Information-Processing Deficits Across Childhood- and Adult-Onset Schizophrenia

by Robert J. Strandburg, James T. Marsh, Warren S. Brown, Robert F. Asarnow, and Donald Guthrie

Abstract

Event-related potentials were recorded for childhood- and adult-onset schizophrenia subjects performing the span of apprehension (Span) task, which is sensitive to vulnerability factors in schizophrenia. Subjects responded to the onset of the Span arrays in a reaction time condition and then responded differentially to the presence of one of two target letters in the Span condition.

While neither the childhood- nor the adult-onset group exhibited abnormalities in preparatory contingent negative variation activity, both groups produced significantly less endogenous negative activity between 100 and 300 ms after Span stimulus onset than age-matched normals.

This endogenous negative activity reflects attentional effort associated with serial search and stimulus identification. These results support the position that schizophrenia subjects are impaired in their ability to allocate adequate attentional resources for processing Span stimuli. Moreover, the similarity of this information-processing deficit in the two groups suggests that childhood- and adult-onset schizophrenia lie on a continuum in this regard.


While it has long been recognized that attentional deficits play an important role in schizophrenia, the precise nature of this cognitive impairment continues to be the focus of considerable debate. We have attempted to characterize this deficit by comparing brain activity in normal and schizophrenia subjects engaged in information-processing tasks sensitive to this disorder. In this brief review, we focus on an early visual information-processing deficit observed in event-related potential (ERP) data recorded from children with schizophrenia during performance of the Span of apprehension (Span; Neale 1971) task.

This report also addresses the relationship of childhood- to adult-onset schizophrenia, presenting preliminary findings from a Span/ERP study of adult-onset schizophrenia subjects and comparing these results with the data from children.

We have focused on children with schizophrenia for several reasons. First, there is a twofold increase in the aggregation of schizophrenic disorders in first-degree relatives of prepubertal- as compared with adult-onset patients (Kallman and Roth 1956; Kolvin 1971). Thus, it is likely that these children are a more homogeneous group with an increased genetic vulnerability. Moreover, the children in our study were typically seen shortly after their first break, had not experienced extended institutionalization, and had brief exposure (if any) to neuroleptic treatment. For these reasons, we believe they provide a particularly good opportunity to examine vulnerability factors in schizophrenia.

The Span

The partial-report version of the Span task was chosen for this re-
search because several studies have shown that it is selectively sensitive to schizophrenia (Neale 1971; Asarnow and MacCrimmon 1981; Harris et al. 1985; and Strauss et al. 1984). Moreover, Span performance appears to tap a trait characteristic of this disorder. Performance deficits have been observed in stabilized schizophrenia outpatients (Nuechterlein et al. 1986), “recovered” schizophrenia patients (Asarnow and MacCrimmon 1978, 1981), foster children whose biological mothers had schizophrenia (Asarnow et al. 1977), mothers of schizophrenia patients (Wagener et al. 1986), and subjects who have no history of psychiatric disorder but have elevated scores on the schizophrenia scale of the Minnesota Multiphasic Personality Inventory (Golden and Meehl 1979) (Asarnow et al. 1983). Thus, it appears that impaired performance on the Span task reflects an information-processing deficit associated with a genetically transmitted vulnerability to schizophrenia.

In the partial-report version of the Span task used in our research, subjects were asked to identify which of two letters, T or F, was present in 5 × 5 arrays presented for 50 ms, each containing 12 randomly placed letters. This task provides a measure of the efficiency of visual information processing. On the basis of behavioral evidence, we have argued that the observed deficits in Span performance in schizophrenia result from deficiencies in resource allocation to controlled attentional processes, most likely those associated with serial search of the visual icon (Sherman and Asarnow 1985; Asarnow et al. 1989, 1991; Asarnow and Granholm 1990). In this article we summarize electrophysiologic data that bear on this interpretation and address the issue of whether childhood-onset schizophrenia lies on a continuum with adult-onset schizophrenia in regard to this information-processing deficit.

