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# Childhood motor coordination and adult schizophrenia-spectrum disorder

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## Abstract

**Objective—**This study examined whether motor coordination difficulties assessed in childhood predict later adult schizophrenia-spectrum outcomes.

**Method—**A sample of 265 Danish children was administered a standardized childhood neurological examination in 1972 when participants were between 10 and 13 years old. Of the sample, 244 children had available diagnostic information as adults. Participants fell into one of three groups. Children whose mothers or fathers had a psychiatric hospital diagnosis of schizophrenia comprised the first group (N=94). Children who had at least one parent with a psychiatric record of hospitalization for a non-psychotic disorder comprised the second group (N=84). Children with no parental records of psychiatric hospitalization comprised the third group (N=66). Psychiatric outcomes of the offspring were assessed through psychiatric interviews in 1992 when participants were between 31 and 33 years of age, as well as through a scan of national psychiatric registers completed in May 2007.

**Results—**Results indicate that children who later developed a schizophrenia-spectrum disorder (n=32) displayed significantly higher scores on a scale of coordination deficits compared with those who did not develop an identified mental illness (n=133).

**Conclusions—**Results from this study provide further support for the neurodevelopmental hypothesis of schizophrenia, and underscore the potential role of cerebellar and/or basal ganglia abnormalities in the etiology and pathophysiology of schizophrenia.

## Introduction

A host of research documents minor neurological abnormalities in people with schizophrenia. Neurological soft signs are frequently cited abnormalities that have been defined as “non-localizing neurological abnormalities that cannot be related to impairment of a specific brain region or are not believed to be part of a well-defined neurological syndrome” (p. 959; 1). Soft signs commonly observed in adults diagnosed with schizophrenia include motor incoordination, motor sequencing impairment, sensory integrative dysfunction, and eye movement abnormalities (e.g., 2–7). Since neurological soft signs are common not only among people with schizophrenia, but also among their first degree relatives, it has been proposed that these signs reflect a genetically transmitted biological marker of risk for the disorder (e.g., 4, 8–11).

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Additionally, a widely cited meta-analysis examining neurocognitive deficits in schizophrenia relative to controls reported neuromotor abnormalities as the second largest effect of the 22 assessed domains (13). The majority of studies revealing neuromotor dysfunction in schizophrenia are based on examination of individuals with full-blown schizophrenia in adulthood. Studying those already diagnosed with schizophrenia makes it impossible to determine if dysfunction precedes schizophrenia or rather if it is a byproduct of disease onset or treatment. Although a few studies have uncovered a link between motor abnormalities and schizophrenia among neuroleptic-naïve individuals (e.g.,14), and in individuals experiencing their first-episode of psychosis (e.g., 15), studies examining individuals before illness onset provide the most direct evidence for pre-existing neurological dysfunction preceding psychosis (8, 10, 16–19).

## Prospective Evidence of Coordination Deficits in Schizophrenia

Motor incoordination stands out as perhaps the most frequently reported category of soft signs in schizophrenia research, as well as among those at risk (12). Motor coordination deficits have frequently been cited as significant discriminators of the schizophrenia neurodevelopmental diathesis (e.g., 20–21), and have been referred to as “the most common childhood neuromotor deviation” (11; pg. 68). Similarly, in a comprehensive review of prospective high-risk projects, it is stated that “motor uncoordination was the most common finding differentiating high-risk children from controls in many high-risk studies.…” (21; pg. 251). Relatively few prospective studies (8, 16), however, have specifically examined whether childhood coordination deficits predict the development of schizophrenia-spectrum disorders in adulthood.

## Present Study

Despite advances, the literature on the pre-morbid neurological functioning of individuals with schizophrenia and schizophrenia-spectrum disorders still leaves unanswered questions. For instance, there is no strong consensus implicating a given site in the brain, as expressed in neurological examination results, which is dysfunctional. Notwithstanding great value in the existing literature in terms of contributing to understanding of neurological dysfunction preceding schizophrenia, further research is needed. As a whole, few high-risk prospective or large prospective cohort studies have investigated coordination deviations in childhood. Of those published prospective studies of neuromotor functioning, the number of individuals who developed a schizophrenia-spectrum disorder was exceedingly small. Additionally, the measures of neuromotor deviations often vary and are not always standardized. Furthermore, some reports employ composite examination ratings, combining not only multiple motor findings, but also cognitive factors (e.g., 19). Unfortunately, summing disparate types of examination data into total scores makes it difficult to extract information with neurological localizing significance.

