75 Neurophysiological Networks Integrating Human Emotions

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ABSTRACT Cortico-limbic-brainstem networks integrate visceral, cognitive, and contextual information in humans to produce emotional feelings and behaviors. The *orienting complex* prepares the organism to process (via arousal and directed attention) and react to (via generalized viscero- and skeletomotor activation) biologically prepotent stimuli. Such stimuli evoke a series of field potentials (N2/P3a/SW) from a distributed frontoparietal circuit subserving attention. Electrical stimulation of the limbic parts of this circuit (rectal and cingulate gyri) evoke visceromotor responses. Emotional input to cognitive *event integration* occurs during the field potentials N4/P3b, generated in high-level sensory and multimodal association cortices and, predominantly, in the hippocampal and amygdala formations and lateral orbital cortex. Medial temporal units respond to both visceral and abstract cognitive information, and medial temporal stimulation evokes viscerosensations, emotionally eloquent hallucinations, and emotional feelings. Emotional influences on voluntary *response selection* may occur during the *readiness potential*, generated in the premotor and central cingulate cortices. Stimulation of the central cingulate cortex evokes partially organized movements. Sustained contextual information, reflected in the frontal contingent negative variation (CNV), provides a long-lasting influence unifying the phasic responses over time. Thus, limbic and perilimbic cortices contribute emotional guidance during successive neurophysiological stages underlying orienting, cognitive integration, voluntary action, and sustained context.

The neurophysiological mechanisms of human emotion encompass a vast range of phenomena, from fixed action patterns triggered by biologically imperative stimuli to deeply considered voluntary acts fulfilling life goals. Such processes that integrate and guide the totality of behavior are necessarily complex. One source of this complexity is the fact that different levels of the human nervous system are organized according to different principles, have access to different sources of information, and control different aspects of behavior. Thus, the motivations that serve biological ends may arise in a system that has direct access to visceral state but no direct means to influence volition and awareness. Conversely, motivations that arise in intellectual projections of possible future scenarios must find means to influence visceral output as well as general cortical tonus. Perhaps most crucially, emotion must include the mechanisms that resolve conflicts between these levels of motivation, such as those that arise, for example, during a religious fast.

In short, while emotion always seems to function to provide a directed integration of the entire organism, both the goals and manifestations of this integration may be extremely diverse. This diversity is reflected in the methods, goals, and terminology used to investigate human emotion. One research approach has concentrated on the psychophysiological manifestations of emotion, especially during visceral conditioning and the orienting response. Another focuses on the cognitive context of emotion, measuring motivation, volition, and interpretation. A third, speaking more directly to clinical concerns, focuses on the personality. In order to relate the discoveries of these distinct research approaches to the neural basis of human emotion, we begin this review by offering a simple classification of emotional processes, according to the time scale at which they operate. We distinguish four successive but overlapping stages in the emotional reaction to a stimulus: (1) the orienting complex, (2) emotional event integration, (3) response selection, and (4) sustained emotional context.

The orienting complex (corresponding to the orienting response in its broadest sense) consists of the orientation of attention toward a potentially important event and the mobilization of resources to cope with it. At its most extreme this reaction is exemplified by the

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startle response. While the orienting complex is automatic and thus preconscious in itself, it facilitates encoding of the eliciting event for awareness in emotional event integration.

During emotional event integration, the neurally encoded stimulus is integrated with semantic associates and other information from long-term semantic memory, relevant events from declarative memory, and the current cognitive and emotional context. This integration is necessary and sufficient for awareness. It results in an affective coloring and cognitive interpretation of even the most mundane events. The event, as encoded during event integration, is itself integrated with the context for action, resulting in voluntary (conscious) acts. This phase—response selection—also integrates a multitude of influences and thus will reflect a continuum of motivations from the routine to the passionate.

Both orienting complexes and event integrations are heavily influenced by the internal context, that is, the sustained neurophysiological background to phasic events—the subject's mood. Conversely, this context only becomes manifest in directly observable behavior through its effects on orienting complexes and event integrations. More permanent influences (corresponding to the temperament or personality) probably reflect hormonal or structural influences that are beyond the scope of this chapter.

Each stage—orienting complex, event integration, response selection, and internal context—can be associated with a distinct neural substrate. For each type of emotional process, an underlying neural network is suggested and supporting evidence is presented from recording, lesion, and stimulation studies in humans.

Orienting complex

Startle: Genetically Programmed and Brainstem Organized   The orienting complex is a mobilization of cerebral and somatic resources in order to effectively cope with a biologically important event. Two aspects of this reaction can be distinguished, which mobilize systems for, respectively, perceiving and comprehending the event, and taking physical action. The essential nature of this reaction can be seen vividly in its exaggerated form, the startle response, an involuntary, genetically programmed response to sudden sensory events that signal a possible impending biological emergency. For example, a loud abrupt sound will elicit arousal if the organism is somnolent or interruption

of current cognitive processing if alert; orientation of attention and of the sense organs toward the apparent source of the sound; and distribution of sensory information regarding the stimulus to widespread cerebral areas for processing. In addition, the sound will evoke a multifaceted somatic response that prepares the body for action or for withstanding injury, including generalized sympathetic arousal, increased skeletal muscular tonus, and hormonal changes (Ohman, 1987). Animal studies have shown that the startle as well as other forms of the orienting complex are neuronally integrated in the midbrain and perhaps the hypothalamus (Jordan, 1990; Klemm and Vertes, 1990). Apparently the same organization obtains in humans, given that the orienting complex is preserved in anencephalic infants (Tuber et al., 1980), and that stimulation of the human central gray and lateral hypothalamus results in sympathetic arousal (Iacono, Blaine, and Nashold, 1982).

Forebrain Implementation and Control   Brainstem output during the orienting complex is apparent in autonomic activity (especially electrodermal; Ohman, 1987), whereas the forebrain's engagement in orienting can be measured as event-related potentials (ERPs). Startling stimuli evoke a large N1c component, and changes in auditory stimulus trains automatically evoke an MMN (Naatanen and Gaillard, 1983). The most characteristic response to orienting stimuli is the N2/P3a/SW complex, recorded over the frontocentral scalp with peaks at about 200, 280, and 350 ms after stimulus onset (figure 75.1; Halgren, 1990b; Marinkovic, in preparation). Across trials, the N2/P3a size is correlated with electrodermal responses (Lyytinen and Naatanen, 1987), and conforms well to habituation-dishabituation orienting complex parameters (Rockstroh and Elbert, 1990). However, the autonomic orienting complex and the N2/P3a can be dissociated on the basis of details of their reaction to repetition or to changes in the task relevance of the stimuli (Naatanen and Gaillard, 1983; Rosler, Hasselmann, and Sojka, 1987). These differences suggest that the N2/P3a/SW and the classical orienting complex may reflect the afferent (preparation-to-process) and efferent (preparation-to-respond) functions of the orienting complex, respectively.

Slightly before the scalp-recorded N2/P3a/SW, peaks with similar waveforms and task correlates are generated in widespread cortical areas, especially in
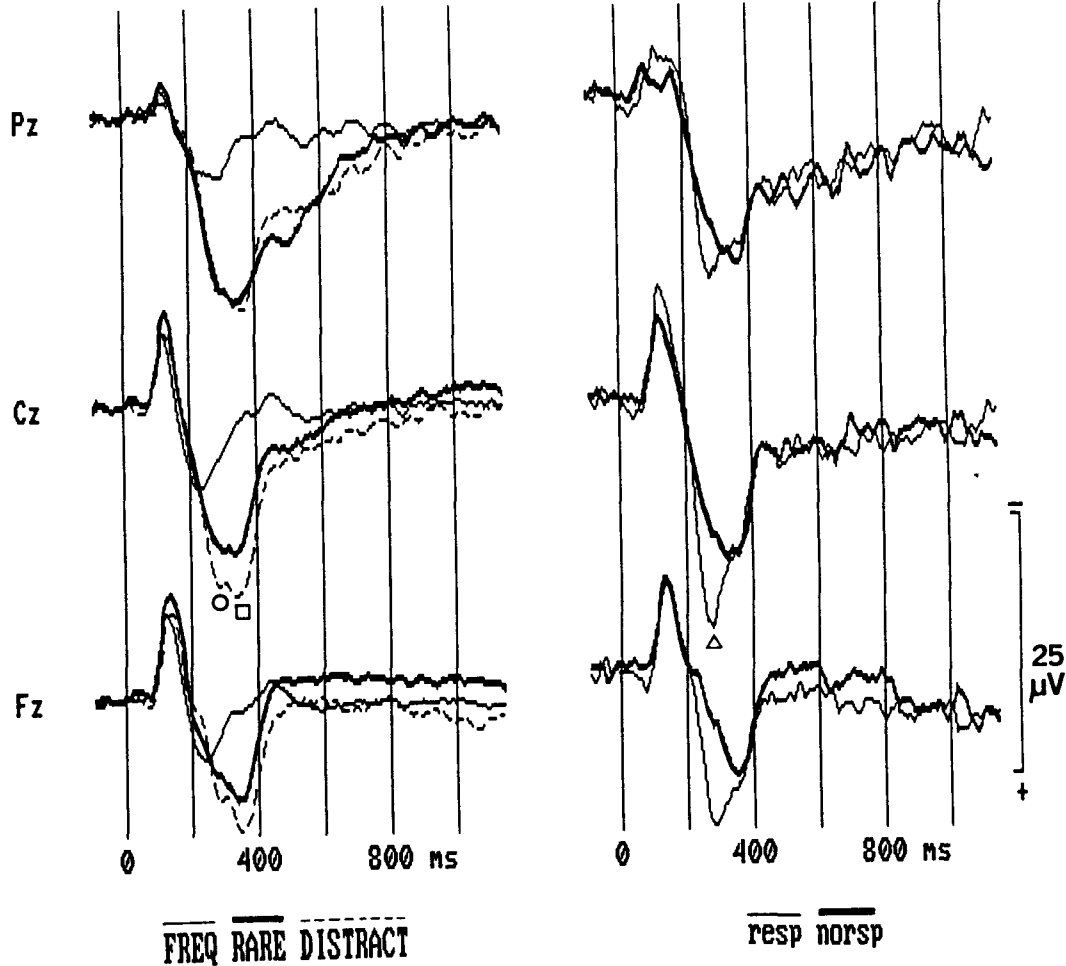
FIGURE 75.1 (Left) Brain potentials recorded from the human scalp during an auditory discrimination task. Interspersed among frequent tones (thin lines), are rare target tones to which the subject pressed a key (thick lines), and occasional unique, strange, distracting sounds to which the subject made no response (dashed lines). Both targets and distractors evoked a large P3 component, with peaks at 285 ms (circle; termed P3a) and 330 ms (square; termed P3b). Depth recordings identify distinct generating systems for the P3a (including cingulate and rectal g.) and the P3b (including hippocampal formation and lateral orbital cortex).

(Right) Correlation of the P3a with the autonomic orienting complex. The brain potentials evoked by rare distractor sounds were divided into two groups, according to whether the sounds did (thin line) or did not (thick line) also evoke an electrodermal response. Trials with autonomic responses also evoke a large P3a, especially at frontal (Fz) and central (Cz) electrode sites (triangle). This is consistent with other studies identifying the P3a as the manifestation of the orienting complex in the forebrain. In contrast, the P3b seems to envelope the closure of the intentional event-encoding process. (From Marinkovic, in preparation)

the anterior cingulate gyrus (plus its posterior and inferior extensions, posterior cingulate gyrus and gyrus rectus; see figure 75.2), the inferior parietal lobule (in particular the supramarginal gyrus), and the region of area 46 (in the inferior frontal gyrus, pars triangularis) (Halgren et al., submitted; Wood and McCarthy, 1985; Alain et al., 1989; Smith et al., 1990). This neural circuit also includes a brainstem modulator or trigger, inasmuch as unilateral unusual stimuli still evoke

part of the ipsilateral brain P3 after complete section of the forebrain commissures (Kutas et al., 1990).

The same parietal-cingulate-dorsolateral prefrontal circuit that generates the N2/P3a/SW has also been identified by metabolic and lesion studies in humans as a functional network for directed attention (see table 75.1 and chapter 39). Furthermore, electrical stimulation of the limbic parts of this circuit (i.e., the posterior orbital cortex; Livingston et al., 1948, and the anterior
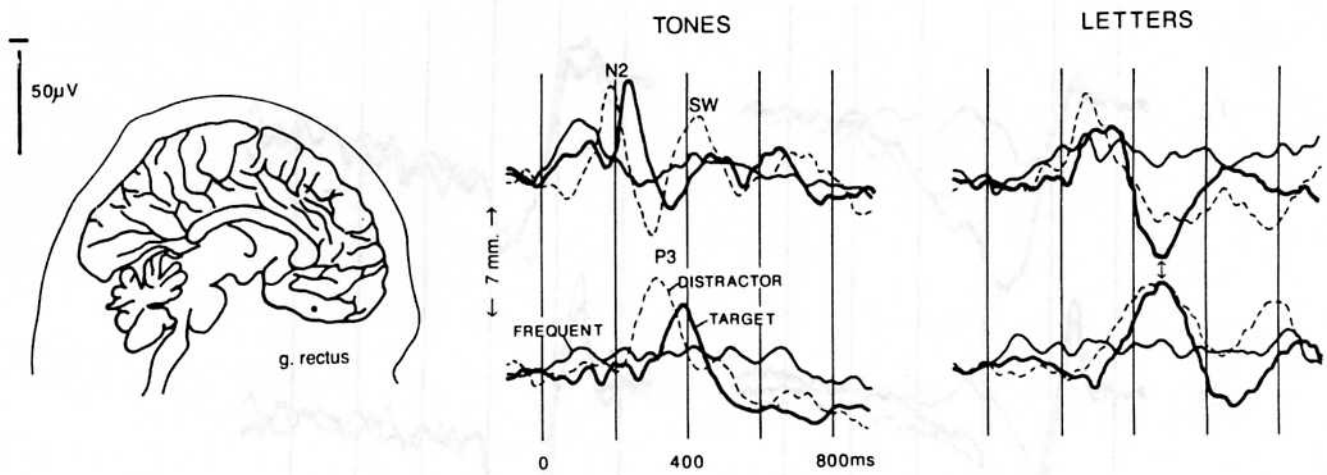
FIGURE 75.2 Generation of the P3a in the human rectal g. (medial orbitofrontal cortex) during the orienting complex. Averaged field potentials from two sites are presented. The location of the most medial site (bottom traces) is indicated at right on a tracing of the sagittal MRI. The upper traces were recorded simultaneously from a site 7 mm directly lateral. In the task at left, 50 ms TONES were presented every 1600 ms. Most of the tones were low-pitched (FREQUENT, thin lines). Interspersed randomly on 11% of the trials was a rising tone indicating that the subject should press a key (TARGET, thick lines). On another 11% of the trials were nontarget (DISTRACTOR) sounds, each unique (dashed lines). In the task at the right (LETTERS), visual symbols were presented for 200 ms every 1600 ms, consisting of rare targets (*) and distractors (letters) interspersed randomly with frequents (x). In the upper traces, a negative (upward)–positive–negative sequence corresponding to the N2/P3a/SW is evoked by rare target and distractor auditory and visual stimuli. In the lower traces, the P3a can be seen to invert to negative polarity, suggesting a local generator. The first vertical line indicates stimulus onset, with successive lines every 200 ms.

cingulate gyrus apparently adjacent to the corpus callosum, area 33; Pool and Ransohoff, 1949) can evoke sympathetic arousal, the efferent limb of the orienting complex.

Thus, during orienting the cortical and limbic areas critical for directed attention are activated more or less synchronously in a series of neurophysiological stages. The task correlates and timing of these stages suggest that they provide an envelope for the integrative construction of an initial rapid neural representation of the imperative stimulus. The prominent involvement of limbic sites in this network suggests a mechanism whereby autonomic aspects of the orienting complex can be integrated with attentional aspects. Furthermore, via this network the orienting complex can be evoked or modulated by cortically recognized events, and not simply by genetically programmed stimuli. Indeed, in humans the learned orienting complex seems to occur only when the subject becomes conscious of the contingency between the conditioned and unconditioned stimuli (Ohman, 1987). The prefrontal P3a has a shorter latency than the posterior parietal (Halgren et al., submitted), and prefrontal lesions eliminate the P3a over both anterior and posterior cortex (Knight, 1984), suggesting that the prefrontal cortex plays a leading role in organizing the cortical orienting complex in humans.

LIMITS OF THE ORIENTING COMPLEX   Since the orienting complex functions to reorient behavior and cognition toward significant stimuli, it embodies the essence of emotion, as defined by those theories that consider emotion to be a generalized arousal that disrupts ongoing behavior (e.g., Plutchik, 1980). However, for other theorists (Panksepp, 1982), the orienting complex is too brief and nonspecific to be considered as more than a larval emotion, or preemotional response, constituting the primary emotional appraisal that may then lead to secondary appraisal and true emotion (Ohman, 1987). That is, the orienting complex is not able to organize a high-level, complex response, nor can a high-level, fine analysis of the event occur before the orienting complex is evoked. It is preconscious, in the temporal as well as the hierarchical sense, often occuring precisely in situations that do not allow a fine analysis before some response is demanded. In humans, the function of this level seems to be in facilitating the later occurrence of a more cognitive evaluation and

volitional reaction, both by mobilizing resources (attentional, visceral, and somatomotor) and by imposing upon the organism an imperative to attend and respond.

