kind of aphasia is not seen with CVAs. It differs from Wernicke's aphasia or posterior transcortical aphasia because speech is usually informative, repetition is intact, and comprehension of conversation is relatively preserved, as long as the meaning is not too dependent on words that the patient fails to understand. Peak atrophy sites are located in the left anterior temporal lobe, indicating that this part of the brain plays a critical role in the comprehension of words, especially words that denote concrete objects. The neuropathology is frequently an FTLD with abnormal precipitates of the 43-kDa transactive response DNA-binding protein TDP-43. The *logopenic variant* is characterized by preserved syntax and comprehension but frequent and severe word-finding pauses, anomia, circumlocutions, and simplifications during spontaneous speech. Peak atrophy sites are located in the temporoparietal junction and posterior temporal lobe, partially overlapping with traditional location of Wernicke's area. However, the comprehension impairment of *Wernicke's aphasia* is absent, perhaps because the underlying white matter, frequently damaged by cerebrovascular accidents, remains relatively intact in PPA. In contrast to Broca's aphasia or agrammatic PPA, the interruption of fluency is variable so that speech may appear entirely normal if the patient is allowed to engage in small talk. Logopenic PPA resembles the anomic aphasia of Table 36-1 but usually has longer and more frequent word-finding pauses. Patients may also have poor phrase and word repetition, in which case the aphasia resembles the *conduction aphasia* in Table 36-1. Of all PPA subtypes, this is the one most commonly associated with the pathology of AD, but FTLD can also be the cause. In addition to these three major subtypes, PPA can also present in the form of *pure word deafness* or *Gerstmann's syndrome*.

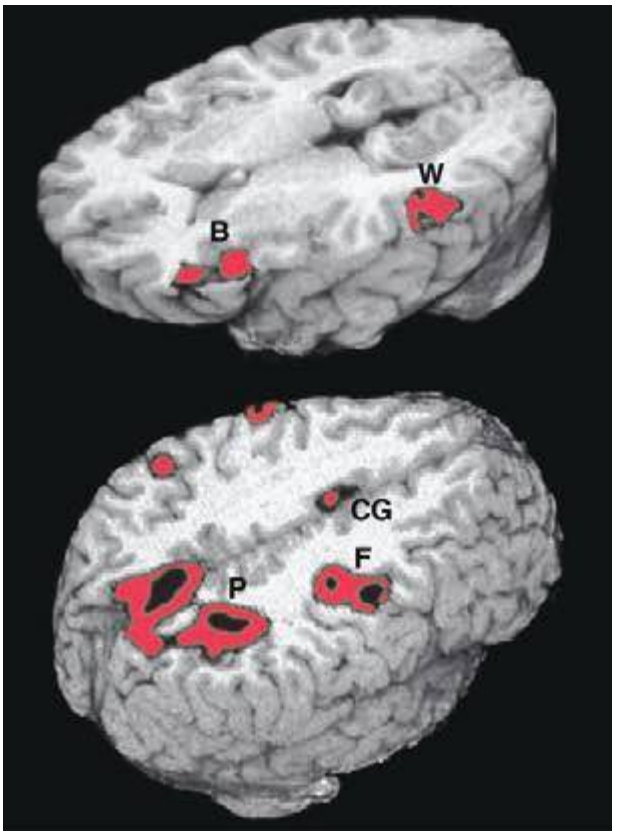

FIGURE 36-2 Functional magnetic resonance imaging of language and spatial attention in neurologically intact subjects. The red and black areas show regions of task-related significant activation. (*Top*) The subjects were asked to determine if two words were synonymous. This language task led to the simultaneous activation of the two epicenters of the language network, Broca's area (B) and Wernicke's area (W). The activations are exclusively in the left hemisphere. (*Bottom*) The subjects were asked to shift spatial attention to a peripheral target. This task led to the simultaneous activation of the three epicenters of the attentional network: the posterior parietal cortex (P), the frontal eye fields (F), and the cingulate gyrus (CG). The activations are predominantly in the right hemisphere. *(Courtesy of Darren Gitelman, MD; with permission.)*

THE PARIETOFRONTAL NETWORK FOR NEGLECT AND RELATED CONDITIONS

Adaptive spatial orientation is subserved by a large-scale network containing three major cortical components. The *cingulate cortex* provides access to a motivational mapping of the extrapersonal space, the *posterior parietal cortex* to a sensorimotor representation of salient extrapersonal events, and the *frontal eye fields* to motor strategies for attentional behaviors (Fig. 36-2). Subcortical components of this network include the striatum and the thalamus. Damage to this network can undermine the distribution of attention within the extrapersonal space, giving rise to hemispatial neglect, simultanagnosia and object finding failures. The integration of egocentric (self-centered) with allocentric (object-centered) coordinates can also be disrupted, giving rise to impairments in route finding, the ability to avoid obstacles, and the ability to dress.

HEMISPATIAL NEGLECT

Contralesional hemispatial neglect represents one outcome of damage to the cortical or subcortical components of this network. *The traditional view that hemispatial neglect always denotes a parietal lobe lesion is inaccurate*. According to one model of spatial cognition, the right hemisphere directs attention within the *entire* extrapersonal space, whereas the left hemisphere directs attention mostly within the contralateral right hemispace. Consequently, left hemisphere lesions do not give rise to much contralesional neglect because the global attentional mechanisms of the right hemisphere can compensate for the loss of the *contralaterally* directed attentional functions of the left hemisphere. Right hemisphere lesions, however, give rise to severe contralesional left hemispatial neglect because the unaffected left hemisphere does not contain ipsilateral attentional mechanisms. This model is consistent with clinical experience, which shows that contralesional neglect is more common, more severe, and longer lasting after damage to the right hemisphere than after damage to the left hemisphere. Severe neglect for the right hemispace is rare, even in left-handers with left hemisphere lesions.

Clinical Examination Patients with severe neglect may fail to dress, shave, or groom the left side of the body; fail to eat food placed on the left side of the tray; and fail to read the left half of sentences. When asked to copy a simple line drawing, the patient fails to copy detail on the left, and when the patient is asked to write, there is a tendency to leave an unusually wide margin on the left. Two bedside tests that are useful in assessing neglect are *simultaneous bilateral stimulation* and *visual target cancellation*. In the former, the examiner provides either unilateral or simultaneous bilateral stimulation in the visual, auditory, and tactile modalities. After right hemisphere injury, patients who have no difficulty detecting unilateral stimuli on either side experience the bilaterally presented stimulus as coming only from the right. This phenomenon is known as *extinction* and is a manifestation of the sensory-representational aspect of hemispatial neglect. In the target detection task, targets (e.g., A's) are interspersed with foils (e.g., other letters of the alphabet) on a 21.5- to 28.0-cm (8.5 to 11 in.) sheet of paper, and the patient is asked to circle all the targets. A failure to detect targets on the left is a manifestation of the exploratory (motor) deficit in hemispatial neglect (Fig. 36-3*A*). Hemianopia is not by itself sufficient to cause the target detection failure because the patient is free to turn the head and eyes to the left. Target detection failures therefore reflect a distortion of spatial attention, not just of sensory input. Some patients with neglect also may deny the existence of hemiparesis and may even deny ownership of the paralyzed limb, a condition known as *anosognosia*.