Given the strong relationship between schizophrenia and coordination in previous research, as well as recognizing the advantages of high-risk prospective research, the aim of the present study is to investigate motor coordination at age 10 to 13, as assessed through a thorough neurological examination, and risk of subsequently developing schizophrenia-spectrum disorders. In light of the existing literature, we tested the hypothesis that motor coordination deficits in childhood predict adult schizophrenia-spectrum disorders in adulthood relative to either an adult outcome of a non-psychotic mental illness or no diagnosis.

# Methods

## Subjects

The present study investigated children at high-risk for schizophrenia from the Copenhagen Perinatal Cohort, comprising 9,125 individuals born between September 1, 1959 and December 31, 1961 at Rigshospitalet in Copenhagen (22). In order to identify high-risk children and comparison subjects, in 1961 the lifetime record of parental psychiatric admissions was checked through the Danish psychiatric record for the parents of the birth cohort. In 1972, 265 10–13-year-old children from this cohort were intensively examined (23).

In 1992, when the offspring were between 31 and 33 years of age, their psychiatric status was ascertained. A psychiatrist administered two structured clinical psychiatric interviews, the Structured Clinical Interview for DSM-III-R (SCID) (24) and the psychosis section of the Present State Examination (25). In addition, Danish psychiatric hospital records of the subjects were examined. A detailed coding scheme yielded DSM-III-R diagnoses. An additional attempt to ascertain diagnostic status was made through scanning of the Danish Psychiatric Central Registry between 1994 and 2007. Previous research suggests that scanning national registers is a valid method for obtaining psychiatric diagnoses (e.g., 26). As a function of this process, we identified six additional participants who met the ICD-10 criteria for schizophrenia, schizotypy, paranoid psychosis, or delusional disorder, and ten who met criteria for a non-psychotic disorder. By May 2007, 32 subjects were identified who met criteria for a disorder within the schizophrenia-spectrum. It is noteworthy to mention that although the use of different diagnostic systems was inevitable given the longitudinal nature of the present study, a number of recent studies 1) use different diagnostic systems interchangeably for the spectrum (e.g. 27), 2) suggest high diagnostic agreement across diagnostic systems for the spectrum (e.g., 28), and 3) indicate comparable estimates for the spectrum between diagnostic systems (29). Given these findings, we do not anticipate significant inconsistencies between diagnostic systems used in the present study.

On the basis of the interviews and/or hospital records, we obtained adult diagnostic outcomes for 244 of the 265 subjects (92% successful follow-up). Follow-up rate did not significantly differ by risk status (94 of 102 High-Risk; 84 of 89 Other-Risk, 66 of 74 Low-Risk). The follow-up sample comprised three groups. All children whose mother or father had a psychiatric hospital diagnosis of schizophrenia comprised the first group (N=94). A second group consisted of 84 children with a parent with a psychiatric record of hospitalization for a non-psychotic disorder. The remaining 66 subjects were comparison subjects drawn from the original cohort with no parental records of psychiatric hospitalization. In the original design of the study, an effort was made to match the control subjects to the high-risk subjects on the basis of race (all subjects were Caucasian), gender, social class, and parents' age. Although the majority of subjects did not change risk status over time, some shifted risk groups as a result of lifetime parental psychiatric hospitalization records ascertained through 2007 Danish Psychiatric Central Registry scanning. Given a shift in the original matching protocol, to assess whether groups were still equivalent based on demographic information, groups were compared on the above mentioned demographic characteristics. No significant differences between risk groups were found. Further consideration of this issue is taken into account in a subsequent statistical analysis. Diagnostic outcome and risk status information is presented in Table 1.

After complete description of the study to the subjects, written informed consent was obtained. All procedures were in accordance with the ethical standards set forth with the committee on human experimentation and with the Helsinki Declaration of 1975.

