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The continuous performance test: a window on the neural substrates for attention?☆

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Abstract

Attention is a complex process whose disturbance is considered a core deficit in a number of disorders [e.g., Attention Deficit Hyperactivity Disorder (ADHD), schizophrenia]. In 1956, Rosvold and colleagues [J. Consult. Psychol. 20 (1956) 343.] demonstrated that the continuous performance test (CPT) as a measure of sustained attention was highly sensitive to brain damage or dysfunction. These findings have been replicated with various populations and with various versions of the CPT. The CPT is now cited as the most frequently used measure of attention in both practice and research. Across studies, results are consistent with models of sustained attention that involve the interaction of cortical (frontal, temporal, parietal), subcortical (limbic, basal ganglia), and functional systems including the pathways between the basal ganglia, thalamus, and frontal lobes. Right hemisphere involvement (asymmetric response) is also evident across multiple studies. As such, the CPT demonstrates sensitivity to dysfunction of the attentional system whether this is due to diffuse or more focal damage/dysfunction or in conjunction with any specific disorder. CPT performance can be viewed as symptom specific (attentional disturbance), but it is not disorder specific (e.g., ADHD). Implications for neuropsychological interpretation of CPT results are presented. © 2002 National Academy of Neuropsychology. Published by Elsevier Science Ltd.

Keywords: Neural substrates; Attention; Continuous performance test; Executive function

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1. Introduction

Attention is a somewhat nebulous and complex construct in general parlance. It refers to a variety of components: (a) initiation or focusing; (b) sustaining attention or vigilance; (c) inhibiting responses to irrelevant stimuli or selective attention; and (d) shifting attention (Denckla, 1996; Mirsky, 1989; Mirsky, Anthony, Duncan, Ahearn, & Kellam, 1991). Others also have included encoding, rehearsal, and retrieval as components of attentional functions (Mirsky, 1989; Mirsky et al., 1991). In addition, the construct of attention has been conceptualized in terms of sensory attention as well as motor intention (Heilman, Watson, & Valenstein, 1985). Cohen (1993a, 1993b, 1993c, 1993d, 1993e) argued that attention is the latent neural mechanism of memory acquisition. Mirsky (1987) proposed restricting the myriad aspects of attention to the focusing of attention, sustaining of attention, and shifting of attention. Using these three components as "organizers," selective attention, for example, becomes part of the process involved in focusing attention. In contrast, sustained attention is the ability to maintain that focus over time (Mirsky et al., 1991). It has been argued that sustained attention, as well as focused attention, involves selective attention, attentional capacity, and response selection over time (Cohen & O'Donnell, 1993b). The shifting of attention is considered to reflect the need for flexibility and adaptation of various elements of attention (Mirsky, 1987; Mirsky et al., 1991; Mirsky, 1996). At the same time, components of attention are impacted by the overall arousal state of the individual (Cohen & O'Donnell, 1993b).

2. Models of attention

Multiple models of attentional processes have been posited (e.g., Goldman-Rakic, 1988; Heilman et al., 1985; Luria, 1966; Mesulam, 1981, 1985; Petersen, Fox, Posner, Mintun, & Raichle, 1989; Posner, 1988; Posner & Petersen, 1990; Pribram & McGuinness, 1975; Stuss & Benson, 1984, 1986). Functional system models of attentional processes include both peripheral autonomic and central nervous system correlates of attention (Cohen & O'Donnell, 1993a, 1993c); current functional system models include cortical and subcortical structures as well as connecting pathways and projections. Taken together, the various models of attention consistently suggest the interaction of cortical (frontal, prefrontal, parietal) with subcortical [limbic system, reticular activating system (RAS), and basal ganglia] structures as well as the pathways/projections between the basal ganglia, thalamus, and frontal lobes to form a complex functional system. Similarly, both cortical and subcortical structures, and the related descending pathways are posited to be involved with inhibition of responses (Cohen & O'Donnell, 1993c; Luria, 1973). The major models will be reviewed briefly in order to provide a backdrop against which to evaluate what has been found with continuous performance test (CPT) studies. It is important to note that while earlier models tended to emphasize the RAS, there is increasing emphasis on the frontal lobes (van Zomeren & Brouwer, 1994).

Mesulam (1981) was one of the first to offer a model of an integrated attentional system. His model was specific to understanding the phenomena of those individuals who exhibited

hemi-attention or hemi-neglect as a result of brain damage and, as such, focused on spatial location. The model, however, continues to be viewed as a viable framework for understanding general attentional processes. In his model Mesulam posited that attentional processes involved the reticular system, the limbic system, the frontal cortex, and the posterior parietal cortex. The frontal lobes are both influenced by and have an influence on the reticular system via the thalamus. This is most evident in the studies related to the orienting response (OR). In this model, the frontal lobes are involved in "fixating" or selective attention to the target as well as for other functions (e.g., scanning, reaching). The contribution of the reticular system in this model is preparedness or level of arousal as well as vigilance or maintenance of that level of arousal. Within the limbic system, Mesulam theorized that the cingulate gyrus in particular was involved in attentional processes and tied to the individual's motivational state. There are some indications that the anterior cingulate is involved in attentional processes as well (Petersen et al., 1989). Subcortical influences from the limbic system, RAS, and hypothalamus are viewed as a "systemic matrix" that is necessary for the control of attention (Mesulam, 1985). As such, sustained attention is the result of the interaction of the neural system of the frontal lobes, limbic structures, and subcortical structures (Cohen, 1993b). The orbital prefrontal cortex is seen as modulating those impulses that originate in the limbic system as well as the hypothalamus (Cohen, 1993b, 1993c). Finally, the posterior parietal cortex is viewed as providing an internal sensory map. Neural systems of the parietal lobe are essential to selective attention (Cohen, 1993b, 1993c).

A second model, the Frontal–Diencephalic–Brainstem System (FDB) was proposed by Stuss and Benson (1984, 1986). As with Mesulam's model the reticular system is hypothesized to be responsible for the individual's level of alertness. While continuing to include the reticular system and the frontal lobes consistent with Mesulam's model, Stuss and Benson placed additional emphasis on the role of the frontal-thalamic gating system and the afferent and efferent projections associated with the thalamus (e.g., to and from the reticular system and the frontal system). The frontal-thalamic gating system is seen as subserving selective attention while the thalamic projections to the reticular system subserve the stability or variability in levels of alertness (van Zomeren & Brouwer, 1994). Specific to CPT performance, the thalamic projections and the frontal-thalamic gating systems of the FDB model would be implicated in the individual's ability to attend to the task both initially and over time. With damage to the thalamic projection system, for example, the individual's level of alertness is variable and the OR is impaired. This may result in difficulty orienting to the stimulus as well as increased distractibility and an inability to sustain attention (Stuss & Benson, 1984, 1986). In contrast, damage to the frontal-thalamic gating system is associated with impaired selective attention and an inability to self-monitor arousal and sustained attention over time (Stuss & Benson, 1984, 1986).

Heilman et al. (1985) offered another model of sensory attention or inattention. As with Mesulam's model, the model formulated by Heilman and colleagues is based on studies of neglect and hemi-attention. Their model posits that the normal attention system is dependent on arousal (reticular formation), projections from sensory areas to association areas, projections to the thalamus, various portions of the cortex (frontal and parietal), and the

limbic system. The involvement of the limbic system as well as the medial and dorsolateral frontal cortex is seen as resulting from their respective inputs into the association areas, which in turn impact on the inferior parietal lobes to inhibit or facilitate attentional response. The posterior parietal lobe takes on increased importance with regard to visual and spatial information. As with Mesulam's model, the limbic system is involved with the motivational state of the individual. Thus, sensory inattention is viewed specifically as a dysfunction of the corticolimbic–reticular-formation loop that impacts predominantly on the arousal–attention component.

Luria (1966) posited that executive functions were of importance in the control of behavior with particular emphasis on the prefrontal cortex and its role in gating as well as motor behavior. The attentional system was central to his models of normal and abnormal brain function. Approaching attention from a combination of perspectives including cognitive processing (Cohen & O'Donnell, 1993c), Luria proposed two attentional systems — reflexive and nonreflexive. The reflexive system is that system that includes the OR and appears early in development. In contrast, the nonreflexive system develops at a slower rate, is the result of social learning, is associated with cognitive and linguistic mediation of behavior, and is necessary for sustained attention. Based on clinical evidence, Luria suggested that both attentional activation and inhibition were mediated by the limbic system and frontal lobes. Clinical studies, for example, found that patients with damage to the limbic system were more likely to tire easily, be distractible, and be unable to sustain attention over time. Similarly, studies of patients with severe frontal damage consistently reported difficulty with sustaining attention and resisting distractions.

The model proposed by Pribram and McGuinness (1975) is very similar to these models and involves the physiological systems associated with arousal, activation, and effort. As such, hypothetically, the neuroanatomical basis of attentional processes involves the reticular system and the limbic system as well as subcortical structures including the basal ganglia and thalamus that are involved in sensorimotor integration and control of attention. Similarly, Mirsky (1989) suggested that the reticular formation is important in the maintenance of arousal and, subsequent to this, attention. The thalamus is responsible as the relay station for projections between the reticular system and the cortical regions. The limbic system contributes to affective control and motivation. The prefrontal cortex is involved in decision making and executive function, and the parietal cortex is involved in selective and spatial attention. Mirsky also included the temporal lobes in his model; he emphasized their role in the integration of sensory information. In addition, Mirsky included the basal ganglia with importance attached to its roles in the gating of information to the frontal lobes and in the control of motoric impulses. Notably, Mirsky's system was based on factor analytic studies of neuropsychological data.