Emotional event integration

COGNITION AND EMOTION In everyday life, emotions are experienced much more frequently as integral parts of complex events—involving the meaning of such stimuli as words, faces, or scenes—than as automatic responses to loud noises. Similarly, emotion is more likely to become manifest as a complex, intentional, goal-directed sequence of movements—such as deciding to undergo elective surgery, or driving to a lover's house—than as global sympathetic arousal.

In contrast to the orienting complex, in which there is little or no distinction between the generalized responses evoked by different stimuli, at the level of event integration the different emotions have distinct subjective qualities and objective consequences. The distinctiveness of different emotions cannot be due to their autonomic concomitants, because these concomitants are very similar for different emotions and only occur *after* the feeling has been defined (Ohman, 1987). Consequently, cognition must play a crucial role in assigning emotional specificity, which reflects mainly the psychosocial context in which the autonomic arousal occurs (Mandler, 1975). Yet emotions have a long evolutionary history and therefore must have a genetic basis (Panksepp, 1982). The *direction of experience* and behavior toward the biological goals of survival and reproduction is clearly too important to be left entirely to either the cognitive (aware) or the visceral (reflexive) brain systems: The crucial essence of emotion is how the contributions of both are effectively integrated.

STIMULATION-EVOKED EMOTIONS, VISCEROSENSATIONS, AND EMOTIONALLY SYMBOLIC MEMORIES In humans, accurate emotional judgments require the contributions of several brain regions, including the frontal, temporal, and parietal lobes, bilaterally (Kolb and Taylor, 1990; Heilman and Bowers, 1992). The regions where electrical stimulation may give rise to subjective emotional feelings tend to be more localized, but nonetheless include all of the limbic system. Among the variety of emotions evoked by human amygdala and hippocampal stimulation, fear is by far the most common (Halgren et al., 1978; Gloor, 1991; Halgren, 1991a). In contrast, stimulation of the anterior cingulate gyrus has been reported to give rise to about equal numbers of positive and negative emotions (Meyer et al., 1973; Bancaud et al., 1976; Laitinen, 1979). Similarly, stimulation of callosal fibers believed to interconnect the posterior orbital and anterior cingulate (area 32) cortices (Barbas and Pandya, 1984) results in mainly pleasant emotions (Laitinen, 1979). Stimulation in the septal area may also result in pleasurable feelings (Heath, John, and Fontana, 1968; Obrador, Delgado, and Martin-Rodriguez, 1973). Finally, uncontrolled laughing may occur with epileptic seizures arising in the anterior hypothalamic-preoptic area (Gaggero et al., 1991). Despite reports of uncontrolled rage after electrical stimulation of the human amygdala, this response is extremely rare, and appears usually or always to reflect a defensive reaction in a highly confused state rather than a well-directed or intentional attack. Conversely, hostile behavior may be decreased by amygdala lesions, but clear effects are observed only in retarded subjects (for review see Halgren, 1991a).

As might be expected given the strong link between subjective feelings and visceral sensations, stimulation of essentially the same anatomical areas gives rise to both types of mental phenomena (Halgren and Chauvel, 1993). The most common visceral sensation evoked from the medial temporal lobe is the epigastric sensation, which typically rises from the stomach up the chest to the throat and head (Halgren et al., 1978). Intragastric recordings during epigastric sensations indicate that they are true hallucinations, rather than the indirect results of evoked gastric movements (Van Buren, 1963). Conversely, bilateral removal of the medial temporal lobe in one case eliminated the subjective sense of hunger or thirst (Hebben et al., 1985).

Stimulation of the hippocampus and amygdala can also evoke respiratory and cardiovascular phenomena (Halgren et al., 1978). Evidence that these phenomena may be viscerosensory is found in the fact that a large proportion of medial temporal lobe neurons are highly sensitive to respiratory and cardiac cycles, as well as to blood gas levels (Halgren, Babb, and Crandall, 1977; Frysinger and Harper, 1989). Although paraesthesias of the external genitalia are evoked in the basoposterior paracentral lobule (i.e., the medial parietal lobe), sexual feelings per se appear to involve the insula and amygdala (Stoffels et al., 1980). Electrical stimulation

TABLE 75.1
Neural systems for integrating emotion into orienting, encoding, acting, and maintaining

	Orienting Complex	Event Encoding

Function	Respond to biologically imperative stimuli
	  Prepare to process
	    Alerting
	    Direct attention
	    Distribute information
	  Prepare to react: Autonomic arousal
		Encode event for cognition
		  Cognitive
		    Semantic associations
		    Individual identificiation
		  Emotional
		    Emotional associations
		    Psychosocial context

Structures	Brain stem
	  Alerting
	  Autonomic activation
	Attentional circuit
	  Neocortical (parietofrontal)
	    Orient attention
	    Distribute information
	  Limbic (cingulate and rectal gyri)
	    Integrate hypothalamic and cortical components
		Neocortical
		  Fusiform gyrus: Object-encoding
		  Superior temporal sulcus: Semantic association
		Limbic
		  Amygdala: Emotional association
		  Hippocampus: Emotional memories
		  Lateral orbitofrontal: Emotional context

Lesions	Brain stem: Death
	Limbic: Dyscontrol of attention/emotion
	Neocortical: Neglect
		Neocortical: Aphasias, agnosias
		Limbic
		  Amygdala: Less emotionality?
		  Hippocampus: Amnesia
		  Lateral orbitofrontal: Poor judgment

Stimulation	Brain stem: Strong universal autonomic
	Limbic: Contextual autonomic
		Limbic
		  Emotions, viscerosensations
		  Emotionally eloquent memories

Evoked potentials	Components N2/P3a/SW
	From 130 to 400 ms post–stimulus onset
	Evoked by unusual stimuli regardless of attention or
	  meaning
		Components N4/P3b
		From 260 to 800 ms post–stimulus onset
		Evoked by words, faces, etc. if attended and processed
		  for meaning
		Modulated by ease of cognitive integration
		  with information-specific unit activity

of the amygdala, hippocampal formation, or superior temporal gyrus may also evoke intense memory- or dreamlike hallucinations (Penfield and Perot, 1963; Halgren et al., 1978; Gloor, 1991; Bancaud et al., 1994). Although they are vivid and detailed, the subject knows that these hallucinations are not current reality. In a minority of patients, the particular experience selected appears to symbolize current psychodynamic concerns (Halgren and Chauvel, 1993).

All three phenomena evoked by limbic stimulation (viscerosensory hallucinations, emotional feelings, and emotionally symbolic hallucinated images) can provide a signal to awareness as to the biological significance of an event (Halgren and Chauvel, 1993). The subjective experience of emotion may be considered to

be composed of such communications from the unconscious to the conscious brain. Anatomically, this would correspond to the fact that the tertiary sensory and supramodal association cortices receive input not only from sensory cortices but also from the amygdala and hippocampal formations (Amaral and Insausti, 1990). The implication, that experience is constructed by association cortex from limbic as well as sensory input, is supported by recordings in the human brain during event encoding.

NEUROPHYSIOLOGICAL STAGES IN EVENT ENCODING
Anatomical and physiological studies in monkeys have defined the successive stages of object processing in the visual system (see chapter 24). In humans also, evoked-

Response Choice	Sustained Context

Integrate encoded event with response templates
Select and organize movement
	Maintain information in active memory
	  Contextual
	  Stimulus

Neocortical
  Premotor: Movement sequencing
  Precentral: Movement command
Limbic
  Middle cingulate gyrus: Integrate emotion with movement
  Supplementary motor cortex: Movement initiation
	Brain stem: Set processing mode
	Neocortical
	  Dorsolateral prefrontal: Cognitive context
	  Ventrolateral prefrontal: Socioemotional context
	  Premotor: Response set
	  Posterior: Stimulus characteristics and identity

Limbic
  Middle cingulate gyrus: Akinetic mutism
  Supplementary motor cortex (global)
Neocortical
  Premotor: Uncoordination
  Precentral: Paralysis (focal)
Middle cingulate gyrus: Complex adaptive movement sequences
Supplementary motor cortex: Postural movements
	Neocortical
	  Ventrolateral prefrontal: Contextually inappropriate behavior
	  Premotor: Impulsive behavior
	
	Prefrontal: Confusion, forced thoughts

Component readiness potential Begins from 3000 to 300 ms premovement onset	Component contingent negative variation Between related stimuli

potential components reveal successive stages of face and word encoding that begin in the primary sensory cortex, pass through levels of association cortex, and rapidly arrive at multimodal and limbic structures in all lobes (table 75.1). A face-specific potential at 180 ms appears to be generated initially in the basal temporo-occipital cortex (areas 19/37, perhaps corresponding to basal V4), and then transmitted widely to superior temporal sulcal, parietotemporal, and dorsolateral prefrontal cortices (figure 75.3; Halgren et al., 1994a). Words evoke potentials at about 220 ms in the general region of the angular gyrus and Broca's area. Following these specific processing potentials are more widespread negativities that culminate in the well-studied N4 (or N400), and P3b (or P300, or late positive component).