### The 1972 Neurological Examination

All 265 children were examined between the ages of 10 and 13 at the Psykologisk Institute in Copenhagen by an experienced child neurologist (N.M.) who was blind to information about the parents' psychiatric status and of course eventual psychiatric diagnosis. A detailed description of the 1972 neurological examination has been given elsewhere (20). In brief, the examination consisted partly of subtests drawn from traditional adult neurological examinations, from subtests known from pediatric neurological examination procedures, and from motor performance tests described in the literature at the time (30). We have previously documented tests of minor physical anomalies, ocular alignment, and laterality from the comprehensive battery used in the current study (22, 31–32). For this study, we were particularly interested in measures of coordination, an area with known associations to schizophrenia. In total there were 13 coordination tasks administered in the 1972 assessment. These tasks roughly covered the same areas of motor coordination functioning as described in the review by Boks et al. (3) and are consistent with major neurological batteries (e.g., 33–34). Four measures of coordination of interest were excluded due to zero variance, 1) left and 2) right finger to nose, 3) left and 4) right heel to knee). The remaining nine tests were considered as measures of coordination (see Table 2).

### Statistical Analyses

The coordination variables were generally scored on a continuum from normal to abnormal. The metric for certain tests varied, so in order to create a “coordination scale” the nine coordination measures were individually standardized and summed. Higher scores indicate poorer performance. To address rare instances of missing data (13 missing data points from a total of 2,385 possible), within group mean substitution was employed. The distributional properties of the resulting coordination scale were such that they appeared not likely to violate assumptions of normality in analyses (skewness=1.20, SE=.16, kurtosis=.94, SE=.31), and the internal consistency of the scale was high (alpha = .89).

The primary analysis was a multinomial logistic regression performed to assess the ability of genetic risk and childhood coordination to predict adult diagnostic outcome. The dependent variable was outcome (spectrum, other, no mental illness), with genetic risk and coordination serving as independent variables (predictors). To increase power for the primary analysis, genetic risk status was dichotomized into High-Risk and Non-High-Risk. The potential interaction between genetic risk and coordination was also assessed. Participants shifted groups from the original case-controlled design, therefore analyses were performed controlling for variables for which the original groups were matched (parental age, marital status, social class, sex).

## Results

Our primary hypothesis was that the coordination scale assessed in childhood would predict those who would later develop schizophrenia-spectrum disorders from those who would not. The average coordination scale score was highest for the spectrum group (mean = 3.37, SD = 8.0), followed by the other psychopathology group (mean = .38, SD = 6.40), and the no mental illness group (mean = −1.03, SD = 6.03). Given the importance of genetic risk factors for developing schizophrenia-spectrum disorders, as well as the role of genetic risk in the design of the project, risk status was considered an independent variable in addition to coordination in predicting outcome. As there were only two individuals in the Low-Risk group who developed a spectrum disorder, the Low-Risk and Other-Risk group were combined. A multinomial logistic regression was conducted to test the ability of genetic risk and the childhood coordination scale to predict adult diagnostic outcome. The overall model was significant ($\chi^2$ (4) = 23.21, $p < .001$) and yielded a Nagelkerke pseudo $R^2$ = .11. Both

the coordination scale and genetic risk status emerged as significant predictors (see Table 3)[1]. Additionally, a possible interaction between coordination and genetic risk was also assessed. The chi-square difference test comparing the model with and without the interaction terms found that the addition of the interaction term to the model was non-significant ($\chi^2(2) = .27$, $p = .87$). We were also interested in whether coordination deficits were mediated by genetic risk. No evidence to support a meditational effect was found.[2]

The project was originally designed as a case-control individually matched study of children with a parent with schizophrenia, a parent with a non-psychotic disorder, and parents with no history of mental illness. Some subjects, however, shifted risk groups as parental diagnostic status changed over time (i.e., some parents developed a mental illness after the birth of their child and after the initial risk groups were established). As a result, we conducted a second multinomial logistic regression controlling for variables for which the original groups were matched (sex, maternal age, paternal age, social status, and marital status). None of the controlling variables were significant and the results for Risk and Coordination remained consistent with our original analysis.

### Individual Coordination Scale Items

To provide a comprehensive view of our findings, Table 4 describes how each outcome group scored in terms of categories in which the individual coordination scale items were scored (“normal,” “suggestive,” and “definitive”). Odds ratios of having a normal versus non-normal score between the spectrum and no mental illness groups are also presented. