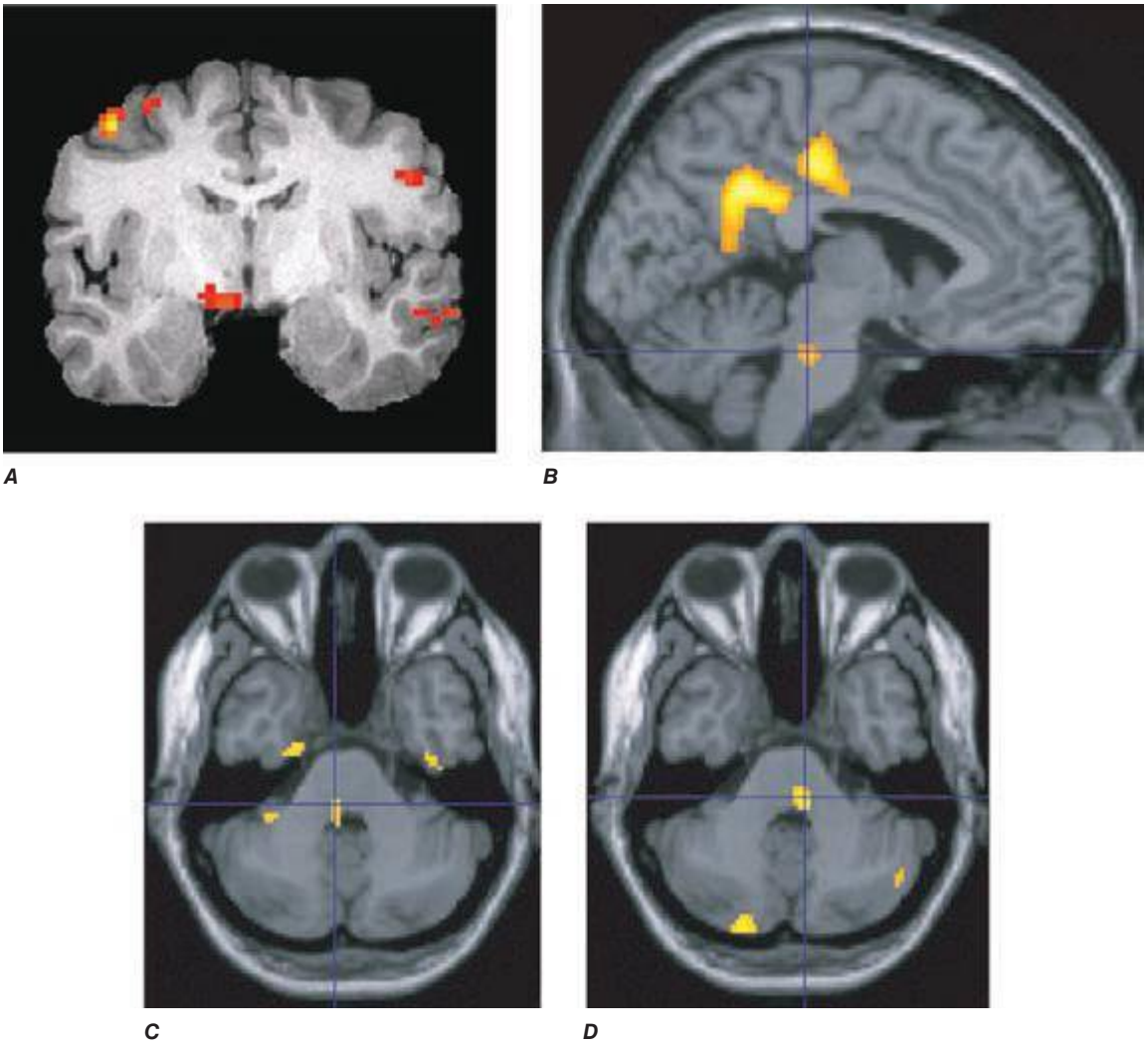

FIGURE 447-2 Positron emission tomography (PET) activation in migraine. Hypothalamic, dorsal midbrain, and dorsolateral pontine activation is seen in triggered attacks in the premonitory phase before pain, whereas in migraine attacks, dorsolateral pontine activation persists, as it does in chronic migraine (not shown). The dorsolateral pontine area, which includes the noradrenergic locus coeruleus, is fundamental to the expression of migraine. Moreover, lateralization of changes in this region of the brainstem correlates with lateralization of the head pain in hemicranial migraine; the scans shown in panels ***C*** and ***D*** are of patients with acute migraine headache on the right and left side, respectively. *(Panel A from FH Maniyar et al: Brain 137:232, 2014; panel B from SK Afridi et al: Arch Neurol 2005;62:1270; Panels C and D from SK Afridi et al: Brain 128:932, 2005.)*