Apparently locally generated N4/P3b's have been recorded in several structures, including the superior temporal sulcus, posterior parietal cortex, supramarginal and cingulate gyri, and area 46 in the dorsolateral prefrontal cortex (Halgren et al., 1994a). Large N4s are also generated in fusiform gyrus, where they may be specific to words or faces. It is striking, however, that the largest N4/P3b generators are in limbic structures (figure 75.4): the medial temporal lobe, where the N4 appears to be generated in the hippocampus, parahippocampal gyrus, and periamygdaloid region and the P3b is probably generated in the hippocampus proper (Stapleton and Halgren, 1987; McCarthy et al., 1989); and the lateral orbital cortex (Halgren et al., 1994b).

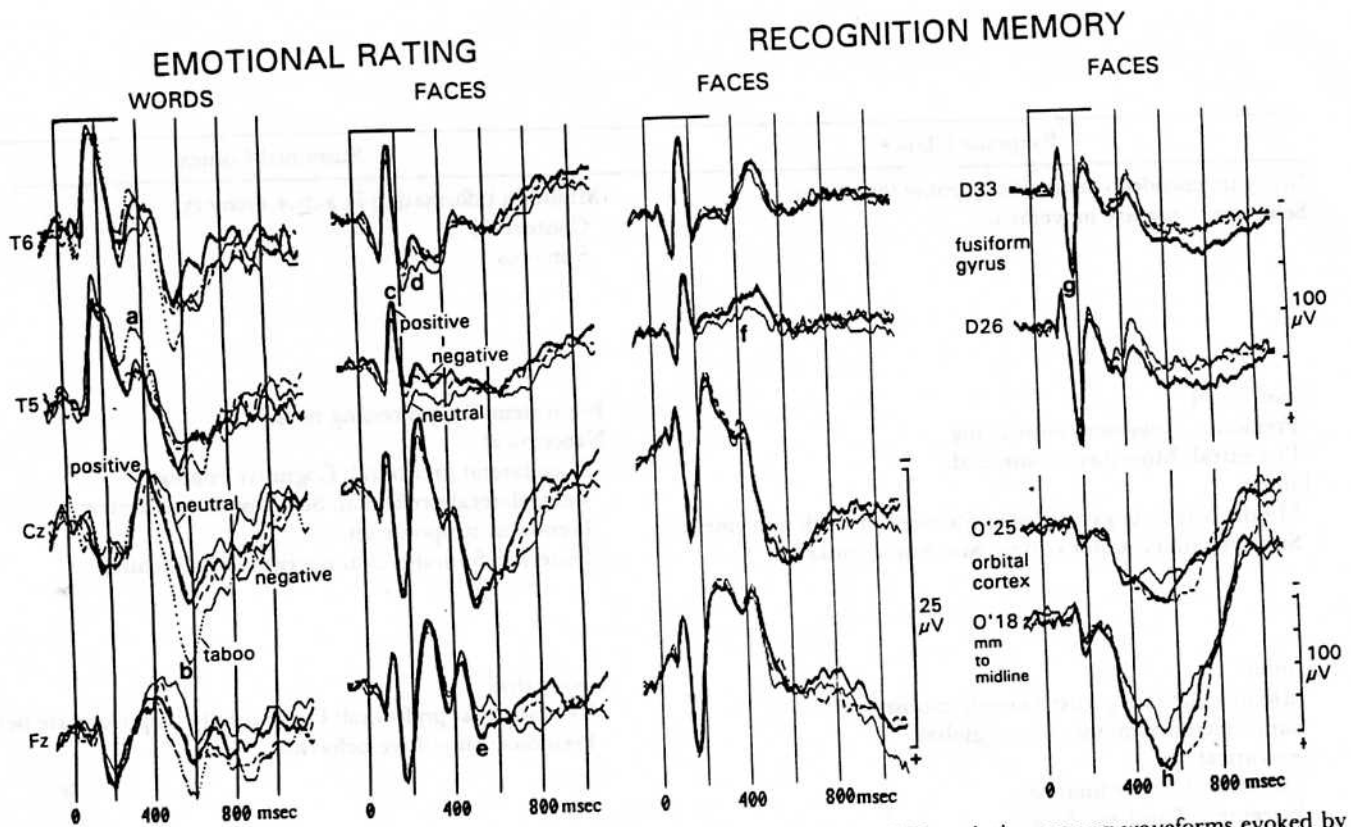The cognitive task correlates, timing, and anatomi-

FIGURE 75.3   Potentials recorded from the human scalp (left 3 columns), and depth (fourth column), in response to faces and words expressing different emotions. In the emotional rating task, single words, or faces of unfamiliar people, were presented, and the subject moved a joystick to the left or right (indicating "very negative" to "very positive"). Preliminary data obtained in the emotional word rating task show a large difference in waveforms evoked by emotionally neutral versus taboo words, with negative or positive valence words evoking waveforms midway between neutral and taboo words at frontocentral sites. The taboo words evoked an early negativity over the left posterior temporal scalp (T5), beginning at 280 ms (a). The difference between emotionally charged and neutral words is maximal over the central midline scalp (Cz), where it appears as an increased P3b beginning about 400 ms after stimulus onset (b). The earliest significant waveform differences with respect to emotional expressions of faces appear at temporo-occipital sites and persist there for several hundred ms. The N175 component is significantly smaller in response to both emotional expressions when compared to neutral expression (c). This difference persists as a negativity between 200 and 400 ms to emotional expressions at temporo-occipital sites (d). The only significant differentiation among waveforms evoked by all three emotions appears frontocentrally in the 400–600ms latency range (e). In the recognition memory task, the subject pressed a key when a face that had previously been presented reappeared. Significant effects of emotional expression in this task were limited to the temporo-occipital sites. These effects were observed most clearly in area measures between 200 and 600 ms after stimulus onset (f). No difference between potentials evoked by positive and negative expressions was observed in this task. Thus, distinct brain potentials to different facial expressions were evoked only in the task where emotional judgments were explicitly required. Direct intracerebral recordings during the recognition task reveal a large focal potential to faces in the fusiform g, peaking at about 180 ms after stimulus onset (g). Like the component of similar latency recorded on the scalp over temporo-occipital cortex, the amplitude of this component changed significantly between different emotions. Emotionally expressive faces also evoke a significantly larger late positivity in the left orbitofrontal cortex (h), with a peak latency (520 ms) similar to that of the scalp P3b (e). (From Marinkovic et al., in preparation)

cal extent of the N4 suggest that it embodies the global integration of the stimulus with the current cognitive context, in order to neurally encode the event. This hypothesis is supported by the following experimental findings:

1. The N4 is evoked only by stimuli (in any modality) that are potentially meaningful within a broad semantic system, such as words or faces—that is, precisely those stimuli that must be integrated with the context in order for their meaning to be determined

(Halgren, 1990a). (The N2 plays an analogous role for simple sensory stimuli.)

2. A large number of conditions modulate N4 amplitude to a given stimulus (e.g., sentence context, previous presentation of a semantic associate, truth of the completed event, lexical frequency, presence of the stimulus in primary or recent memory), and the size of the N4 is decreased when this information facilitates integration of the N4 within the cognitive context (Kutas and Van Petten, 1988).

3. The modulating stimuli may be in a different modality (e.g., auditory versus visual), or in a different knowledge domain (e.g., words versus faces), suggesting that the modulated network has access to all of these types of information and/or encodes information at a level deeper than modality or knowledge domain (Domalski, Smith, and Halgren, 1991).

4. Hippocampal formation and amygdala unit-firing during the N4 shows specificity both for the stimulus (i.e., a particular word or face) and for the context in which it is presented (Heit, Smith, and Halgren, 1988).

5. The N4 is the first evoked-potential component to show clearly these sensitivities to meaningfulness, specificity, and context, and is the last to occur before the response must be specified (Halgren, 1990a).

6. The global integration of an event is considered to be the essence of controlled or conscious processing, and the N2/N4/P3b has been associated with such processes via self-report and latency (see Halgren, 1993; Halgren and Chauvel, 1993, for review).