**Subjects.** Although there has been considerable controversy over the diagnosis of schizophrenia in children, the 13 children with schizophrenia discussed here meet DSM-III-R (American Psychiatric Association 1987) criteria for adult schizophrenia and satisfy a variety of exclusionary criteria designed to exclude persons with organic brain damage or other developmental disorders. All 13 children experienced hallucinations, and 9 had thought disorder, delusions, or both. (For a full discussion of this group see Russell et al. 1989 and Strandburg et al. 1991.) Six of the subjects were inpatients at the time of testing; the remaining seven were former inpatients at the same institution. These children with schizophrenia (7 males, 6 females) were compared with a group of 17 normal children (9 male, 8 female) matched for mean age (11.3 years) and handedness (1 left-handed child per group).

To address the relationship of childhood-onset to adult-onset schizophrenia, we will also discuss ERP data from 18 adult-onset schizophrenia patients and 18 normal controls matched for age (26.2 years), sex (1 female in each group), and education level (13 years of schooling). The mean duration of illness (since first psychotic episode) for the adults with schizophrenia was 34.0 ± 31.4 months, and all were outpatients (11 were living at home, 2 were in board-and-care facilities, and 5 were fully or semi-independent) stabilized on maintenance doses of prolixin decanoate. In contrast, only 6 of the 13 childhood-onset schizophrenia subjects were on medication. At time of testing, the adult schizophrenia patients exhibited few positive symptoms as measured by the Brief Psychiatric Rating Scale (BPRS; Overall and Gorham 1962; Guy 1976). This group is discussed in detail in Strandburg et al. (1994a).

**ERP Recording and Analysis.** The course of information processing in the Span task is particularly well suited for ERP analysis: A discrete stimulus is presented repeatedly, and the relevant processing is completed within a brief period (average response time is approximately 1 s). In both the child and the adult studies, a 50-ms warning tone was included 500 ms before the onset of each visual Span array to permit an examination of central nervous system preparatory activity in addition to Span processing. Two seconds of electroencephalographic (EEG) data (beginning 500 ms before the warning tone) were digitized and stored on line. EEG was recorded at the 19 standard International 10–20 System loci referenced to linked ears. The single-trial data were edited for muscle and eye-movement artifacts and sorted by response accuracy before averaging. (See Strandburg et al. 1991 for a detailed description of the electrophysiologic methods.) The results presented here focus primarily on data from correct-response trials, since these represent the best sample of trials in which the subjects were actively engaged in Span processing.

The ERP literature makes an important distinction between exogenous and endogenous compo-
Exogenous components are influenced by physical characteristics of the stimulus (brightness, duration, etc.), while endogenous components reflect task-related processing (Donchin et al. 1978). Because we are concerned with the attentional demands of the Span task, our interest is in endogenous processing. Two regions of the ERP were examined for endogenous activity: the contingent negative variation (CNV) and the processing negativity. While a third endogenous component, the P300, has been studied extensively in schizophrenia patients, we have not analyzed it in these studies for two reasons. First, P300 is small in Span ERPs because every trial contains a target. Second, and more important, P300 amplitude decreases when the subject is uncertain of correct target detection. In a forced-choice task, ERPs for correct response trials contain some trials in which the subject was uncertain but guessed correctly. This would more likely be the case in schizophrenia subjects than in normals, as indicated by the lower correct response rates of schizophrenia patients.

**CNV.** To assess mobilization of general attention or readiness, CNV was measured using the mean amplitude at midline leads over the last 200 ms of the 500-ms interval between the auditory warning tone and the onset of the visual Span stimuli. Before measurement, the ERPs were low-pass filtered to more accurately evaluate this slow component. CNV on correct-response trials was compared first with the CNV generated in the reaction time (RT) condition and then to the CNV generated during error trials. These measurements were made at Fz, Cz, and Pz in the children, while in adults the measurements were made at Fz only (the field maximum in both age groups). Comparisons were also made between homologous frontal leads over the two hemispheres in both groups to assess laterality. On the basis of our earlier work (Strandburg et al. 1984), we anticipated that CNV amplitude would be smaller in schizophrenia subjects than in normal subjects and would exhibit abnormal laterality at frontal leads.

**Processing Negativity.** There is a considerable body of research on the early attention-related processing negativities (Naatanen 1982; Hillyard and Kutas 1983; Harter and Aine 1984; Ritter et al. 1984; Hillyard and Hansen 1986; Naatanen and Picton 1987). This endogenous activity overlaps the exogenous P1-N1-P2 complex and is typically isolated by subtraction procedures in which ERPs to unattended stimuli (ignored or passively viewed) are subtracted from ERPs obtained when the same stimuli are actively processed. This research falls into two broad categories: One focuses on channel-specific selective attention and is dominated by a single behavioral paradigm; the other addresses activity associated with stimulus identification and categorization and embraces a broad range of visual and auditory information-processing tasks.

The research reported here reflects the latter approach, using subtraction procedures similar to those developed by Ritter and his colleagues. Ritter et al. (1988) recorded ERPs while subjects responded differentially to the presence (20% of the trials) or absence of a vowel in a line of five letters. This was preceded by an RT condition in which the subject merely responded to the onset of the visual stimuli. Subtracting ERPs obtained in the RT condition from those obtained to nontarget stimuli in the consonant/vowel (C/V) discrimination task produced a difference potential that included a negative-going wave that peaked at approximately 300 ms. Ritter et al. referred to this processing negativity as the "NA" component. In a series of studies Ritter and colleagues have demonstrated that NA represents a family of negative-going endogenous potentials associated with stimulus identification.

In a similar fashion, we examined ERP activity in the period immediately after the onset of the Span stimuli to assess endogenous activity required for successful Span performance. To isolate this task-related activity, ERPs were first recorded when subjects responded with a button press to the onset of the Span stimuli before receiving the Span instructions. The ERPs obtained in this RT condition were then subtracted from ERPs elicited when the subjects were later engaged in actual Span processing. This Span-minus-RT difference potential produced a negative wave that peaked 280 ms after Span array onset. This process is illustrated in figure 1.

For the top and middle rows, ERPs from the RT and Span conditions are overlapped for adults and children with schizophrenia and normal adults and children. In all traces, three exogenous components can be discerned following Span array onset at 500 ms (P1 at +130 ms, N1 at +190 ms, and P2 at +320 ms; note also the auditory P1-N1-P2 complex following onset
The upper two rows are cross-subject grand mean event-related potentials (ERPs) in the Span (solid lines) and reaction time (RT; dashed lines) tasks. Left traces are obtained from the O1 electrode in children, and the right traces are from the Pz electrode in adults; normal subjects are shown in the top row, and schizophrenia subjects, in the middle row. Traces in the bottom row are the difference potentials created by subtracting the RT ERPs from the Span ERPs shown above. Solid traces are difference potentials for normal subjects; dashed traces are for schizophrenia subjects. The first vertical dashed line (0 ms) marks the onset of the warning tone; the second line (500 ms) marks the onset of the Span array. Note differences in amplitude scaling between children and adults.

The bottom traces show the residual endogenous activity resulting from the Span minus RT subtraction, which cancels the shared exogenous P1-N1-P2 activity for both the auditory and visual stimuli. This process permits more accurate quantification of the negative-going activity generated when the subjects locate, identify, and categorize the target stimulus. Although the negativity elicited by the Span task appears in most respects to be a processing negativity, because of differences in the task between our experiment and that of other investigators, we refer to this negativity as Span endogenous negativity. Peak amplitude and latency as well as onset and offset time were measured at posterior leads and were statistically evaluated at the field maximum in both groups (O1 and O2 in children, Pz in adults; see figure 2).

We believe that the Span endogenous negativity obtained in our studies reflects processing comparable to the NA in the Ritter et al. (1988) studies. We expected the attentional impairment in schizophrenia to result in diminished Span endogenous negativity in our schizophrenia subjects.