Posner and Petersen (1990) have posited an anterior–posterior and vigilance model of attention. Based on animal studies, the structures involved in the posterior network include the posterior parietal lobe (as in Mesulam's model), the lateral pulvinar nucleus of the thalamus, and the superior colliculus (Petersen et al., 1989). The posterior network in this model subserves covert shifts in orientation of the visual system (Posner & Cohen, 1984). In contrast to previous models, the role of the parietal lobes is seen as specifically involved in covert shifts of attention and attentional disengagement. Posner (1988) found

that focal injuries in humans to any of these three areas reduced the ability to shift attention from one target to another target. Consistent with Mesulam's model, Posner found that damage to the posterior parietal lobe affected the ability to shift from a target on the same side as the injury to a target located contralaterally to the injury resulting in hemi-neglect or hemi-attention. Similarly, individuals with damage to the pulvinar nucleus of the thalamus had difficulty attending to targets located contralateral to the damage. Damage to the superior colliculus on the other hand appeared to be related to a slowed response or slowed attentional shifts as well as to saccadic eye movements in response to the OR (Posner, 1988).

The anterior network is connected to the posterior system via connections between the parietal lobe and the lateral and medial frontal lobes (Goldman-Rakic, 1988). The anterior network in this model is hypothesized to be related to voluntary control of attention and focusing of attention. Posner (1988) suggested a hierarchical model such that the anterior system can transfer attentional control to the posterior system as needed (van Zomeren & Brouwer, 1994). The anterior network component of this model is more theoretical and posited to include the anterior cingulate, the midline frontal areas, and the supplementary motor areas. The third component to this model is specific to vigilance. Posner and Petersen's (1990) vigilance network is related to alertness and the ability to sustain attention. The norepinephrine system is implicated in vigilance as well and as such, the locus ceruleus, medial and lateral frontal cortex, and the posterior parietal areas of the brain are believed to be involved in vigilance. It is believed that norepinephrine works via the posterior attention system.

Probably the most complex model offered with regard to attentional processes is based on the work of Goldman-Rakic (1988) and focuses on the corticostriatothalamic (CST) neural circuits and structures. The basal ganglia, and specifically, the striatum (caudate and putamen), are believed to subserve attentional processes in conjunction with the frontal and parietal areas (Damasio, Damasio, & Chang Chui, 1980). Basal ganglia involvement is believed to be related to selective attention in visual perception and reception of stimuli in addition to motor control (van Zomeren & Brouwer, 1994). The striatum is seen as having a gating function with relays to the cortex via the thalamus resulting in selective attention (Hassler, 1978), and has emerged as a "hub" of influence over the thalamus and motor structures due to the number of crossing pathways that lead to and from the cortex (Selemon & Goldman-Rakic, 1990).

In applying this model to the study of Attention Deficit Hyperactivity Disorder (ADHD), Voeller (1991) found this model to be most consistent with the myriad of attentional problems found in such children. The model includes not only the frontal lobes, basal ganglia, and the thalamus, but also the ascending pathway from the RAS (responsible for arousal) and the descending pathways from the frontal lobes via the thalamus to the RAS (inhibition of behavior) that connect these structures and activate or inhibit other regions of the brain at the cortical or subcortical level. These pathways provide the means for transmission of the primary neurotransmitters involved in arousal (e.g., dopamine, norepinephrine, epinephrine). Thus, it is hypothesized that interference at any level of the system would lead to a cluster of clinically similar behavior (inattention, difficulty concentrating, distractibility, impulsivity, hyperactivity).

In his discussion of attentional disorders, Levy (1991) suggested that the underlying dysfunction rested in the dopaminergic circuits between the prefrontal and striatal centers. In conjunction with the dopaminergic models, it has been suggested that the frontal lobes are the locus of the attentional system, whereas the parietal lobes are involved in covert shifting of visual attention such that both work together to regulate attentional processes (Posner, Inhoff, & Fredrich, 1987). A reduction in dopamine in the prefrontal cortex has been implicated in the attentional problems associated with schizophrenia as well (Cohen & Servan-Schreiber, 1992).

3. Asymmetry of attention

It has been suggested that attentional control involves two separate neural systems. The first of these is believed to be an activation system that is centered in the left hemisphere and is involved with sequential and analytic operations. The second is postulated to be an arousal system that is centered in the right hemisphere and is responsible for holistic and parallel processing as well as maintenance of attention (Pribram & McGuinness, 1975; Tucker, 1986; Tucker & Williamson, 1984). Consistent with this, although the vigilance network posited by Posner and Petersen (1990) has not yet been mapped onto brain structures, it is hypothesized that the right hemisphere subserves the initiation and maintenance of arousal. Hemi-neglect is most frequent and more intensive after damage to the right hemisphere (Heilman et al., 1985; Mesulam, 1985). Furthermore, although the right hemisphere appears able to compensate when there is damage to the left hemisphere, the reverse is not true. Based on this, it has been hypothesized that there is asymmetry in the neurological substrates of attention with greater involvement of the right hemisphere (e.g., Heilman & van den Abell, 1980; Heilman et al., 1985). This has been further substantiated in simple reaction time studies where individuals with right hemisphere damage have been found to have longer response times as compared to those with left hemisphere damage (DeRenzi & Faglioni, 1965; Howes & Boller, 1975; Tartaglione, Birio, Manzino, Spadevecchia, & Favale, 1986). The idea of greater right hemisphere involvement is also evident in Posner and Petersen's (1990) model as well as other theories that are specific to right hemisphere dysfunction (e.g., Rourke, 1989).

In contrast to findings based on simple reaction time tasks and theoretical models that posit greater right hemisphere involvement in attention, studies with more complex reaction time tasks have found that individuals with left hemispheric damage demonstrated greater impairment on both speed and accuracy measures as compared to individuals with right hemisphere damage (Benton & Joynt, 1958; Dee & van Allen, 1973). This may be due to the difference in the extent of information processing, and as such language processing, that is required in simple vs. choice reaction time tasks. As CPTs can to some extent be conceptualized as a type of ''choice'' reaction time task, greater left hemisphere involvement would be predicted. At the level of the reticular system, damage to the left (language dominant) hemisphere is hypothesized to have a greater impact on arousal than would be evident with similar damage to the right hemisphere (Salazar et al., 1986). This would be consistent with Luria's (1973) premise that most psychological processes are related to language processes (van Zomeren & Brouwer, 1994).

4. Models of inhibition

Neural inhibition (as opposed to behavioral inhibition) is the manner in which one neural structure/system brings about the cessation of activity of another neural structure/system (Brunton, 1983). Cohen (1993b) identified four types of neural inhibition — reciprocal inhibition, antagonistic inhibition, unidirectional inhibition, and lateral inhibition. Reciprocal inhibition is said to occur when the same system is involved in both the initiation and cessation of the activity. Antagonistic inhibition involves incompatible responses (as in reciprocal), but the responses are controlled by differing structures. Unidirectional inhibition involves one subsystem having an impact on another through direct pathways; it is generally believed to involve both cortical (frontal) and subcortical structures (hypothalamus, limbic system). Unidirectional inhibition is critical to system control of behavior. Lateral inhibition is said to occur when adjacent neurons influence each other resulting in a modulated response (Cohen, 1993b). In CPT performance, with a recurring need to decide to respond or not respond, as well as the need to maintain arousal and self-monitoring of behavior (self-control), using Cohen's typology of inhibition, both unidirectional and antagonistic inhibition are most likely being measured. It is, however, unlikely that the four types of inhibition are easily disentangled when complex behavior is executed.

When all forms of inhibition are considered, the collective neurological influences to inhibition include limbic system, medial temporal lobe, cingulate, and forebrain pathways. Based on Luria's (1973) model, the caudate and hippocampus are both involved in the elimination or inhibition of responses to irrelevant stimuli such that damage to either of these areas likely to result in impaired selective attention. Cohen and O'Donnell (1993c) further indicate that the frontal/limbic systems as well as the hypothalamus are involved in the inhibition process.

5. Continuous performance tests

The very complexity of the attentional and inhibition systems makes the question of how to adequately and accurately assess the integrity of the components of these systems formidable. CPTs represent one group of paradigms for the evaluation of attention and, to a lesser degree, impulsivity. CPTs may be the most popular clinical measures of sustained attention and vigilance (DuPaul, Anastopoulos, Shelton, Guevremont, & Metevia, 1992). The basic paradigm for CPTs involves selective attention or vigilance in response to an infrequently occurring stimulus (Eliason & Richman, 1987). CPTs are generally characterized by rapid presentation of continuously changing stimuli with a designated "target" stimulus or "target" pattern; duration of the task varies but is intended to be sufficient to measure sustained attention.

The purpose of this review is to provide a qualitative synthesis of CPT research with a focus on what is currently known about the brain–behavior correlates of CPT performance and the consistency of these findings with current functional system models specific to attention, and to a lesser extent, to impulsivity or inhibition. Existing reviews of the literature and meta-analyses include only the research specific to a single population, e.g., ADHD

(Corkum & Siegel, 1993; Guevremont, DuPaul, & Barkley, 1990; Halperin, 1991; Losier, McGrath, & Klein, 1996), the effects of methylphenidate (Losier et al., 1996) or schizophrenia (Cornblatt & Keilp, 1994; Erlenmeyer-Kimling & Cornblatt, 1978). In contrast, this review will examine the existing literature in conjunction with models of attention. Contemporary models of the neurological basis of attention suggest that attention involves the interaction of cortical (frontal, prefrontal, parietal) with subcortical (limbic system, RAS, and basal ganglia) structures that comprise functional systems (including pathways between the basal ganglia, thalamus, and frontal lobes) a la Lurian theories of functional neuroanatomy. The basic paradigm that underlies the CPT are reviewed first in order to lay a foundation for later discussion.

CPTs are used frequently to obtain quantitative information regarding an individual's ability to sustain attention over time. The initial CPT was developed by Rosvold, Mirsky, Sarason, Bransome, and Beck (1956) to study vigilance. In the original task of Rosvold et al., letters were presented visually one at a time, at a fixed rate with 920 ms between presentations. The subject was to respond by pressing a lever whenever the letter "X," designated as the target, appeared and to inhibit responding when any other letter appeared (X-type CPT). Rosvold et al. also introduced a variation of this task in which the target was the letter "X", but only if the "X" was immediately preceded by the letter "A" (AX-type CPT). Rosvold et al. found the X-type CPT correctly classified 84.2–89.5% of younger subjects with identified brain damage. Group differences were found between subjects with brain damage and controls on the X-type CPT; the ability to classify subjects accurately based on CPT performance increased with the increased difficulty level of the AX-type CPT.