7. The extensive anatomical distribution of putative N4 generators in most or all supramodal association cortex areas provides a sufficiently broad neural substrate to encode all of the knowledge domains that contribute to an encoded event. Again, the triggering of the N4 is routed through the brain stem, inasmuch as the N4 is still evoked bilaterally after presentation of words to only the left hemisphere in patients with com-
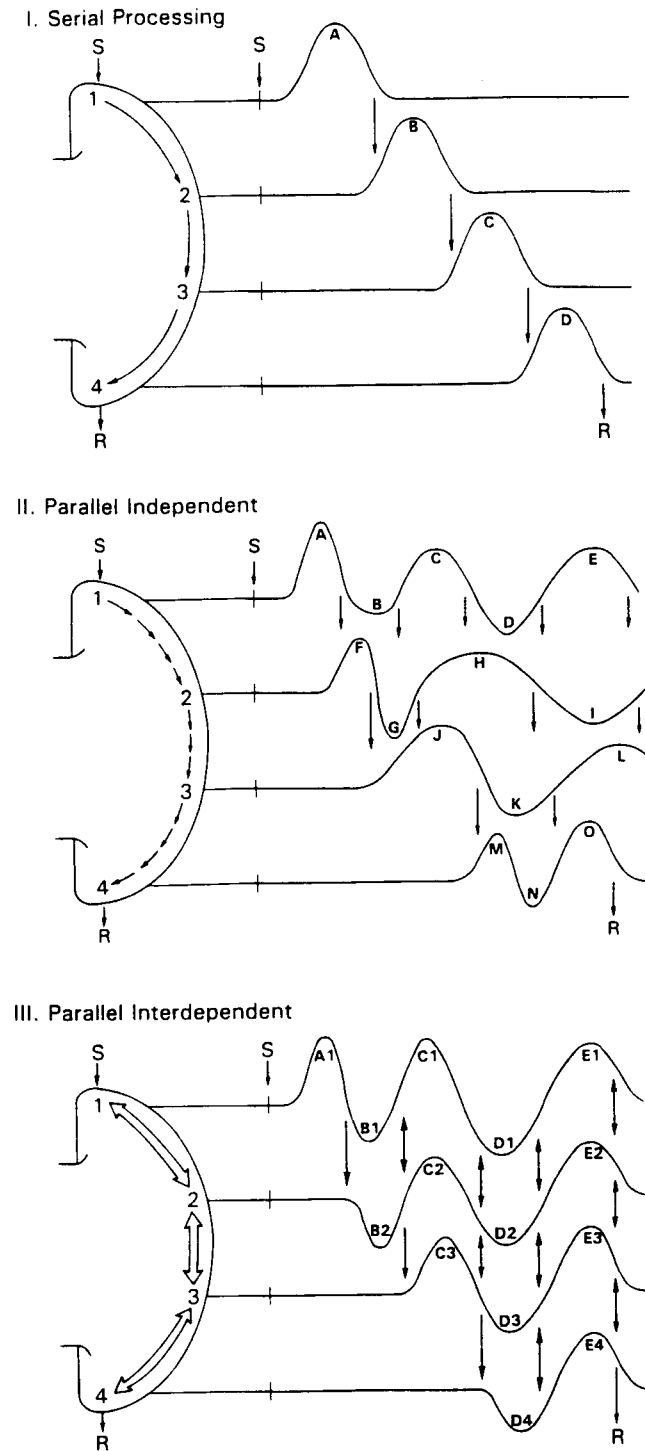

FIGURE 75.4 Cognitive evoked potential (EP) components as integrative stages of information processing in humans. A priori, one could imagine that information processing between stimulus (S) and response (R) is serial (top), with successive EP components (A, B, C, D, E) passing through successive anatomical structures (1, 2, 3, 4). Alternatively (middle), after the activation arrives in each successive structure, it could could persist indefinitely, but independently of other activated structures. What is observed is closer to the third possibility (bottom): Although the earlier stages of processing may be somewhat localized to sensory or sensory association cortex (A, B, C), the later stages of contextual integration (D, E) engage, more or less synchronously, multiple sensory association, motor association, multimodal association, prefrontal, and limbic structures (bottom: Parallel interdependent processing).

plete section of the forebrain commissures (Kutas, Hillyard, and Gazzaniga, 1988).

The P3b follows the N4 in these paradigms, and tends to be modulated by the same conditions that modulate the N4, but in the opposite direction (Halgren, 1990a). Indirect evidence suggests that the P3b represents the second phase of the same cognitive contextual integration process as the N4. The first (N4) phase would provide relative excitation to facilitate the spread of information from its sensory and memory entry points into the cognitive network, and the second (P3b) phase would provide both immediate recurrent inhibition to prevent this spread from recruiting spurious elements, and delayed recurrent inhibition to disrupt recurrent excitatory loops and thus permit the evolution of new networks (Read, Nenov, and Halgren, 1994). Consistent with this model, behavioral responses indicating successful event encoding are issued at the latency of the N4-to-P3b transition (McCarthy and Donchin, 1981).

INTEGRATION OF EMOTIONAL INFORMATION   In summary, the N4/P3b is generated in multiple association cortex and limbic areas, and has cognitive correlates and other characteristics that suggest it embodies contextual integration. The specific contribution of each area during the N4/P3b can be inferred from the effects of stimulating or lesioning it, or from the behavioral correlates of its unit activity (figure 75.4). In particular, the data reviewed above and in chapters 69, 72, and 73 would suggest that the amygdala contributes an emotional evaluation of the presented stimulus, and the lateral orbitofrontal cortex provides a psychosocial context (Halgren and Chauvel, 1993).

Thus, the fact that the limbic system is heavily activated beginning with the early stages of event encoding implies that emotional information is integrated in very early stages of event encoding. The prominent limbic activity beginning 120 ms after stimulus onset permits limbic input to shape the content of the encoded experience rather than simply to react to its content. Thus, the emotional evaluation of events occurs synchronously with their cognitive evaluation, prior to any conclusion having been obtained from that processing (see chapter 69; Plutchik, 1980; Panksepp, 1982; Ohman, 1987). Conceivably, this would allow the limbic system to contribute to the myriad psychological defense mechanisms (repression, denial,

undoing, projection, displacement, etc.) that may distort or eliminate the conscious experience of an emotionally significant event (Brenner, 1974). When sensory input is absent (e.g., during dreams), event integration would be dominated by limbic input, resulting in emotionally eloquent hallucinations.

These inferences are further supported by the potentials evoked by faces showing differing emotional expression (figure 75.3; Marinkovic et al., submitted). Facial expressions provide a rapid, emotion-specific, and largely innate system for the reliable interpersonal communication of emotional state (Izard and Buechler, 1979; Ekman, 1982). Thus, beginning with the initial face-specific component peaking at 180 ms after the face is presented, emotionally expressive faces evoke significantly different scalp potentials in comparison to unexpressive faces. Anatomically, such differences are seen as early in the visual system as the fusiform gyrus, but are much larger in later processing stages, especially in the lateral orbitofrontal cortex during the N4/P3b. Similar differences in scalp potentials are observed to emotionally significant words. These data are consistent with other studies that have generally found that, of stimuli that are perceived but otherwise equated, the scalp P3b is larger to stimuli with greater affective valence (Johnston, Burleson, and Miller, 1987; but see Vanderploeg, Brown, and Marsh, 1987).

Emotion in volition

By definition, an emotion motivates, and usually what it motivates is an action. When the action is volitional, its selection and production must integrate the conclusion of the event-encoding process with the context for action. Key structures in the volitional origins of action are the supplementary motor cortex and the underlying central cingulate gyrus, where lesions can abolish voluntary movement (see Vogt, Finch, and Olson, 1992, for review) and which are activated metabolically during the planning of voluntary movements (Roland, 1985). Electrical stimulation of the supplementary motor cortex can evoke either movement arrest or coordinated, stereotypical "postural" turning movements (Penfield and Welch, 1951; Talairach and Bancaud, 1966). Low-level electrical stimulation of the middle part of area 24 in the cingulate gyrus evokes "highly integrated types of motor behavior which are sometimes very well adapted to the situation, including

sucking, nibbling, licking and tactile exploration of the body and surrounding space" (Talairach et al., 1973).

The involvement of these structures in the formulation of voluntary movements can be monitored with the readiness potential (also known as Bereitschaftspotential), a broad negativity beginning 300 to 3000 ms before voluntary movements. Field potentials and/or unit activity correlated with the readiness potential in humans suggests generators in the precentral, premotor, supplementary motor, and central cingulate cortices (Halgren et al., 1994b; Heit, Smith, and Halgren, 1990; Groll-Knapp et al., 1980) and possibly other subcortical areas (Halgren, 1991b), with thalamic modulatory influences (Halgren, 1990b; Raeva, 1986). In simple tasks, the readiness potential appears to have more restricted generators (Neshige, Luders, and Shibasaki, 1988).

Following the same logic as for the cognitive evoked-potential components discussed previously, it may be proposed that the emotional coloring of actions is contributed during the readiness potential to the progressively defined movement (table 75.1). It is possible that the middle cingulate gyrus plays a crucial role in contributing this information, receiving input from anterior cingulate, medial temporal, orbitofrontal, and dorsolateral frontal areas, and projecting to the supplementary motor cortex (Van Hoesen, Morecraft, and Vogt, 1993). Lesions that affect these areas result in a reduction in the emotional coloring of facial expressions and speech (Kolb and Taylor, 1990; Heilman and Bowers, 1992).