Results

Behavior. Consistent with previous work, the normal children in our study performed significantly better than the children with schizophrenia on the Span task (total correct: normal subjects = 82%, schizophrenia subjects = 70%, p < 0.001). Medication status did not affect performance levels in the schizophrenia subjects (on medications = 69%, off medications = 70%). Similar results were obtained...
Figure 2. Scalp topography of the span of apprehension (Span) endogenous negativity

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Topographic field maps for the peak latency of the Span endogenous negativity difference potential for children and adults; normal subjects are shown in upper maps, and schizophrenia subjects in lower maps. Greater negativity is indicated by darker shades, with the electrode at the field maximum indicated by the large black circle. Each map is uniquely scaled to emphasize the scalp distribution of the endogenous negative component; that is, the number of gray-scale steps between 0 and the field maximum is the same. Amplitude differences for each gray-scale step are as follows: normal children = 1.00 \mu V; normal adults = 0.67 \mu V; children with schizophrenia = 0.40 \mu V; adults with schizophrenia = 0.40 \mu V. Maps are from the following latencies: normal children, 234 ms; normal adults, 269 ms; children with schizophrenia, 246 ms; adults with schizophrenia, 234 ms.

In the adult groups, with normals again achieving significantly higher hit rates than schizophrenia subjects (total correct: normal subjects = 85%, schizophrenia subjects = 73%, p = 0.003).

CNV. In figure 3, CNV data at F7, Fz, and F8 for both groups are presented. Contrary to our predictions, CNV was neither reduced nor abnormally lateralized in the schizophrenia subjects. In children, CNV amplitude did not differ between groups or conditions (RT vs. Span). The only significant CNV finding in our younger subjects was a group-by-response accuracy interaction (F = 5.48; df = 1.28; p = 0.027) in which a reduction in CNV amplitude was observed on error trials only in children with schizophrenia.

As with the children, we found no reduced CNV in subjects with adult-onset schizophrenia. In fact, within the last 100 ms before Span array onset (where CNV reaches peak amplitude in both groups), there was a significant group effect (F = 6.51; df = 1.35; p = 0.016) in which schizophrenia subjects produced greater CNV amplitudes than the controls (−8.3 vs. −4.1 \mu V). In addition, both groups produced larger CNVs in the Span when compared with the RT condition (F = 9.34; df = 1.35; p < 0.005). The group \times condition interaction was not significant. No laterality effects were seen in either the adult or the child group.

Span Endogenous Negativity. At the bottom of figure 1 are overlap plots of the Span minus RT difference potentials at Pz and O1 for normal versus schizophrenia subjects. While both normal adults and adults with schizophrenia differ from children in the locus of maximum Span endogenous negative activity (O1 for children and Pz for adults, presumably a function of maturation), it is apparent that the time course of this posterior negative-going activity is similar in all four groups. Moreover, inspection of topographic field maps (figure 2) reveals that, within age groups, normal and schizophrenia subjects do not differ in the location of the field maximum of the Span endogenous negativity. As predicted, schizophrenia subjects produce significantly less Span endogenous negativity than normal subjects at the field maximum in both the child (−11.1 vs. −4.8 \mu V; F = 5.85; df = 1.28; p = 0.022) and the adult (−8.1 vs. −5.5 \mu V; F = 5.19; df = 1.35; p = 0.029) groups. Latency comparisons within age groups did not yield any significant effects, and in both age groups the Span endogenous negativity obtained on error trials did not differ in amplitude or latency from that obtained on correct-response trials. Children
with schizophrenia who were not on medication did not differ significantly in the amplitude of Span endogenous negativity from those on medications. (These medication subgroups differed in inpatient status but not in age or age at onset.)