Since 1956, the CPT has continued to be used in the study of attention as well as impulsivity, with multiple variations in the components of the task. For example, the target stimulus in the CPT may be the letter "X," as in the original version, or a number (e.g., Gordon, 1983), a picture of an object or person (e.g., Anderson, Siegel, Fisch, & Wirt, 1969), a word (e.g., Earle-Boyer, Serper, Davidson, & Harvey, 1991). The task may be the simpler X-type CPT, or an AX-type CPT, or a further modification of the AX such that the target must be preceded by itself (XX-type; e.g., Fitzpatrick, Klorman, Brumaghim, & Borgstedt, 1992) or where color and letter are critical features (e.g., orange T followed by blue S; Garfinkel & Klee, 1983) or such that two digits in a number series (or letters in a letter string) are the same in two consecutive stimuli (Identical Pairs or IP-type, Cornblatt, Lenzenweger, & Erlenmeyer-Kimling, 1989; Cornblatt, Winters, & Erlenmeyer-Kimling, 1989). Another modification involves a change in the directions to respond except when the target is presented (not X type) as in the Conners' (1995) CPT. Other variations involve changing the modality such that presentation may be visual as in the initial version, or auditory (e.g., Earle-Boyer et al., 1991), or variable within the same task between auditory and visual stimuli (e.g., Sandford & Turner, 1995).

The target type and criteria are not the only variations among CPTs. Studies have varied the frequency of the target presentation to provide a higher frequency or a lower frequency (e.g., Beale, Matthew, Oliver, & Corballis, 1987), the duration of stimulus presentation (e.g., Chee, Logan, Schachar, Lindsay, & Wachsmuth, 1989), and the quality of the stimulus. Many adult studies used a degraded or "blurred" stimulus to increase task difficulty (e.g., Buchsbaum et al., 1990; Ernst et al., 1996; Hazlett, Dawson, Buchsbaum, & Nuechterlein, 1993; Mansour, Haier, & Buchsbaum, 1996). The time lapse between presentations of the stimuli [inter-

stimulus interval (ISI)] also has been varied with studies using a shorter interval, a longer interval, or even a variable interval (e.g., Girardi et al., 1995; Rueckert & Grafman, 1996). Variable intervals may be preset, such that for some blocks of trials, the ISI is at one rate, while for other blocks, the ISI is either longer or shorter and is "test-generated" in the sense that this is predetermined by the software program used (e.g., Conners, 1995). Another method that has been used involves an "adaptive" variable rate such that the computer program automatically increases or decreases the ISI by 5% based on the accuracy of the subject's last response (e.g., Brumm, 1994; Girardi et al.,1995; Rapoport et al., 1980; Weingartner et al., 1980). In this way, the ISI is "subject-driven" and the mean ISI is used as one index of CPT performance.

Although mean ISI may be one measure of performance on some CPT variants, at the time when Rosvold et al. introduced the CPT, the focus was on the number of correct hits as an indication of attention. Since that time other variables, including ISI, have been used as measures of attention including omission errors (number of targets not responded to), and relative accuracy (number of correct hits of total targets presented) as opposed to simple accuracy (number of correct hits). The number of commission errors (responses to stimuli other than the target) is frequently reported as a measure of impulsivity. In those studies that differentiate commission errors into false responses as opposed to delayed responses (i.e., a response was made but not within a predetermined response window), the false responses are believed to reflect impulsivity, while delayed responses are believed to be a secondary indication of inattention (Halperin, Wolf, Greenblatt, & Young, 1991). Halperin et al. (1988) suggested that there are subtypes of commission errors. Specifically, they identified a "fast reaction-time response" associated with impulsivity and hyperactivity and a "slow reaction-time response" or delayed response associated with inattention. In another study, commission errors were subtyped to include "random" errors that were not clearly associated with inattention, impulsivity, or hyperactivity (Halperin, Wolf, et al., 1991). It has been suggested that the "random" type of commission error may be reflective of dyscontrol (Halperin, Sharma, Greenblatt, & Schwartz, 1991). Based on their findings, Halperin, Sharma, et al (1991), Halperin, Wolf, et al. (1991), and Halperin et al. (1988) used combinations of commission error types and omission errors and developed various indexes including an Inattention/Passivity Index, an Impulsivity Index, and a Dyscontrol Index.

Reaction time is another measure frequently reported. Some CPTs also provide information on the standard deviation of the reaction time across blocks as a measure of consistency in responding and ability to sustain attention over time. CPTs can be viewed as measures of cognitive efficiency, such that children with memory disorders may demonstrate increased omission errors and a slower rate of responding without an associated increase in commission errors. This type of pattern is believed to support the hypothesis of difficulty with allocation of processing resources (Eliason & Richman, 1987). Slicker (1991) used an Inconsistency Index to reflect differences in variability over time, while Levav (1991) used a Sustain Factor. Still, others use the standard error of the reaction time and the standard deviation of the standard error over time as indications of consistency (Levin et al., 1996; Mahan, 1996).

Many researchers use the measures of sensitivity (d') and bias (beta) based on signal detection theory in reporting CPT scores (e.g., Keilp, Herrara, Stritzke, & Cornblatt, 1997; Klorman, Brumaghim, Fitzpatrik, & Borgstedt, 1991; Koelega, Brinkman, Hendriks, & Verbaten, 1989; Liu, Hwu, & Chen, 1997). Based on signal detection theory, the sensitivity/

bias index may be more sensitive to differences in performance on CPTs than omission or commission errors (Lam & Beale, 1991). Signal detection theory posits that the decision to respond is based on the subject's setting him/herself a certain standard or criterion for responding. Sensitivity is dependent on the intensity of the stimulus and the sensitivity of the individual and reflects the individual's ability to discriminate among stimuli. Bias is presumed to relate to the strategy used in making the decision to respond or the individual's response style. Concerns have been raised by some, however, as to the applicability of signal detection theory to CPT performance on CPT tasks that do not provide sufficient signal to noise ratio due to insufficient length of the task and the proportion of targets to nontargets (e.g., Jerison, 1967; Parasuraman, 1979).

It has long been recognized that CPT performance is impacted by brain damage or dysfunction. The findings of Rosvold et al. (1956) have been replicated with various populations and various versions of the CPT (Reynolds, Lowe, Moore, & Riccio, 1998). A number of studies with children and adults with known lesions, head injury, and epilepsy have looked to identify the brain–behavior correlates of CPT performance. Other researchers have investigated the brain–behavior relationships to CPT performance using event-related potentials (ERPs) during CPT testing. Positron emission tomography (PET), single photon emission computed tomography (SPECT), and near infrared spectroscopy (NIRS) studies with normals, as well as with individuals with schizophrenia or attention-deficit hyperactivity disorder, have been completed in order to better identify the neurological substrates involved in attention as well as inhibition.

6. Methodology

The literature search specific to CPTs included review of citations listed in PsychInfo (310 entries), Medline (192 entries), and ERIC (13 entries) from 1966 to 1999 using "continuous performance test" as a search prompt; elimination of duplicate records resulted in 342 unique citations. Several of the studies cited in the databases, however, are not available in English and therefore could not be included. Dissertation abstracts was used as a source with particular attention to those dissertations completed within the past 5 years; in the event the dissertation had been published, the publication (as opposed to the dissertation or dissertation abstract) was included. Additional references were generated from review of the cited articles. In some cases, while the research reviewed included a vigilance task, it was determined that the task used was not a "continuous performance test." Due to the vast variety of vigilance tasks as well as the variety of CPTs available, only those studies that included a vigilance task that constituted a CPT were included. For this review, the defining characteristics of a CPT were based on the criteria of the Rosvold et al. (1956) paradigm such that the task had to involve the presentation of constantly changing stimuli with some clearly defined "target" stimulus or pattern that occurred at a low frequency relative to the number of stimuli presented over the duration of the task. Based on these criteria, tasks that involved responding only when some aspect of a stimulus (e.g., clown's nose, star on wing of plane) changed color or lit up (e.g., Goldberg & Konstanareas, 1979; Kirchner, 1976) were excluded (for a review of various vigilance tasks in general, see Koelega, 1989, 1992; Koelega & Verbaten, 1991).

Although predominantly qualitative in nature due to the variations of CPTs in the literature, meta-analysis was conducted on those 26 studies that addressed differences in P3 to targets and nontargets. Effect size (ES) was calculated when sufficient information (descriptive or inferential) was provided in the study. When means and standard deviations were reported, the ES was calculated by dividing the difference in amplitude at the parietal site or Pz (target–nontarget) by the pooled standard deviation (Glass, 1977). When descriptive data were not reported, the ES were derived from inferential statistics using procedures set forth by Wolf (1986). Amplitude at Pz was selected, as it was the only variable consistently reported across studies.

7. CPT performance: relation to neural substrates

Cohen and O'Donnell (1993a) proposed that differential impact on the attentional system can occur depending on the localization of the lesion. More diffuse damage typically impacts more structures and pathways and results in greater impairment to the attentional system. Based on their formulation and review, selective or focused attention is hypothesized to be most impaired following damage to the parietal lobes; less impairment is believed to be associated with damage to the frontal lobes or RAS. Processing speed can be determined through the use of an adaptive ISI, measured through actual reaction time data, or inferred by evaluation of error types. It is posited to be most impacted by hypothalamic lesions, but also impacted by damage to the parietal lobes and RAS. Vigilance (i.e., the readiness to respond or alertness) and persistence (i.e., the ability to sustain the level of alertness) are both impacted most by damage to the frontal lobes and hypothalamus with mild impairment evident with lesions to the parietal lobe. While the limbic system is seen as more important to persistence, the RAS is viewed as having a stronger impact on vigilance (Cohen & O'Donnell, 1993c). Theoretical differences in the models (i.e., relative importance of structures of the basal ganglia, the hypothalamus, the RAS, the frontal lobes) have yet to be explored or demonstrated conclusively. Despite these differences, there is general agreement that the ability to direct and sustain attention to tasks is compromised by both neurological and psychiatric problems.