Sustained emotional context

Orientation, event encoding, and response choice all occur against the background of sustained neural activity embodying the current schema of the world and of possible actions to be taken within it. Sustained specific firing has been noted in the primate frontal lobe, where different aspects of the current cognitive context appear to be held in different regions (for example, sensory and spatial information in area 46), response mappings appear to be held in premotor cortices (Goldman-Rakic, 1987; Fuster, 1989), and lesion evidence suggests that the socioemotional context may be maintained in orbitofrontal neuronal firing (see chapter 73). Although such sustained responses have not been reported, phasic responses by orbitofrontal neurons to events that fail to match contextually estab-

lished expectancies are described in chapter 72. Deficient socioemotional context would explain how orbitofrontal lesions produce impulsivity, irresponsibility, and social inappropriateness (Stuss, Gow, and Hetherington, 1992): In the absence of the socioemotional input to the N2/P3a/SW network, attention might be oriented toward irrelevant or inappropriate items; without the socioemotional input to the encoding of events during the N4/P3b, their emotional dimension may be unappreciated; and without a socioemotional input during the readiness potential, response choices could be impulsive and unrelated to the social situation.

This hypothesis can be tested because the sustained firing of frontal neurons induces a current that ultimately causes the CNV (contingent negative variation). Intracranial recordings have found evidence for CNV generation in multiple regions, especially the prefrontal cortex (Papakostopoulos, Cooper, and Crow, 1976; Groll-Knapp et al., 1980). The CNV is strongly modulated by a cholinergic pathway ascending from the brain stem (see Halgren, 1990b, for review). As predicted, the level of the CNV has been found to have a strong effect on the P3 (Rockstroh et al., 1992). This level, in turn, is strongly affected by the level of anxiety or arousal (Tecce and Cole, 1976; Proulx and Picton, 1984), as well as the presence of psychopathology, or even of normal personality variations (Rockstroh et al., 1982).

From physiology to personality

At the psychological level, sustained firing may correspond to a temporary mood, goal, or conviction. Neurobiological influences of longer duration such as hormonal levels, excitability changes, or structural modifications, may underlie such psychological constructs as constitution or temperament. These influences provide a coherence to behavior over time, becoming manifest in the emotions of the moment through their influences on the neurophysiological processes embodying orienting, event integration, volition, and context. As a whole, such influences may be considered to constitute the personality.

There are complex but nonetheless convincing differences in the orienting complex correlated with different personality types (Eysenck, 1981; Cahill and Polich, 1992). Furthermore, lesions of the limbic areas where the N2/P3a/SW is generated may ameliorate the

excessive fixation of attention by emotion in obsessional disorders and chronic pain (Teuber, Corkin, and Twitchell, 1977; Laitinen, 1979). Similarly, the P3b is abnormal in schizophrenia (Faux et al., 1987; Pritchard, 1986; Mirsky and Duncan, 1986), and such patients may have abnormalities in the medial temporal limbic structures active during event encoding (see chapters 69, 74, 76, and 77; Halgren and Chauvel, 1993).

Conclusion

In mammals, basic emotional patterns are encoded in the brain stem, but are elaborated and controlled by various limbic structures (see chapter 69). Modern data from humans support this general view, but put greater emphasis on the integration of the neocortex in emotion via the limbic structures' important, direct, and continuous role in cognition (figure 75.5). These forebrain contributions can be differentiated according to their temporal stages and core neural substrates: orienting in the cingulate gyrus and associated prefrontal and posterior parietal cortices; event encoding in the hippocampus and amygdala and associated inferotemporal and lateral-orbital cortices; response choice in the central cingulate gyrus and associated supplementary and premotor cortices; sustained context predominantly in prefrontal areas. In addition to its core substrate, each stage is triggered and modulated by brainstem circuits, and secondarily activates much of limbic and association cortex.

Thus, emotion as a directed integration of the entire organism toward a biological goal is implemented in each of these stages via the simultaneous interaction of neural networks spanning brainstem, limbic, and neocortical structures. Yet, mechanisms exist whereby different levels of the neuraxis can respond independently to biological imperatives: the brain stem by triggering fixed action patterns in response to genetically defined releasing stimuli; the forebrain by implementing different action schemata with respect to a complex world model. In both unconscious reflexes and cold calculations, emotion is absent. Rather, emotional feelings are the subjective aspect of a nervous system in which these disparate neural levels are functionally linked. Thus, emotion can be thought of as a state of communication between brain stem and forebrain in which their distinct resources are mobilized in response to an event recognized as important by either.
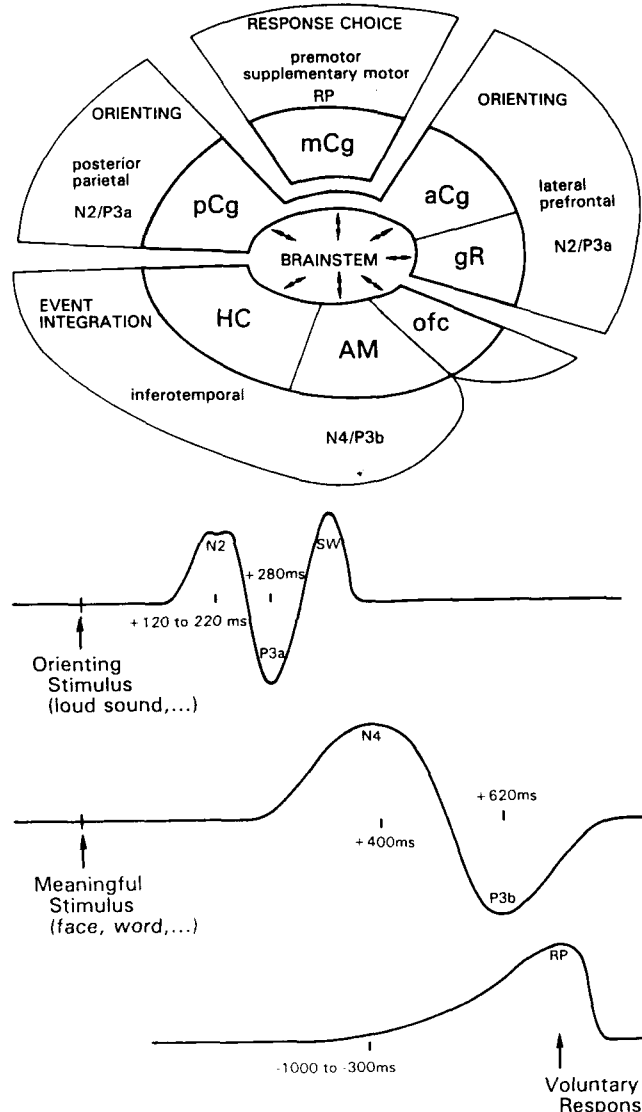

FIGURE 75.5 Limbic-neocortical networks (top) and evoked potential components (bottom) embodying different functional systems that integrate cognitive with visceral aspects of emotion in humans. Biologically imperative stimuli evoke the orienting complex and the N2/P3a evoked potential components, generated in limbic (gR, gyrus rectus; aCg, anterior cingulate gyrus; pCg, posterior cingulate gyrus) and neocortical sites (including sMg, supramarginal gyrus, and a46, area 46). During the N4/P3b, event integration takes place, integrating attended semantic stimuli with the cognitive and emotional context in limbic (AmF, amygdaloid formation; HCF, hippocampal formation; lOFc, lateral orbitofrontal cortex) and neocortical sites (including Fg, fusiform gyrus, and sTs, superior temporal sulcus). This may be followed by neurophysiological activity underlying response choice, the readiness potential (RP), generated in limbic (mCg, middle cingulate gyrus) and neocortical sites (preC, precentral cortex; preM, premotor cortex; and sMc, supplementary motor cortex).

REFERENCES

ALAIN, C., F. RICHER, A. ACHIM, and J. M. SAINTHILAIRE, 1989. Human intracerebral potentials associated with target, novel and omitted auditory stimuli. *Brain. Topogr.* 1:237–245.

AMARAL, D. G., and R. INSAUSTI, 1990. The hippocampal formation. In *The Human Nervous System*, G. Paxinos, ed. New York: Academic Press, pp. 711–755.

BANCAUD, J., F. BRUNET-BOURGIN, P. CHAUVEL, and E. HALGREN, 1994. Anatomical origin of déjà vu and vivid 'memories' in human temporal lobe epilepsy. *Brain* 117:71–90.

BANCAUD, J., J. TALAIRACH, S. GEIER, A. BONIS, S. TROTTIER, and M. MANRIQUE, 1976. Manifestations comportementales induites par la stimulation électrique du gyrus cingulaire antérieur chez l'homme. *Rev. Neurol. [Paris]* 132:705.

BARBAS, H., and D. N. PANDYA, 1984. Topography of commissural fibers of the prefrontal cortex in the rhesus monkey. *Exp. Brain. Res.* 55:187–191.

BRENNER, C., 1974. On the nature and development of affects: A unified theory. *Psychoanal. Q.* 43:532–556.