Discussion
These results support the position that schizophrenia patients exhibit a deficit in early attentional processing and that this deficit is comparable in childhood- and adult-onset schizophrenia subjects. In the first 250 ms of stimulus processing, both schizophrenia groups generate significantly less attention-related negative activity (Span endogenous negativity) in response to the processing demands of the Span task. Significantly, we did not observe a deficit in the preceding CNV component.

Although a number of authors have observed abnormalities in attention-related negativities in adults with schizophrenia (Baribeau-Braun et al. 1983; Saitoh et al. 1984; Barrett et al. 1986; Michie et al. 1990; see also reviews by Pritchard 1986 and Holzman 1987), these studies all involve channel-specific auditory selective attention. Identification of the specific computational processes reflected in the Span endogenous negativity requires examination of the research on processing negativities in more complicated discrimination tasks. On the basis of time course, topography, and the processing demands of the eliciting behavioral paradigm, the Span endogenous negativity could include activity associated with both the NA and N2 components described by Ritter and colleagues (Ritter et al. 1982, 1983, 1988; Simson et al. 1985; Lovrich et al. 1986). According to these authors, the endogenous negativities in their tasks reflect attentional effort allocated to stimulus identification (NA) and categorization (N2). Ritter et al. isolate the NA component by subtracting ERPs obtained in an RT condition from those generated by nontarget stimuli in a subsequent visual discrimination task. They obtain the N2 component by subtracting nontarget from target trial ERPs. Because these two negative potentials have posterior topographies and in some cases overlap in peak latency (see Lovrich et al. 1986), they can

Figure 3. Contingent negative variation (CNV) at frontal leads

Event-related potential data for normal (solid lines) and individuals with schizophrenia (dashed lines), children (C) at the top, and adults (A) below. Vertical marker indicates +5.0 μV, and the horizontal scale indicates 1,000 ms in 100 ms divisions. CNV is measured over the 200 ms preceding the second marker (span of apprehension stimulus onset).
be distinguished reliably only on the basis of these specific subtraction procedures (nontarget minus RT vs. target minus nontarget). These individual subtractions cannot be carried out in our data, since every trial in the Span task is a target. Thus, it is not possible to determine precisely which of these two components (NA or N2) is affected in the children with schizophrenia.

The problem of interpreting the cognitive processes reflected in the Span endogenous negativity can be approached by comparing this negative wave with difference potentials generated in tasks that have processing demands similar to the Span, but in which the NA and N2 components can be separated more easily.

The Span task involves serial search of an icon containing 12 randomly placed letters for 1 of 2 target letters. Because stimulus duration is only 50 ms, there is insufficient time to scan the visual display itself. Thus, task performance is constrained by the capacity and duration of the mental representation of the stimulus as well as by the efficiency of the search process. In an extensive review of the literature on the Span, Asarnow et al. (1991; see also Asarnow and Granholm 1990) conclude that the poorer performance of schizophrenia subjects on the Span task is most likely due to an impairment in some aspect of serial scanning.

The Ritter et al. (1988) study cited earlier in this article also used a discrimination task that required serial search: the scanning of a line of five consonants for the occasional appearance of a single vowel. As in the Span task, the C/V task involves the comparison of several briefly presented, spatially distributed letters (5 in the C/V task; 12 in the Span task) with a target memory set that includes multiple items (5 vowels in the C/V task; T or F in the Span task).

The difference potentials obtained in the Span and the NA obtained in the C/V task were quite similar. Both included two negative waves: an earlier subcomponent that was greatest at O1 and O2 for children and at Pz for adults and a later, more centrally distributed peak. Thus, because of similarities in the specific processing demands of the tasks that elicit the Span endogenous negativity and NA, as well as the waveform shape and topography of these two components, we believe that the computational processes responsible for NA and Span endogenous negativity are similar.

Despite the greater difficulty of the Span task, Span endogenous negativity peaks earlier than the C/V-elicited NA, perhaps because of the greater constraints on the search process in Span stimulus identification. Because of the size, complexity, and brief duration (50 ms) of the Span arrays, there is insufficient time for recoding into short-term memory. Thus, the search process must be carried out on the icon, and it seems likely that this quickly fading mental representation defines a temporal limit for the search, after which the subject must simply make a guess about the identity of the target. With fewer letters and a longer stimulus duration (100 ms), the entire stimulus array in the C/V task may be successfully transmitted to working memory.

The hypothesis that Span endogenous negativity represents serial search activity terminated by the disappearance of the icon could also account for the observation that peak latencies for correct-response and error trials do not differ. While it might be expected that peak latency would be greater on error trials because a failure to detect the target would force the search to proceed through the entire 12-letter array, disappearance of the icon would cut this search short, reducing peak latency differences between hits and misses.

The fact that the amplitude of Span endogenous negativity did not vary between correct-response and error trials (Strandburg et al. 1991, 1994a) rules out the possibility that the group difference in amplitude for data from correct-response trials is an indirect result of the larger proportion of identification failures, which leads to correct guesses in the averages for the poorer-performing schizophrenia subjects. Constancy in the amplitude of the Span endogenous negativity across error and correct-response trials also suggests that detection failures were not simply a result of attention lapses. Rather, it appears that our subjects are consistently allocating processing resources on each trial and that errors result from demands that exceed the resources available for target identification. Overall, it appears that the schizophrenia subjects are either less able (having limited or less efficient resources) or less cognitively inclined (inappropriate in their choice of processing strategy) to allocate normal levels of attentional resources to this task.

Finally, it should be noted that while less Span endogenous negativity is observed in schizophrenia subjects in both age groups, this difference between normal and schizophrenia subjects occurs in somewhat different ways in adult
and child subjects. The amplitude of the Span endogenous negativity is calculated by subtracting ERPs obtained in RT and Span conditions. In the adult ERP data, there was little difference in N1 and P2 amplitudes during the RT condition in the two groups. Subsequently, in the Span condition, large group differences were observed in N1 and P2 (schizophrenia subjects less negative) because more endogenous negativity overlaps these components in the normal subjects. In the children, however, group differences in amplitude in this region of the ERP were larger in the RT condition, with the children who had schizophrenia more negative. (Compare the dashed traces in the top and middle panels for children in figure 1; also see Strandburg et al. 1991.) Since there is no evidence in the literature to suggest that children who have schizophrenia produce larger N1s and smaller P2s (which, if true, would account for the greater negativity over the Span endogenous negativity epoch during RT processing), it appears that the childhood-onset schizophrenia subjects are exhibiting inappropriate endogenous processing in the RT condition. Nevertheless, negativity in the N1 region is still greater in normal than in children who have schizophrenia when comparing unsubtracted waveforms. Thus, it appears that while adult- and childhood-onset schizophrenia subjects share a deficit in resource allocation for discriminative processing, they differ in that children with schizophrenia also inappropriately allocate this resource in situations where it is not required (the RT task).

Examination of the endogenous processing that preceded Span stimulus onset (CNV) adds further support to the position that impaired Span processing in schizophrenia subjects results from a deficit in the allocation of resources to specific computational processes rather than in the prior mobilization of general attention. CNV amplitude is a measure of readiness for stimulus processing or preparedness to respond (Snyder and Lindsay 1977; Donchin et al. 1978; Naatanen and Michie 1979). While there are several studies, including our own 1984 article, in which schizophrenia subjects have been shown to produce less CNV than normals (see Pritchard 1986), we did not observe a diminished CNV in either the adult- or childhood-onset schizophrenia subjects reported here.

Although we previously reported (Strandburg et al. 1984) that children with schizophrenia exhibited abnormal lateralization of CNV activity at frontal leads (F7 and F8), this finding has not been replicated for either age group in current studies (Strandburg et al. 1991, 1994a). This difference could be the result of differences in the subjects. The children with schizophrenia in the earlier study were more notably symptomatic; all were institutionalized at the time of testing. However, we believe that the difference is more likely a result of methodologic differences between the earlier and current studies—that is, the way the Span stimuli were presented, the sequence of tasks, calibration procedures, and the method of component identification and measurement. In particular, failure to detect group differences in CNV as in the early study has led us to conclude that the CNV differences reported earlier most likely resulted from inadequate baseline adjustment procedures.