When Rosvold et al. initially developed the CPT and demonstrated its sensitivity to brain damage, the subjects included a variety of groups including those with mental retardation (known and unknown etiology) and those who were identified as "brain damaged." Since that time others have replicated the sensitivity of CPT measures with subjects with more general types of brain damage (e.g., Alexander, 1973; Schein, 1962). In addition, a number of CPT studies have been completed that were specific to individuals with traumatic brain injury (TBI), stroke, or seizure disorder. These studies are reviewed here in addition to studies that have used electrophysiological methods or other measures of brain activity (e.g., PET, regional cerebral blood flow, and so on) in conjunction with CPT performance.

7.1. TBI/lesion studies

A total of 20 studies included subjects with TBI; 1 study included children with identified lesions; and 1 included subjects with multiple infarcts (see Table 1). In the majority of the

Table 1
Physiological/neurological correlates of CPT performance in individuals with TBI or identified lesions

Study	CPT type/ stimulus/ISI	Mean age, years (S.D.)	*N*	Groups	Results
Arcia & Gualtieri, 1994	X/L/1000/visual	29.93 (11.02) 23.42 (9.30) 27.12 (9.80)	36 23 35	mild TBI ADD controls	Mild TBI group had no focal deficits on neurological exam. Both TBI and ADD demonstrated more difficulty with sustained attention; TBI had slower reaction time, while ADD was more impulsive based on types of errors made.
Baker, 1990	AX/L/1500/visual	11 (2.73)	8	TBI/ADHD	Mean number of months since TBI = 20.73 months (12.37). Sample was impaired on d′, bias, omission errors, and commission errors. Lesion location was not specified in study.
Burg, Burright, & Donovick, 1995	AX/N/800/visual	40 41	30 25	TBI controls	TBI group demonstrated mild to moderate deficits on vigilance and distractibility tasks; no difference found on delay task. Significant (highest) correlations found between vigilance total correct and Stroop 2 ($r = -.52$; $P < .01$), GDS distractibility correct ($r = .57$, $P < .001$), and delay task correct ($r = .66$, $P < .001$) for TBI. Correlations differed for control group; it was suggested that the ceiling effect on vigilance task may have affected correlations. Lesion location was not specified.
Chadwick, Rutter, Brown, Shafer, & Traub, 1981	X/L/920/visual	Children (age not reported)	28 28	TBI controls	At 1-year postinjury, no difference on "fast correct" or "false positive" responses. Lesion location was not specified.
Cicerone, 1997	X/L/1000/auditory MultipleX/L/ 1000/auditory AX/L/1000/auditory	34.6 (9.7) 33.3 (12.4)	50 40	TBI controls	Significant between group differences ($P < .001$) found for omission, commission, and total errors; greatest difference was on total errors. Lesion location was not specified.
Dennis, Wilkinson, Koski, & Humphries, 1995	AX/N/800/visual	11.0 (3.2)	83	TBI	Overall, sample showed impaired attention. A total of 64% of the children were identified as abnormal or borderline. Age at injury, coma scale score, nor time since injury were found to be significant predictors of performance.
Ewing-Cobbs et al., 1988	X/O/adap/visual	age not reported	57 34	severe TBI mild-moderate TBI	Subjects were evaluated 5–8 years postinjury; significant age by ISI effect. Mild-moderate and severe were more impaired in younger groups; severe TBI were more impaired in older group. Also, significant age by severity interaction for commission errors with distractor. Lesion location was not specified.

Gribble, 1989	X/N/1400/visual	28.73 (6.47)	10	ataxic TBI	All groups showed slight decrement in performance over time; ataxic and nonataxic TBI both performed slower than controls; no difference on commission or omission errors; lesions not localized in study
		30.3 (6.94)	10	non-ataxic TBI	
		24.8 (4.04)	10	control	
Katz et al., 1996	AX/L/nr/visual	6.75 (0.44)	22	no lesion	Children in lesion groups were all premature. Mean differences among groups of premature were not significant. Children with severe lesions made significantly more errors of omission and commission than controls. Children with mild lesions were poorer than controls on commission errors; no lesion premature group made more errors than controls. Lesion location not specified in study.
		6.64 (0.41)	32	mild lesion	
		6.89 (0.59)	10	severe lesion	
		6.78 (0.47)	40	control	
Kaufmann, Fletcher, Levin, & Miner, 1993	X/F/adap/visual	12.4 (3.3)	11	mild TBI	Duration of impaired consciousness associated with CPT performance. Severe head injury associated with impaired CPT performance 6 months after injury. Lesion location not specified in study.
		11.5 (2.5)	13	moderate TBI	
		9.6 (2.1)	12	severe TBI	
		11.1 (2.8)	36	control	
Laidlaw, 1993	AX/L/500/visual	27.85	21	TBI	Modified task such that subjects had to respond yes/no for each trial. TBI group differed significantly on CPT for hits, misses, and false alarms ($P < .05$). Lesion location not specified in study.
		24.32	21	volunteers	
Levin et al., 1986	AX/L/adap/visual	adults	16	TBI	Study did not include controls and no comparison was made to normative data. Shortest mean ISIs were with subjects with bilateral frontal, right frontotempoparietal, and bilateral frontotemporal lesions. Longest ISIs were associated with focal damage to basal ganglia/midbrain (right and bilateral) and brainstem.
Loken, Thornton, Otto, & Long, 1995	X/CF/var/visual	29.0 (11.6)	20	severe TBI	TBI subjects were evaluated 1–9 months postinjury ($x = 2.9$ months). CPT used two-, four-, eight-choice pattern with warning stimulus; ISI varied from 2000–5000 ms. For commission errors, no main effect for group or interaction effect, but there was a main effect for complexity (more errors on two- and eight-choice tasks), and trial block (more errors in early and late blocks). For omission errors, there was a main effect for group for four- and eight-choice task with a linear increase in errors by the TBI group. For CPT latencies, there was no Group × Complexity × Trial block interaction; the Group × Trial block was significant with TBI group latencies increasing over duration of task. Group × Complexity was also significant; overall controls were faster than the TBI group (main effect for group).
		29.1 (10.3)	20	controls	

(continued on next page)

Table 1 (*continued*)

Study	CPT type/ stimulus/ISI	Mean age, years (S.D.)	*N*	Groups	Results
Parasuraman, Mutter, & Molloy, 1991	X/N/1000/visual (standard and degraded)	29.7 28.6 21.1	10 10 15	mild TBI control college students	TBI had lower d′ for degraded stimuli but not standard CPT; TBI also had lower hit rate and higher false alarm rate than either control group. Lesion location was not provided.
Ponsford & Kinsella, 1992	AX/C/2000/visual	26.1 (8.0) 25.6 (8.1)	19 20	severe TBI orthopedic	Subject required to respond on all trials. For accuracy, there was a main effect of time but no significant group effect for accuracy. For latency, TBI group was significantly slower (main effect for group). For both groups, reaction time to target was slower than to nontarget. Lesion location was not specified.
Ringholz, 1989	AX/N/adap/visual	24.1 (3.5) 26.3 (7.9) 23.2 (3.1)	29 28 25	TBI — dorsolateral TBI — orbital control	Both TBI groups differed from control on all but vigilance decrement; no difference between dorsolateral and orbital groups on CPT performance.
Risser & de S. Hamsher, 1990	AX/N/800/visual	18–60	44	TBI	All subjects were in motor vehicle accidents. As a group, subjects performed within normal limits on vigilance, but had difficulty with distractibility task. Those with most diffuse brain injury had the most difficulty on the distractibility task.
Rueckert & Grafman, 1996	X/L/var[1]/visual	45.2 46.6 50.9	11 10 16	left frontal right frontal control	Right frontal group had longer reaction times, missed more targets, and had greater decrement with time on CPT. Lesion size did not affect performance. Those with callossal involvement missed more targets over time, but did not differ from the control group on reaction time or commission errors.

Rueckert & Grafman, 1998	X/L/var[1]/visual	48.2 47.3	4 6 6	left posterior right posterior control	Both posterior lesion groups had more omission errors. Difference between groups increased with time on task. Posterior groups performed worse when stimuli presented at faster rate.
Schein, 1962	X/L/1000/visual AX/L/1000/visual	41.3 (12.3) 31.5 (13.9)	53 25	TBI control	Of TBI group, 42 had cortical damage; 11 had subcortical damage. No between group differences on X-CPT, however, AX-CPT differentiated groups with TBI group demonstrating impaired performance. Lesion location was not specified.
Timmermans & Christensen, 1991	AX/N/800/visual	10.72 (3.67)	38	TBI	Performance differed significantly from normative data for group. Majority of subjects' scores for commission and omission on vigilance task were in the borderline or abnormal range. Lesion location was not specified.
Wolfe et al., 1990	AX/L/nr/auditory	64.6 (6.0) 63.0 (8.6)	11 11	infarcts controls	Infarcts were multiple and subcortical; 9 of 11 had infarcts to basal ganglia; 2 of 11 had infarcts to thalamus. Based on group data, significant difference on omission errors with infarct group making more errors than the controls.

ISI = interstimulus interval; Stimulus: L = letters, N = numbers, C = color, F = form/shape, O = object; adap = adaptive rate; var = variable rate; var[1] = two rates: fast = 800 ms, slow = 1800 ms; nr = not reported; d′ = sensitivity; ADD, ADHD = attention deficit hyperactivity disorder.

studies, the subjects with TBI were grouped by lesion severity based on coma duration with no specific information provided on the lesion location. Across studies, the CPT performance of subjects with TBI, lesions, or infarcts were significantly below that of controls. For those studies using the CPT developed by Gordon (1983), the Gordon Diagnostic System, deficits were noted on vigilance as well as distractibility tasks with increased deficits on distractibility associated with more diffuse brain injury. In one study (Levin et al., 1986), it was noted that the three subjects with the longest ISIs ($X = 2829.17$ ms) included the only two subjects with damage to the basal ganglia and midbrain (one with right lesions only, one with bilateral lesions) and the subject with damage to the brainstem. The remainder of cases had damage (bilateral or unilateral) to the frontal or frontotemporal areas. In the Wolfe, Linn, Babikian, Knoefel, and Albert (1990) study, where the majority of subjects had infarcts to the basal ganglia, significant omission errors were found. Taken together with Levin et al., results suggest that CPT omissions with a nonadaptive ISI are related to functioning of the basal ganglia in particular with more diffuse damage impacting on distractibility as well.