CAHILL, J. M., and J. POLICH, 1992. P300, probability, and introverted/extroverted personality types. *Biol. Psychol.* 33:23–35.

DOMALSKI, P., M. E. SMITH, and E. HALGREN, 1991. Cross-modal repetition effects on the N4. *Psychol. Sci.* 2:173–178.

EKMAN, P., 1982. *Emotion in the Human Face.* Cambridge: Cambridge University.

EYSENCK, H. J., ed., 1981. *A Model for Personality.* Berlin: Springer.

FAUX, S. F., M. W. TORELLO, R. W. MCCARLEY, M. E. SHENTON, and F. H. DUFFY, 1987. P300 topographic alterations in schizophrenia: A replication study. *Current Trends in Event Related Potential Research (EEG Suppl. 40)*, R. Johnson, J. W. Rohrbaugh, and R. Parasuraman, eds. Amsterdam: Elsevier, pp. 688–694.

FRYSINGER, R. C., and R. M. HARPER, 1989. Cardiac and respiratory correlations with unit discharge in human amygdala and hippocampus. *Electroencephalogr. Clin. Neurophysiol.* 72:463–470.

FUSTER, J. M., 1989. *The Prefrontal Cortex: Anatomy, Physiology and Neuropsychology of the Frontal Lobe.* New York: Raven.

GAGGERO, R., M. P. BAGLIETTO, F. BORAGNO, M. ODDONE, P. TOMA, and M. DE NEGRI, 1991. Epilepsy with laughing seizures, hypothalamic hamartoma and precocious puberty: Contributions of MRI, computed EEG topography (CET) and ambulatory EEG (A-EEG). *Minerva Pediatrica* 43:801–810.

GLOOR, P., 1991. Neurobiological substrates of ictal behavioral changes. In *Neurobehavioral Problems in Epilepsy.* Vol. 55, *Advances in Neurology*, D. B. Smith, D. M. Treiman, and M. R. Trimble, eds. New York: Raven, pp. 1–34.

GOLDMAN-RAKIC, P. S., 1987. Circuitry of the prefrontal cortex: Short-term memory and the regulation of behavior by representational knowledge. In *Handbook of Physiology.* Sec. 1, *The Nervous System.* Vol. 5, *Higher Functions of the Brain*, part 1, V. B. Mountcastle, ed. Bethesda, Md.: American Physiological Society, pp. 373–417.

GROLL-KNAPP, E., J. GANGLBERGER, M. HAIDER, and H. SCHMID, 1980. Stereoelectroencephalographic studies on event-related slow potentials in the human brain. In *Electroencephalography and Clinical Neurophysiology*, H. Lechner and A. Aranibar, eds. Amsterdam: Excepta Medica, pp. 746–760.

HALGREN, E., 1990a. Insights from evoked potentials into the neuropsychological mechanisms of reading. In *Neurobiology of Cognition*, A. Scheibel and A. Weschsler, eds. New York: Guilford, pp. 103–150.

HALGREN, E., 1990b. Evoked potentials. In *Neuromethods.* Vol. 15, *Neurophysiological Techniques: Applications to Neural Systems*, A. A. Boulton, G. Baker, and C. Vanderwolf, eds. Clifton, N. J.: Humana, pp. 147–275.

HALGREN, E., 1991a. Emotional neurophysiology of the amygdala within the context of human cognition. In *The Amygdala: Neurobiological Aspects of Emotion, Memory, and Mental Dysfunction*, J. P. Aggleton, ed. New York: Wiley, pp. 191–228.

HALGREN, E., 1991b. Firing of human hippocampal units in relation to voluntary movements. *Hippocampus* 1:153–161.

HALGREN, E., 1993. Physiological integration of the declarative memory system. In *The Memory System of the Brain*, J. Delacour, ed. New York: World Scientific, pp. 69–155.

HALGREN, E., P. BAUDENA, G. HEIT, J. CLARKE, and K. MARINKOVIC, 1994a. Spatio-temporal stages in face and word processing. 1. Depth-recorded potentials in the human occipital, temporal and parietal lobes. *J. Physiol. (Paris)* 88:1–50.

HALGREN, E., P. BAUDENA, G. HEIT, J. CLARKE, K. MARINKOVIC, and P. CHAUVEL, 1994b. Spatio-temporal stages in face and word processing. 2. Depth-recorded potentials in the human frontal and Rolandic cortices. *J. Physiol. (Paris)* 88:51–80.

HALGREN, E., T. L. BABB, and P. H. CRANDALL, 1977. Responses of human limbic neurons to induced changes in blood gases. *Brain. Res.* 132:43–63.

HALGREN, E., and P. CHAUVEL, 1993. Experiential phenomena evoked by human brain electrical stimulation. *Adv. Neurol.* 65:87–104.

HALGREN, E., R. D. WALTER, D. G. CHERLOW, and P. H. CRANDALL, 1978. Mental phenomena evoked by electrical stimulation of the human hippocampal formation and amygdala. *Brain* 101:83–117.

HEATH, R. G., S. B. JOHN, and C. J. FONTANA, 1968. The pleasure response: Studies by stereotaxic techniques in pa-

tients. In *Computers and Electronic Devices in Psychiatry*, N. Kline and E. Laska, eds. New York: Grune and Stratton, pp. 178–179.

HEBBEN, N., S. CORKIN, H. EICHENBAUM, and K. SHEDLACK, 1985. Diminished ability to interpret and report internal states after bilateral medial temporal resection: Case H. M. *Behav. Neurosci.* 99:1031–1039.

HEILMAN, K. M., and D. BOWERS, 1992. Neuropsychological studies of emotional changes induced by right and left hemispheric lesions. In *Psychological and Biological Approaches to Emotion*, N. L. Stein, B. Leventhal, and T. Trabasso, eds. Hillsdale,N.J.: Erlbaum, pp. 97–113.

HEIT, G., M. E. SMITH, and E. HALGREN, 1988. Neural encoding of individual words and faces by the human hippocampus and amygdala. *Nature* 333:773–775.

HEIT, G., M. E. SMITH, and E. HALGREN, 1990. Neuronal activity in the human medial temporal lobe during recognition memory. *Brain* 113:1093–1112.

IACONO, R. P., S. BLAINE, and S. NASHOLD, JR., 1982. Mental and behavioral effects of brain stem and hypothalamic stimulation in man. *Hum. Neurobiol.* 1:273–279.

IZARD, C. E., and S. BUECHLER, 1979. Aspects of consciousness and personality in terms of differential emotions theory. In *Emotion: Theory, Research and Experience*, R. Plutchik and H. Kellerman, eds. New York: Academic Press, pp. 349–366.

JOHNSTON, V. S., M. H. BURLESON, and D. R. MILLER, 1987. Emotional value and late positive components of ERPs. In *Current Trends in Event-Related Potential Research (EEG* suppl. 40), R. Johneson, J. W. Rohrbaugh, and R. Parasuraman, eds. Amsterdam: Elsevier, pp. 198–203.

JORDAN, D., 1990. Autonomic changes in affective behavior. In *Central Regulation of Autonomic Functions*, A. D. Loewy and K. M. Spyer, eds. New York: Oxford University Press.

KLEMM, W. R., and R. P. VERTES, eds., 1990. *Brainstem Mechanisms of Behavior*. New York: Wiley.

KNIGHT, R. T., 1984. Decreased response to novel stimuli after prefrontal lesions in man. *Electroencephalogr. Clin. Neurophysiol.* 59:9–20.

KOLB, B., and L. TAYLOR, 1990. Neocortical substrates of emotional behavior. In *Psychological and Biological Approaches to Emotion*, N. L. Stein, B. Leventhal, and T. Trabasso, eds. Hillsdale, N.J.: Erlbaum, pp. 115–144.

KUTAS, M., S. A. HILLYARD, and M. S. GAZZANIGA, 1988. Processing of semantic anomaly by right and left hemispheres of commissurotomy patients: Evidence from event-related brain potentials. *Brain* 111:553–576.

KUTAS, M., S. A. HILLYARD, B. T. VOLPE, and M. S. GAZZANIGA, 1990. Late positive event-related potentials after commissural section in humans. *J. Cognitive Neurosci.* 2:258–271.

KUTAS, M., and C. VAN PETTEN, 1988. Event-related brain potential studies of language. In *Advances in Psychophysiology*, P. K. Ackles, J. R. Jennings, and M. G. H. Coles, eds. Greenwich, Conn.: JAI Press, pp. 139–187.

LAITINEN, L. V., 1979. Emotional responses to subcortical electrical stimulation in psychiatric patients. *Clin. Neurol. Neurosurg.* 81:148–157.

LIVINGSTON, R. B., W. P. CHAPMAN, K. E. LIVINGSTON, and L. KRAINTZ, 1948. Stimulation of orbital surface of man prior to frontal lobotomy. *Res. Publ. Assoc. Res. Nerv. Ment. Dis.* 27:421–432.