Although we did not find reduced amplitude of CNV in the ERPs to correct-response trials of adults or children who had schizophrenia, we did find differences in CNV between correct-response and error trials. Children with schizophrenia produced less CNV on error trials than on correct-response trials. This difference was not evident in normal children or in either group of adults. This finding might suggest that children with schizophrenia were somewhat less prepared for the onset of the Span stimuli on error trials. However, this difference had no effect on the subsequent onset, peak latency, or offset latency of the Span endogenous negativity and thus cannot account for attention allocation deficit (reduced Span endogenous negativity) during stimulus discrimination.

Adults (both normal and those with schizophrenia) produced less CNV in the RT than in the Span condition. However, this was not the case for the children. Because the Span task is very demanding, we anticipated that it would elicit greater mobilization of attentional resources than the RT condition in both age groups. It might be that adults were able to automatize the simple RT task or that the children have less control in regulating resource mobilization.

As in any study of schizophrenia subjects, the deficits observed may reflect, at least in part, motivational factors, preoccupation with internal states, and other more general aspects of the disorder. While we cannot rule out these possibilities, several factors lead us to conclude that the deficits reflect more specific information-processing difficulties. First, we observed no reductions in the amplitude of CNV in either children or
adults who have schizophrenia. In fact, adult schizophrenia subjects showed greater CNV amplitude than their controls. To the extent that the CNV reflects readiness for stimulus processing, preparedness to respond, and arousal level, the schizophrenia subjects appear to be within the normal range with respect to motivation, effort, and task engagement. Second, as noted above, children with schizophrenia showed more endogenous negativity in the RT condition than did control children, thus reducing the amplitude of their difference potentials. We have suggested that this greater endogenous activity reflects greater allocation of attentional effort by the schizophrenia subjects in the simple RT task. This explanation appears to be at odds with the notion of reduced motivation in schizophrenia subjects, since it suggests increased, rather than decreased, effort.

While the results reviewed here pertain only to the Span task, we have obtained similar findings in children who have schizophrenia with two versions of the continuous performance test (CPT; Rosvold et al. 1956) (Strandburg et al. 1990, 1994b). On both easy and hard versions of the CPT, children with schizophrenia had more errors of both omission and commission than normal controls. While CNV amplitude was normal in children with schizophrenia, their ERPs failed to show the increased amplitude of the processing negativity from nontarget to target trials evident in the ERPs of normal children. Thus, both the essential behavioral and ERP findings from the CPT experiments parallel those for the Span.

In summary, despite differences in age at onset, clinical state, and medication status, childhood- and adult-onset schizophrenia subjects exhibit similar deficits in Span performance and produce less endogenous negative activity (Span endogenous negativity) than normal subjects in the 100 to 300 ms following Span stimulus onset. At the same time, the normal CNV amplitudes obtained in schizophrenia subjects during Span performance demonstrate that this deficit is not in their initial preparedness to respond but rather in the specific computational processes that mediate target identification in the Span. Similarities in the correct-response and error trial endogenous negative activity, in the Span endogenous negativity and the NA component identified by Ritter and colleagues (1982, 1983, 1988), and in the processing demands of the Span and C/V tasks suggest that impaired Span performance results from a deficit in serial search processes in schizophrenia subjects. Moreover, the fact that Span performance deficits and abnormalities in Span endogenous negativity are so similar in two schizophrenia groups that differ not only in age at onset, but also in clinical state and the relative predominance of positive and negative symptoms, suggests that this impairment is an expression of an underlying vulnerability to this disorder. Finally, the fact that childhood- and adult-onset schizophrenia subjects share behavioral and electrophysiological manifestations of this attentional/information-processing deficit supports the notion that both groups fall on the same continuum of schizophrenic disorders.

References


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