Two studies included subjects with frontal damage (Ringholz, 1989; Rueckert & Grafman, 1996). Ringholz found that both dorsolateral and orbital frontal lesions impacted on overall attention but found no difference between the two lesion groups. In comparing left and right frontal lesion effects on CPT performance, Rueckert and Grafman found that the right frontal group had longer reaction times, more omission errors and the greatest vigilance decrement relative to the left frontal and control groups. As such, although the subjects did not demonstrate the extreme ISIs of the basal ganglia/brainstem subjects in Levin et al. (1986), subjects with frontal damage also demonstrated attentional deficits. Indications from at least one study suggested that the effect is greatest if the damage is to the right frontal area.

7.2. Seizure disorders

Nine studies have used CPTs with subjects who had seizure disorders (generalized, complex partial, temporal lobe). Based on these studies, the presence of generalized seizures is associated with impaired CPT performance (increased omission errors, increased reaction time) regardless of age (Brandt, 1984; Campanelli, 1970; Goldstein, Rosenbaum, & Taylor, 1997; Hara & Fukuyama, 1992; Lansdell & Mirsky, 1964; Miller, 1996; Mirsky, Primac, Marsan, Rosvold, & Stevens, 1960; Mirsky & van Buren, 1965). In contrast, individuals with temporal lobe epilepsy (right or left) tended to perform comparably to controls (Brandt, 1984; Goldstein et al., 1997). All adult studies and most child studies that compared subjects with generalized vs. temporal lobe or complex partial seizures found that with more diffuse damage (generalized) the performance declined with a higher frequency of omission errors. For example, Miller (1996) compared children with generalized epilepsy, children with complex partial epilepsy, and control children. The children with generalized epilepsy made significantly more omission errors suggesting attentional problems. In contrast, no significant group differences were found for commission errors. Lansdell and Mirsky (1964) found that the duration of seizure disorder (years since onset) was negatively correlated with CPT performance on both X-CPT ($r = -.40$) and AX-CPT ($r = -.47$). Only the Aman, Werry, Paxton, and Turbott (1987) study with children found no difference in the performance of subjects with generalized seizures as compared to

subjects with complex partial seizures. Overall, results suggest that CPT performance is sensitive to seizure disorder type. Generalized seizures and longer duration of the disorder have been associated with poorer CPT performance.

7.3. Evidence from electrophysiology and tomography studies

Physiological responses as measured by electroencephalography (EEG), PET, SPECT, NIRS, or other methods, can provide direct indications of the physiological mechanisms underlying attention or inhibition as measured by CPT performance. With regard to attention, the physiological responsiveness should provide some measure of allocation of attentional resources (Cohen & O'Donnell, 1993b). Studies on the OR and changes in EEG to the OR provide a basis for assuming that attentional processing can result in changes to brain electrical activity (Cohen & O'Donnell, 1993b).

7.3.1. EEG studies

Seven studies included EEG measures during performance of a CPT (see Table 2). One study included subjects with seizure disorder and found that there was an association in correct responses with seizure related "burst" activity (Mirsky & Van Buren, 1965). Specifically, it was found that correct responses declined immediately prior to and during "burst," and then improved sharply as the "burst" ended and normal EEG pattern returned. Across studies that used EEG, it has been found that CPT activation is associated with increased frontal, frontotemporal, and temporal beta activity, suggesting increased alertness and attentiveness to the environment. This increased beta was more evident in the right frontal and right frontotemporal than in the corresponding areas of the left hemisphere (R > L asymmetry). In contrast, there is a decrease found in alpha (associated with restfulness) and theta (associated with the transition from sleep to wakefulness) wave forms in the posterior portion of the brain, and better CPT performance was associated with a greater anterior to posterior gradient. Consistent with this, increased frontal and frontotemporal theta activity was associated with increased omission errors.

7.3.2. ERP studies

An alternative to EEG measurements is the use of ERPs. By using averaged ERP, many of the problems with measuring EEG responses (e.g., time, signal-to-noise ratios) can be reduced, and it has been found that ERP components are highly reactive to attentional processes (Cohen & O'Donnell, 1993a). The most common paradigm for ERP study is the oddball paradigm. In this paradigm, the subject is to attend to a rare target (e.g., higher tone) among more frequently occurring nontargets (e.g., lower tone). It has been found that the target results in N2 and P3 components followed by a slow wave, while nontargets produce N1 and P2 components. These findings would be consistent with interpretations P3 as reflective of decision-making and cognitive processes (Snyder, Hillyard, & Galambos, 1980). N1 has been interpreted as reflecting attention independent of response, and tends to be lower in children with hyperactivity (Loiselle, Stamm, Maitinsky, & Whipple, 1980; Prichep, Sutton, & Hakarem, 1976), as well as in individuals with alcoholism (Porjesz & Begleiter, 1979) or schizophrenia (Brecher & Begleiter, 1983). N2 tends to be modality-specific with

Table 2
Physiological/neurological correlates based on EEG studies

Study	CPT type/ stimulus/ISI	Mean age, years (S.D.)	*N*	Groups	Results
Costa, Arruda, Stern, Somerville, & Valentino, 1997	XX/L/500/ auditory X/Word/1200/ auditory XX/word/ 1200/auditory	32.7 (6.0) 33.1 (5.8)	11 11	HIV+ controls	Target "word" is by semantic category; activation of left hemisphere greater for words (semantic) than letters. Significant difference for fast beta such that XX-word > X-word, XX-word> resting, and XX-letter>resting. Also significant for delta, such that XX-word>X-word, XX-word >resting, and X-word>?resting.
Hoffman et al., 1991	X/N/nr/visual	33.7 34.6	13 9	schizophrenic normal	Found relationship between decreased prefrontal alpha and d′ in schizophrenics. Correlation between F_4-O_s and F_4-T_6 and d′ significant for schizophrenics, but not for normals.
Mirsky & Van Buren, 1965	X/L/var/visual XX/L/var/ visual X/L/var/ auditory XX/L/var/ auditory	22 13–42	18	epilepsy	For those subjects whose evidenced burst, there was a significant ($P < .03$) drop in correct responses in pre-burst period (1–2 s) with marked drop in performance once burst-evident; then steady and sharp improvement in performance beginning in last 5 s of burst and continuing past the occurrence of the burst. Tendency for errors to occur during EEG burst was significant ($P < .00001$) for pooled visual and auditory forms of the CPTs.
Schein, 1962	X/L/1000/ visual AX/L/1000/ visual	41.3 (12.3)	53	TBI	Of TBI group, 15 demonstrated normal EEGs, 14 demonstrated diffuse impairment on the EEG, 13 demonstrated focal impairment on the EEG, and 3 had borderline normal EEGs. When CPT performance was examined based on EEG performance, the group with diffuse impairment on the EEG was significantly impaired on the CPTs as well.
Teixeira, 1993	AX/L/500/ auditory	19.3 (1.49) 19.1 (1.46)	20 20	depressed not depressed	CPT was done twice. No difference between groups in accuracy or commission errors. CPT performance is associated with increased delta activity in frontal, and frontal-temporal regions as compared to resting state. Theta significant at

(continued on next page)

Table 2 (*continued*)

Study	CPT type/ stimulus/ISI	Mean age, years (S.D.)	*N*	Groups	Results
					frontal. Alpha significant at frontal-temporal and temporal. Beta 1 significant at frontal, frontal-temporal and temporal; beta 2 significant for frontal and temporal regions.
Valentino, Arruda, & Gold, 1993	XX/L/500/ auditory	Adults (age not reported)	27 27	students — high students — low	EEG for two groups of undergraduates rated as either high or low vigilance. CPT performance is associated with increased beta in frontal, frontotemporal, and temporal and decrease in alpha and theta in posterior portion. Frontotemporal, anterior slow waves increased. Better vigilance performance on CPT associated with greater anterior to posterior gradient.
Weiler, 1992	XX/L/500/ auditory	19.8 (1.49)	102	students	Frontal beta, frontal and right frontotemporal theta correlated with omission errors ($P < .05$). Right frontotemporal, right temporo-occipital, right temporal theta, occipital theta, and right occipital delta increased with CPT, and all were correlated with vigilance decrement ($P < .05$).

ISI = Interstimulus interval; Stimulus: L = letters, N = numbers, C = color, F = form/shape, O = object; var = variable rate; nr = not reported; EEG = electroencephalography; CPT = continuous performance test; TBI = traumatic brain injury.

peak amplitudes at different locations depending on the modality of stimuli presentation and occurs regardless of whether the stimulus is a target or nontarget. P3, however, does not appear to be modality-specific and the peak amplitude is in the parietal area (Cohen & O'Donnell, 1993c). In addition, medial temporal lobe and frontal lobe structures may be involved (Okada, Kaufman, & Williamson, 1983; Squires, Halgren, Wilson, & Crandall, 1983). Although it is believed that the medial temporal lobe may be involved in attentional processes, lesions to the medial temporal lobe have little impact on P3 (Cohen & O'Donnell, 1993a). Furthermore, it has been found that N2 and P3 are resistant to habituation effects (Cohen & O'Donnell, 1993a). Most CPTs differ from the oddball paradigm in that the nontarget stimuli may be variable (e.g., multiple digits or letters may be the nontarget). Additionally, as noted earlier, there are multiple variations to the CPT that may also impact on results of ERP studies. For example, ERP studies suggest that selection by color (selective attention) may be subserved by different mechanisms with greater activity evident in the left hemisphere as compared to selection by spatial location that would result in greater activity in the right hemisphere (Harter, Aine, & Schroeder, 1982; Hillyard & Munte, 1984).