LYYTINEN, H., and R. NAATANEN, 1987. Autonomic and ERP responses to deviant stimuli: Analysis of covariation. In *Current Trends in Event-Related Potential Research (EEG* suppl. 40), R. Johnson, J. W. Rohrbaugh, and R. Parasuraman, eds. Amsterdam: Elsevier, pp. 108–117.

MANDLER, G., 1975. *Mind and Emotion*. New York: Wiley.

MCCARTHY, G., and E. DONCHIN, 1981. A metric for thought: A comparison of P300 latency and reaction time. *Science* 211:77–80.

MCCARTHY, G., C. C. WOOD, P. D. WILLIAMSON, and D. D. SPENCER, 1989. Task-dependent field potentials in human hippocampal formation. *J. Neurosci.* 9:4253–4268.

MEYER, G., M. MCELHANEY, W. MARTIN, and C. P. MCGRAW, 1973. Stereotactic cingulotomy with results of acute stimulation and serial psychological testing. In *Surgical Approaches in Psychiatry*, L. V. Laitinen and K. E. Livingston, eds. Lancaster: Medical and Technical Publishing, pp. 39.

MIRSKY, A. F., and C. C. DUNCAN, 1986. Etiology and expression of schizophrenia: Neurobiological and psychosocial factors. *Annu. Rev. Psychol.* 37:291–319.

NAATANEN, R., and A. W. K. GAILLARD, 1983. The orienting reflex and the N2 deflection of the ERP. In *Tutorials in Event Related Potential Research: Endogenous Components*, A. W. K. Gaillard and W. Ritter, eds. Amsterdam: Elsevier, pp. 119–141.

NESHIGE, R., H. LUDERS, and H. SHIBASAKI, 1988. Recording of movement-related potentials from scalp and cortex in man. *Brain* 111:719–736.

OBRADOR, S., J. M. R. DELGADO, and J. G. MARTIN-RODRIGUEZ, 1973. Emotional areas of the human brain and their therapeutical stimulation. In *Cerebral Localization*, K. J. Zuelch, ed. Berlin: Springer, pp. 171–183.

OHMAN, A., 1987. The psychophysiology of emotion: An evolutionary-cognitive perspective. In *Advances in Psychophysiology: A Research Annual*, vol. 2, P. K. Ackles, J. R. Jennings, and M. G. H. Coles, eds. Greenwich, Conn.: JAI, pp. 79–127.

PANKSEPP, J., 1982. Toward a general psychobiological theory of emotion. *Behav. Brain. Sci.* 5:407–422.

PAPAKOSTOPOULOS, D., R. COOPER, and H. J. CROW, 1976. Electrocorticographic studies of the contingent negative variation and "P300" in man. In *The Responsive Brain*, W. C. McCallum and J. R. Knot, eds. Bristol: Wright, pp. 201–204.

PENFIELD, W. P., and P. PEROT, 1963. The brain's record of auditory and visual experience: A final summary and discussion. *Brain* 86:595–696.

PENFIELD, W., and K. WELCH, 1951. Supplementary motor area of the cerebral cortex. *Arch. Neurol. Psychiatry* 66:289–317.

PLUTCHIK, R., 1980. *Emotion: A Psychoevolutionary Synthesis.* New York: Harper and Row.

POOL, J. L., and J. RANSOHOFF, 1949. Autonomic effects of stimulating the rostral portion of cingulate gyri in man. *J. Neurophysiol.* 12:385–392.

PRITCHARD, W. S., 1986. Cognitive event-related potential correlates of schizophrenia. *Psychol. Bull.* 100:43–66.

PROULX, G. B., and T. W. PICTON, 1984. The effects of anxiety and expectancy on the CNV. In *Brain and Information: Event-Related Potentials (Annals of the N.Y. Academy of Sciences,* vol. 425), R. Karrer, J. Cohen, and P. Tueting, eds. New York: New York Academy of Sciences, pp. 617–628.

RAEVA, S., 1986. Localization in human thalamus of units triggered during "verbal commands," voluntary movements and tremor. *Electroencephalogr. Clin. Neurophysiol.* 63:160–173.

READ, W., V. I. NENOV, and E. HALGREN, 1994. The role of inhibition in memory-retrieval by hippocampal area CA3. *Neurosci. Biobehav. Rev.* 18:55–68.

ROCKSTROH, B., and T. ELBERT, 1990. On the relations between event-related potentials and autonomic responses: Conceptualization within a feedback loop framework. In *Event-Related Brain Potentials: Issues and Applications,* J. W. Rohrbaugh, R. Parasuraman, and R. Johnson, Jr., eds. New York: Oxford University Press, pp. 90–108.

ROCKSTROH, B., T. ELBERT, N. BIRBAUMER, and W. LUTZENBERGER, 1982. *Slow Brain Potentials and Behavior.* Baltimore, M.D.: Urban and Schwartzenberg.

ROCKSTROH, B., M. MÜLLER, M. WAGNER, R. COHEN, and T. ELBERT, 1992. Probing the functional brain state during P300 and CNV. *EPIC* [Hungary] 134.

ROLAND, P. E., 1985. Cortical organization of voluntary behavior in man. *Hum. Neurobiol.* 4:155–167.

ROSLER, F., D. HASSELMANN, and B. SOJKA, 1987. Central and peripheral correlates of orienting and habituation. In *Current Trends in Event-Related Potential Research (EEG* suppl. 40), R. Jr. Johnson, J. W. Rohrbaugh, and R. Parasuraman, eds. Amsterdam: Elsevier, pp. 366–372.

SMITH, M. E., E. HALGREN, M. SOKOLIK, P. BAUDENA, A. MUSSOLINO, C. LIEGEOIS-CHAUVEL, and P. CHAUVEL, 1990. The intracranial topography of the P3 event-related potential elicited during auditory oddball. *Electroencephalogr. Clin. Neurophysiol.* 76:235–248.

STAPLETON, J. M., and E. HALGREN, 1987. Endogenous potentials evoked in simple cognitive tasks: Depth compo-

nents and task correlates. *Electroencephalogr. Clin. Neurophysiol.* 67:44–52.

STOFFELS, C., C. MUNARI, A. BONIS, J. BANCAUD, and J. TALAIRACH, 1980. Manifestations genitales et "sexuelles" lors des crises epileptiques partielles chez l'homme. *Rev. Electroencephalogr. Neurophysiol. Clin.* 10:386–392.

STUSS, D. T., C. A. GOW, and C. R. HETHERINGTON, 1992. "No longer Gage": Frontal lobe dysfunction and emotional changes. *J. Consult. Clin. Psychol.* 60:349–359.

TALAIRACH, J., and J. BANCAUD, 1966. The supplementary motor area in man. *Int. J. Neurosci.* 5:330–347.

TALAIRACH, J., J. BANCAUD, S. GEIER, M. BORDAS-FERRER, A. BORRIS, G. SZIKLA, and M. BUSER, 1973. The cingulate gyrus and human behavior. *Electroencephalogr. Clin. Neurophysiol.* 34:45–52.

TECCE, J. J., and J. O. COLE, 1976. The distraction-arousal hypothesis, CNV, and schizophrenia. In *Behavior Control and Modification of Physiological Activity,* D. I. Mostofsky, ed. Englewood Cliffs, N.J.: Prentice-Hall, pp. 162–219.

TEUBER, H.-L., S. H. CORKIN, and T. L. TWITCHELL, 1977. Study of cingulotomy in man: A summary. In *Neurosurgical Treatment in Psychiatry, Pain and Epilepsy,* W. H. Sweet, S. Obrador, and J. G. Martin-Rodriguez, eds. Baltimore, Md.: University Park Press, pp. 355–362.

TUBER, D. S., G. G. BERNTSON, D. S. BACHMAN, and J. N. ALLEN, 1980. Associative learning in premature hydranencephalic and normal twins. *Science* 210:1035–1037.

VAN BUREN, J. M., 1963. The abdominal aura: A study of abdominal sensation occurring in epilepsy and produced by depth stimulation. *Electroencephalogr. Clin. Neurophysiol.* 15:1–19.

VANDERPLOEG, R. D., W. S. BROWN, and F. T. MARSH, 1987. Judgements of emotion in words and face: ERP correlates. *Int. J. Psychophysiol.* 5:193–205.

VAN HOESEN, G. W., R. J. MORECRAFT, and B. A. VOGT, 1993. Connections of the monkey cingulate cortex. In *Neurobiology of Cingulate Cortex and Limbic Thalamus,* B. A. Vogt and M. Gabriel, eds. Boston: Birkhäuser.

VOGT, B. A., D. M. FINCH, and C. R. OLSON, 1992. Functional heterogeneity in cingulate cortex: The anterior executive and posterior evaluative regions. *Cerebral Cortex* 2:435–443.

WOOD, C. C., and G. McCARTHY, 1985. A possible frontal lobe contribution to scalp P300. *Soc. Neurosci. Abstr.* 11:879–870.