Thirty-five studies were found that looked at ERPs during CPT performance; 33 of these involved visual stimuli, 1 involved auditory stimuli, and 1 involved both auditory and visual stimuli. Across studies, P3 was maximal at the Pz for targets (e.g., Coons, Klorman, & Brogstedt, 1987; Coons et al., 1981; Friedman, Vaughn, & Erlenmeyer-Kimling, 1978, 1981; Kaskey, Salzman, Cicone, & Klorman, 1980; Klorman et al., 1991; Leuthold & Sommer, 1993; Michael, Klorman, Salzman, Borgstedt, & Dainer, 1981; Stamm et al., 1982; Strandburg et al., 1990, 1994; Verbaten, Overtoom, Koelega, & Swaab-Barneveld, 1994), but differences were noted based on CPT differences in stimuli, latency, and ISI. Task variations that have been found to impact on P3 amplitude and latency include variations in task frequency (Sutton, Braren, Zubin, & John, 1985), relevance or semantic loading (Sutton et al., 1985), and shorter ISI (Campbell, Courchesne, Picton, & Squires, 1979). Changing the signal rate (ISI) of the CPT produced changes in latency as well as changes in the distribution of the ERP components with a significant correlation between the ISI of the CPT and P3 latency (Leuthold & Sommer, 1993; Näätänen, 1992).

Another group of findings relates to reaction time and its relation to P3 latency. Wagner, Kurtz, and Engel (1989) found that the variable ISI correlated not only with P3 latency but also with CPT reaction time. Stimulus intensity and difficulty of discrimination have been found to be related to P3 latency (Papanicolaua, Loring, Raz, & Eisenberg, 1985), suggesting that a longer reaction time would be expected on CPTs with degraded stimuli. In contrast, where task difficulty is increased by other factors (e.g., AX-CPT vs. X-CPT), only the reaction time and latency, but not the amplitude of the P3 is impacted. Reaction time has been linked to the amplitude and latency of ERPs (N2 and P3), such that it is believed that the vigilance decrement is analogous to a slowing (increased latency) of the reaction time and P3. This is evident in the significant correlation between CPT reaction time and P3 latency and amplitude (e.g., Wagner et al., 1989). It has also been found that changes in arousal are associated with faster reaction time and higher error rates on vigilance tasks (Cohen & O'Donnell, 1993b). As a result of the association between reaction time and P3 latency, it has been argued that reaction time on CPTs may be interpreted as a direct measure of executive functioning of the brain (Davies & Parasuraman, 1977).

Twenty-five studies examined the differences between P3 to targets and nontargets in terms of structures involved, as well as latency and amplitude (see Table 3). For the X-CPT, there is a significant difference in P3 for target vs. nontarget stimuli (referred to as P3b) for both amplitude and latency with parietal maximum for the target (e.g., Coons et al., 1981; Klorman et al., 1983, 1991). ES for target–nontarget differences for amplitude support the notion that performance on the X-CPT (respond only when "X" appears) is reflecting differential brain activity (Table 4). For the X-CPT, the difference in P3 amplitude between the target stimulus (e.g., "X") and the nontarget stimuli (e.g., any other letter) was significant with a mean ES across 15 studies of 1.46 (S.D. = 0.67). Although less impressive, similar findings emerged for the difference between target sequence and nontarget sequence on the AX-CPT or XX-CPT, where all possible nontarget sequences were combined (mean ES of 0.48, S.D. = 0.31; 1.03, S.D. = 0.69, respectively across seven studies each). Notably, for AX-CPT and XX-CPT, studies suggest significant differences in P3 for target vs. nontarget based on the type of nontarget. For example, Roberts, Rau, Lutzenberger and Birbaumer (1994) investigated the differing response to relevant vs. irrelevant nontargets

using an AX-CPT (letters, visual) with 21 normal adult males with a mean age of 33.4 years. A "relevant" nontarget sequence included one of the two required stimuli (A or X) but not the other. Thus, the two relevant nontarget conditions could be designated AN and NX. The "nonrelevant" stimulus sequences included neither A nor X and were designated NM. Differences in P3 amplitude or latency between target and nontarget stimuli were noted to vary depending on the saliency of the nontarget (i.e., whether it included A or X but not the other).

In addition, latency of P3 was related to nontarget type with nontarget sequences including X having the longest latency (Roberts et al., 1994). Paralleling the differences in amplitude, differences have been found for latency with longer latency at the frontal site for relevant nontargets (i.e., those sequences that include either X or A) and the shortest latency for nontarget sequences that do not include A or X. The greatest differences between target and nontarget P3 were found at frontal and central sites for both latency and amplitude. Consistent with the findings of Schupp, Lutzenberger, Rau and Birbaumer (1994), a longer latency in response to nontarget sequences that contained X as compared to the shorter latency of response to the target (AX or XX) was found at the anterior central site (Gevins et al., 1989). This suggests initiation of widespread centrofrontal positivity in response to the nontarget sequences that included X that may reflect response inhibition of the motor response (i.e., inhibiting the response to the X when it is determined that the AX or XX sequence was not present). Thus, these differences in physiological response that parallel differences in nontarget salience support the idea of involvement of the prefrontal cortex in interruption of response activation (Gevins et al., 1989). These differences of amplitude and latency in ERP components to relevant vs. nonrelevant nontargets also provides physiological support to Halperin and colleagues' (e.g., Halperin, Sharma, et al., 1991; Halperin, Wolf, et al., 1991) assertions that the types of errors may be as important as the number of errors made when interpreting CPT performance.

Attentional asymmetry has been addressed only minimally in ERP studies. Asymmetry (R > L) has been found for P1, N1, and P2 in normals for targets. In contrast, P3 had leftward asymmetry for nontargets (Roberts et al., 1994; Strandburg et al., 1994). This would suggest that although attention and decision making are associated with right hemisphere activation in response to target (relevant) stimuli, it may be that the decision making associated with irrelevant stimuli (i.e., selective attention) is associated with increased left hemisphere activation. However, since most studies did not address asymmetry, any conclusions must be preliminary. The differential involvement of right and left attentional systems is an area in need of further investigation.

7.3.3. PET, SPECT, MRI, and NIRS studies

Use of tomographic and imaging methods provides additional information with regard to actual brain structures that are involved with task performance, particularly with regard to subcortical structures. Twenty-three studies used PET scans, two used SPECT scans, one used MRI, one used functional MRI (fMRI), and one used NIRS. Based on their review of selected PET studies, Posner and Petersen (1990) concluded that the right posterior parietal and frontal areas are involved in vigilance and that sustained attention as measured by CPT performance results from activation of the frontal system. On PET, normals have increased/higher

Table 3
Target/nontarget differences evidenced from ERP studies

Study	CPT type/ stimulus/ISI	Mean age (SD)	*N*	Groups	Results
Coons et al., 1981	Study 1 X/L/var/visual AX/L/var/visual	23.84 years (2.85)	13	control	Target/non target response was most pronounced at Pz. Lawful differences were found in the LPC amplitude between targets and nontargets. On the more difficult (AX) task, further differentiation of LPC amplitude was evident as a function of the salience of the nontarget (AN vs. NM).
	Study 2 X/L/var/visual AX/L/var/visual XX/L/var/visual	19.68 years (2.72)	23	control	Similar findings as with Study 1. Both AX and XX differed from nontargets.
Fallgatter, Brandeis, & Strik, 1997	AX/L/1650/visual	29.1 years (2.8)	10	control	ANOVA's revealed significant topographical differences between target and non target with peak more anterior for nontarget; nontarget P3 had longer latency than target P3
Fallgatter, Wiesbeck, Weijers, Boening, & Strik, 1998	AX/L/1650/visual	44.1 years (9.1)	20	alcohol dependent	For both groups, the nontargets were associated with greater anteriorization. Topographical differences found in previous study were supported in control group.
		40.8 years (11.1)	20	control	
Fitzpatrick et al., 1992	XX/N/1500/visual	8.7 years	19	ADD	P3 had expected parietal maximum and larger amplitude for targets than nontargets with mean latency of 474 ms; P3 amplitude to targets larger under MPH
	X/L/var/visual XX/L/var/visual AX/L/var/visual	19.68 years (2.72)	23	control	MPH effects found on LPC for XX such that amplitude increased for both target and nontarget
Friedman et al., 1978	X/N/1500/visual XX/N/1500/visual	13 years	6	at risk	Subjects were at risk for schizophrenia; XX-CPT had longer reaction times than X-CPT; LPC was segregated into stimulus-related and response-related components by quartiles — Quartile 1: positive deflection maximal at Pz; P340 larger at Cz, but P420 and 520 larger at Pz; P286 (frontocentral) component similar to P3 — larger to targets; P340 (parietal) component similar to P3 — larger to nontargets; P539 (parietal) component related temporally to reaction time

Friedman et al., 1981	X/N/1000/visual XX/N/1000/visual	14.4 years (1.8)	30	control	The average of ERPs was computed separately for target hits and nonresponses. Principal components (varimax rotation) was performed separately on target and nontarget ERP. VEP at Oz for N120, P150, N200 was similar for both tasks and target/nontarget anteriorly. Both N150 and slow negativity were larger in XX-CPT for nontargets. For P240, the target>nontarget were frontocentral; for P350, peak was parietal for both CPTs in nontarget with inflection in XX-CPT for target that was not evident in X-CPT. P450 was the most prominent with a parietal focus. P450 was larger to target than nontarget. P450 had lower amplitude to target on XX-CPT than X-CPT but was larger in XX-CPT nontarget than X-CPT nontarget. Prolonged positive activity is negative frontally and positive parietally for targets but for nontargets positivity reaches a positive maximum centrally.
Friedman, Boltri, Vaughn, & Erlemeyer-Kimling, 1985	X/N/1000/visual XX/N/1000/visual		74	controls	P450 was most prominent and larger to target than nontarget. X-CPT resulted in larger amplitude than XX-CPT for targets, but XX produced larger amplitudes for nontargets. XX-CPT produced larger slow wave activity than X-CPT; nontargets had larger slow wave than target.
Friedman, Cornblatt, Vaughan, & Erlenmeyer-Kimling, 1986	X/N/1000/visual XX/N/1000/visual	15.3 years 15.1 years 14.5 years	26 34 74	psychiatric high risk controls	Difference between target and nontarget similar to findings in previous study (Friedman, Boltri, et al., 1985). P240 was found to have frontrocentral topography, while P350, 450, and 550 all had parietal maxima.
Holcomb, Ackerman, & Dykman, 1985	XXX/L/2600/visual	109.1/129.4 months	21	ADD/Wo	Subjects were grouped by younger/older. Target was three symbols that had to be present simultaneously as opposed to in sequence.
		108.4/129.5 months	24	ADD/H	Results indicated a group effect for the amplitude of P3 but no age effect. There was a significant difference found for P3
		109.0/131.5 months	24	RD	amplitude to target vs. nontarget. At Pz, P3 amplitude differentiated ADDH from ADD/Wo, but did not differentiate
		114.3/134.4 months	24	control	RD from Control. P3 target vs. nontarget difference was lower only for both ADD groups. Latency of P3 to target vs. nontarget also differentiated groups with Control P3 significantly earlier than other groups. Main effect for P3 latency for age was found, but no interaction (Group × Age) effect.

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Table 3 (*continued*)

Study	CPT type/ stimulus/ISI	Mean age (SD)	*N*	Groups	Results
Kaskey et al., 1980	X/L/950/visual AX/L/950/visual	32.2–35.6 years	15	bipolar	Only parietal for LPC reported. Without lithium, for X-CPT there was no difference in P3 for target vs. nontarget on passive task (no response required). With lithium, the P3 differed from target to nontarget on passive task. On active task, P3 to target was significantly greater than nontarget and this difference increased with lithium. For AX-CPT no difference on P3 evident for AN nontarget, but difference was present for all other nontargets.
Klorman, Salzman, Pass, Borgstedt, & Dainer, 1979	X/L/800/visual	9.53 years (1.38)	18	ADDH	Smaller amplitude of LPC found in ADDH group (placebo condition). With MPH, there was an increased in the amplitude of LPC. Target–nontarget differences were significant.
		9.60 years (1.20)	17	control	
Klorman et al., 1983	X/L/1000–1500/ visual	8.78 years	14	ADDH — pervasive	P2 and P3 differed from X-CPT to XX-CPT. P3 larger at Pz than Cz ADDH-borderline ($P < .0001$) and the difference (target–nontarget) was greater at Pz than Cz. Same pattern was found for slow-wave (P740) amplitude. Only P3 and slow wave were affected by MPH.
	XX/L/1000–1500/ visual	9.00 years	14	ADDH-borderline	
Klorman et al., 1988	X/L/var/visual	8.53 years (1.65)	(15/1) 16	ADHD/ aggr.	MPH resulted in enlargement of P3 differences (target–nontarget) on XX-CPT. Maximum P3 at Pz.
		8.14 years (1.43)	(18/2) 20	ADHD/ no aggr.	
	XX/L/var/visual	8.72 years (1.52)	(16/1) 17	not ADHD	
		8.44 years (1.52)	(49/4) 53	combined groups	
Klorman et al., 1991	X/N/1500/visual	14.16 years (1.70)	46	ADD	Reaction time to nontargets was faster than targets. Ingestion of MPH increased the amplitude of the P3 component for nontargets and shortened P3 latency for targets and nontargets. P3 amplitude had parietal maximum and was greater for targets than nontargets, particularly at posterior sites and for older subjects. Amplitude of P3 decreased slightly in second half of task.

Michael et al., 1981	X/L/800/visual	9.31 years (1.92)	21	ADDH	AX-CPT more difficult; younger children were less accurate; performance deteriorated over the duration of the tasks, especially for X-CPT; ADDH had poorer performance; ADDH had smaller LPC amplitude with these differences more marked for younger children (5.9–8.2 years); amplitude of parietal LPC for both X and AX tasks enhanced by MPH; similar drug-related increases of LPC present at vertex, but less convincing MPH effects on performance and LPC for AX version; target evoked LPCs characterized by longer latencies than those associated with nontargets.
	AX/L/800/visual	9.35 years (1.94)	21	control	
Pass, Klorman, Salzman, Klein, & Kaskey, 1980	X/L/950/visual	28.58 years (7.64)	17	schizophrenic	Larger LPC (P3) evident to target than nontarget in both groups, but lower amplitude with schizophrenia.
		29.06 years (9.07)	16	controls	
Peloquin & Klorman, 1986	XX/L/1500/visual	8–14 years	18	normals	Nontargets resulted in larger amplitude in posterior lead, but this was significant only at Pz and Cz. Nontargets were found to evoke greater variability in latency of P3 ($P < .006$).
Roberts et al., 1994	AX/L/2000/visual	33.4 years	21	normals	P3 evoked to NX sequence larger at central and frontal and contralateral to prepared movement, while P3 to AX was symmetric with parietal maximum. Difference in P3 (AX-AN-NM) significant ($P < .0001$). P3 larger frontally on NX. Greatest difference (target–nontarget) in P3 found at frontal and central sites. For P3 latency, NX-AX-NM differences significant ($P < .0001$).
Schupp et al., 1994	AX/ L/nr/visual	19–37 years	37	normals	P3 to AN nontarget was central, while AX had parietal maximum. Latency varied by AX, AN (longest), and NM (shortest). Latencies differed across anterior posterior gradient with shortest in frontal area.
Simson et al., 1977	XX/L/2000/auditory XX/L/2000/visual	21–45 years	8	control	For auditory task, P2 showed a small nonsignificant enhancement to X2. The P3 for X1 showed a mid-parietal extension into parietotem-poral areas. When XX, then there was a mid-parietal extension. If not target (XN), then P3 was later in posterior area and more central. For visual task, P3 peak was greater for target than nontarget (XN) and had a longer peak latency. LPC culminated anteriorly at 400 ms, and posteriorly at 460 ms. P3 was maximal at Pz (mid-parietal); for X1 and target (XX), extending parietotemporally for nontarget (XN).

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Table 3 (*continued*)

Study	CPT type/ stimulus/ISI	Mean age (SD)	*N*	Groups	Results
Stamm et al., 1982	AX/O/2000/visual	9–10 years 9–10 years adults	31 25 150	inattentive attentive volunteers	When P3 was compared across ages, a distinct positive negative deflection at 300 ms after stimulus onset was noted (P2) in children, that did not show up in adults. P3 associated with targets and nontargets differed. P3 to nontargets was most evident at Pz and more dominant in children than adults. P3 to targets also most evident at Pz, but amplitudes were similar for adults and children.
Strandburg et al., 1990	XX/N/1250/ visual	11.2 years (1.5) 11.2 years (1.3)	13 19	schizophrenic control	Amplitude and latency of CNV did not differ by group. Rightward asymmetry found for P1, N1, P2, and this was greater in normals. P3 amplitude was maximal at Pz and greater for normals to target. P3 amplitude for target greater than nontarget at Pz. Rightward asymmetry also noted for posterior temporal area in normals.
Strandburg et al., 1994	X/N/1250/visual XX/N/1250/visual	12.50 years (2.67) 12.75 years (1.67)	16 16	schizophrenic controls	Schizophrenic group did not evidence target–nontarget difference and had absent P1/N1 amplitudes. P3 was maximal at Pz with rightward laterality for P1, N1 amplitude in normals only. Schizophrenic group had N peak at Pz that was not evident in normals for no response and nontarget. For normals only, there was a condition-related effect (P3 for XX>X for nontarget) as well as cross-over effect (P3 for XX<X for target). Although demonstrating a similar pattern differences were not significant for the schizophrenic group.
Verbaten et al., 1994	X/L/1600/visual	11.2 years (2.1)	12	ADHD	Increases in P3 amplitude to nontargets with MPH concurred with increase in hits with MPH but in a nonlinear manner. Target–nontarget differences were noted for P3.
Wagner et al., 1989	X/L/var/visual	25 years (5) 29 years (5)	14 14	schizophrenics control	Clear difference for both amplitude and latency for target vs. nontargets for normals.

Stimulus: L = letters, N = numbers, C = color, F = form/shape, O = object; var = adaptive rate; var[1] = two rates: fast = 800 ms, slow = 1800 ms; nr = not reported; ADD, ADHD = attention deficit hyperactivity disorder, ADD/Wo = Attention deficit disorder without hyperactivity; ADDH = Attention deficit disorder with hyperactivity; MPH = methylphenidate; LPC = late positive component; CNV = contingent negative variation.

metabolic rates in anterior frontal, mesial frontal, and posterior frontal areas during CPT (e.g., Buchsbaum et al., 1990; Cohen, Semple, Gross, Holcomb, et al., 1988; Cohen, Semple, Gross, Nordahl, et al., 1988; Hazlett et al., 1993; Mansour et al., 1996; Rezai et al., 1993; Zemishlany et al., 1996). Similarly, for normals, an increased anterior–posterior gradient was found to be associated with better performance; this was not evident in clinical groups (Buchsbaum, Haier, et al., 1992; Buchsbaum & Hazlett, 1989; Schröeder et al., 1994). For visual CPTs, there was a significant and quite substantial correlation between Area 18 (occipital lobe) metabolic rate and CPT performance ($r = .70$, Buchsbaum, Potkin et al., 1992). CPT performance also was correlated with the angular gyrus bilaterally and Area 17 (occipital lobe) of the left hemisphere (Buchsbaum, Potkin, et al., 1992). Unlike the findings of Buchsbaum, Potkin, et al. (1992), Keilp et al. (1997) found no significant correlations between task performance and regional perfusion. Increased metabolic rates in the limbic system, basal ganglia, and thalamic areas have been found to be associated with CPT performance (Wu et al., 1991, 1992). Wu et al. (1991) found that poor performance on the CPT was associated with decrements in metabolic rate bilaterally for the amygdala ($r = .90$), thalamus ($r = .93$), caudate ($r = .83$), and the putamen ($r = .90$). These findings are consistent with theoretical models suggesting involvement of the basal ganglia, as well as the limbic system in attentional processes. The magnitudes of these relationships is considerable and approached the theoretical limit of the correlation given the reliability of measurements of the independent and dependent variables.

Using MRI, Sax et al. (1999) investigated the association between volumetric measurements of specific brain structures and CPT performance. They found prefrontal volume and hippocampal volume to be correlated significantly with the sensitivity index from Signal Detection Theory ($r = .59$ and .69, respectively). Häger et al. (1998) used fMRI during XX-CPT performance with 12 adult volunteers (mean age of 27.9 ± 6.4 years) and found rightward asymmetry of brain activation. With regard to specific structures involved, greater right hemisphere activation was evident in the anterior cingulum, dorsolateral prefrontal cortex, superior temporal gyrus, caudate nucleus, and thalamic nuclei. In reviewing their results, Häger et al. considered the emergence of the caudate nucleus activation as the most significant finding.

Asymmetry of attention has been studied using NIRS. To examine the correlates of CPT performance, Fallgatter and Strik's (1997) study found that activation of the frontal areas occurred during CPT performance. While frontal activation was evident bilaterally, the increase in activation relative to baseline was only significant for the right frontal area. Using PET, Buchsbaum and colleagues (Buchsbaum & Hazlett, 1989; Buchsbaum, Siegel, et al., 1992) also found asymmetry of the frontal cortex (R > L). Siegel, Nuechterlein, Abel, Wu, and Buchsbaum (1995) found that sensitivity correlated with metabolic rate in the medial superior frontal gyrus and lateral inferior temporal gyrus. For the control group, d' correlated significantly with the right lateral frontal cortex ($r = .46$), the right lateral inferior gyrus ($r = .45$), and the right medial superior frontal gyrus ($r = .50$). In contrast, two other studies (Hazlett et al., 1993; Keilp et al., 1997) found asymmetry differences depending on the nature of the stimuli with numbers resulting in more leftward asymmetry as compared to activation in response to shapes. Using SPECT, there was an increase in anterior areas including the left cingulate, left frontal white matter, left basal ganglia, left thalamus, and

Table 4
ES for amplitude of target/nontarget differences evidenced at Pz from ERP studies

Study		X vs. not X	AX vs. not AX	XX vs. not XX	AX vs. NX	AX vs. AN	AX vs. NM
Coons et al., 1981	Study 1	1.52	–	–	1.31	0.09	2.15
	Study 2	1.88	–	1.11	1.87	0.36	2.08
Coons et al., 1987		–	–	1.29	–	–	–
Fallgatter et al., 1997		–	0.81[c]	–	–	–	–
Fallgatter et al., 1998	ADD	–	0.59[c]	–	–	–	–
	control	–	0.32[c]	–	–	–	–
Fitzpatrick et al., 1992		–	–	1.50	–	–	–
Friedman et al., 1978		CNC	–	CNC	–	–	–
Friedman et al., 1981		0.37	–	0.37	–	–	
Friedman, Erlenmeyer-Kimling, & Vaughan, 1985		CNC	–	CNC	–	–	–
Friedman et al., 1986		CNC	–	CNC	–	–	–
Holcomb et al., 1985		2.47	–	CNC	–	–	–
Kaskey et al., 1980		1.56	0.01	–	0.04	0.01	0.82
Klorman et al., 1979		0.62	–	–	–	–	–
Klorman et al., 1983		2.59[a]	–	–	–	–	–
Klorman et al., 1988		1.75	–	1.75	–	–	–
Klorman et al., 1990		2.45	–	–	–	–	–
Michael et al., 1981[b]	5–8 years	0.94	–	–	0.58	0.33	0.49
	8–10 years	1.18	–	–	0.23	0.24	1.53
	10–13 years	1.67	–	–	0.86	0.71	1.61
Pass et al., 1980		0.89	–	–	–	–	–
Peloquin & Klorman, 1986	–	–	1.76	–	–	–	–
Roberts et al., 1994	–	0.58	–	–	–	–	–
Schupp et al., 1994	–	0.18	–	–	CNC	CNC	CNC
Simson et al., 1977	auditory	–	–	0.32	–	–	–
	visual	–	–	0.34	–	–	–
Stamm et al., 1982		–	0.85	–	CNC	CNC	CNC
Strandburg et al., 1990	–	–	–	CNC	–	–	
Strandburg et al., 1994		2.19[a]	–	–	–	–	–
Verbaten et al., 1994		2.48	–	–	–	–	–
Wagner et al., 1989	control	1.04	–	–	–	–	–
	schizophrenic	1.02	–	–	–	–	–
Number of studies		15	7	8	6	6	6
Mean ES		1.46	0.48	1.09	0.82	0.29	1.45
S.D.		0.67	0.31	0.65	0.69	0.25	0.67

ERP = Event Related Potential; X vs. Not X = target stimulus vs. nontarget stimulus for X type (respond if "X") CPT; AX vs. Not AX = target stimulus vs. nontarget stimulus for AX type (respond if "AX") CPT; XX vs. Not XX = target stimulus vs. nontarget stimulus for XX type (respond if "XX") CPT; NX = sequence of stimuli was a nontarget stimulus followed by X; XN = sequence of stimuli was X followed by an nontarget stimulus; NM = sequence of stimuli was a nontarget stimulus followed by a nontarget stimulus; CNC = could not compute the ES due to insufficient data; ADD = Attention Deficit Disorder.

[a] Combined CPT forms for target vs. nontarget; lead not specified.

[b] Average of ES over Phases 1 and 2.

[c] Averaged across leads.

the occipital lobes bilaterally (e.g., Herrera et al., 1991; Keilp et al., 1997). Mansour et al. (1996) found no hemispheric asymmetry, but suggested that there may be gender differences on frontal activation. Preliminary evidence from Benedict et al. (1998) suggested that the modality of the stimulus presentation (auditory vs. visual) may impact on brain activation patterns. Thus, some PET studies during CPT performance support the premise that attention is asymmetrical (R > L) (e.g., Buchsbaum & Hazlett, 1989; Buchsbaum, Siegel, et al., 1992) while other studies (e.g., Hazlett et al., 1993; Keilp et al., 1997; Mansour et al., 1996) suggest that the extent and direction of asymmetry evidenced during CPT performance may differ as a function of task parameters of the CPT (i.e., the linguistic or spatial nature of the target; modality of presentation) as well as gender. These findings suggest that additional research is needed with regard to asymmetry of attentional function and CPT performance.

8. Discussion

Attention as a construct is extremely complex and theories that map this construct onto underlying neurological substrates are equally as complex. Multiple models have been proposed to address the myriad aspects of attention (e.g., Goldman-Rakic, 1988; Heilman et al., 1985; Posner, 1988). Contemporary models of attention portray multiple, interactive functional systems that involve cortical (frontal, prefrontal, parietal) and subcortical (limbic system, basal ganglia) structures, as well as descending and ascending pathways between the basal ganglia, the frontal lobes, and the thalamus. Increasingly, the basal ganglia emerge as a central component to the functional system underlying attention (Goldman-Rakic, 1988; Luria, 1973; Mirsky, 1989; Pribram & McGuinness, 1975; van Zomeren & Brouwer, 1994; Voeller, 1991). In conjunction with these models, anterior–posterior gradients as well as asymmetrical models of hemispheric involvement to the attentional system have been suggested. With advanced technology and continued research, our understanding of the neural substrates of attention as well as inhibition will continue to be enhanced. The complexity of the system, as well as the different structures and pathways involved in maintaining the functional system, suggest that damage to any component(s) of the attentional system could differentially impact on behavior. As such, it is of interest to determine the extent to which measures of attention provide information regarding the integrity of this functional system.

Do CPTs provide a window on the neural substrates of attention? Based on the studies reviewed, areas of activation during CPT performance as well as identified lesions would suggest significant parallels with current models of attention. Certainly, regardless of the version of the CPT used, results clearly substantiate Rosvold et al.'s (1956) contention that CPTs are sensitive to brain damage or dysfunction. Studies reviewed suggest a direct relationship between impairment on the CPT and extent to which the damage/dysfunction is diffuse regardless of the etiology of that damage. Localized damage at a single point in the attentional system appears to be likely to result in less impaired performance than diffuse damage. This suggests that the level of sensitivity of the CPT may in fact compromise its specificity or ability to localize dysfunction. Due to the sensitivity to various types and locations of brain dysfunction within the attention and inhibition systems, impaired perfor-

mance may best be interpreted as evidence of dysfunction as opposed to being indicative of a specific etiology or disorder.

Various components of the CPT tasks have been found to be associated with neural substrates that are associated with attention, and at some level, with inhibition. Variations in CPT types (X-CPT vs. AX-CPT) may impact on the level of sensitivity. Similarly, the nature of the target (linguistic vs. nonlinguistic) may affect the sensitivity to damage/dysfunction in related areas of the brain as well as being associated with differential impacts on findings of attentional asymmetry. Variations in ISI may impact on sensitivity as well; shorter ISIs are more likely to result in performance suggestive of dysfunction. ERP studies further suggest that the nature of the nontargets (similar to target or dissimilar) may also impact on results. The error patterns of the individual as opposed to the total error score may provide additional information. The differences in ERP components to similar or irrelevant nontargets support Halperin, Sharma, et al. (1991) and Halperin, Wolf, et al.'s (1991) contentions regarding the need to consider the types of errors made in the interpretation of CPT performance.

Existing research clearly supports CPT sensitivity to brain damage or dysfunction, and there is an abundance of research related to brain behavior relationships and CPT performance. However, additional study is needed in order to interpret CPT results accurately from a neuropsychological perspective beyond the identification of some level of impairment. The abundance of variations in task parameters (stimuli type, stimuli quality, ISI) as well as CPT demands (X-CPT, AX-CPT, not X-CPT) make generalization of performance data difficult at best particularly with regard to attentional asymmetry. There is a need for consistent, comparative study to be done that investigates the differences in sensitivity to various areas of brain function using multiple combinations of CPTs on common populations before any type of generalizations relating to specific brain structures or hemispheres can be made with certainty.

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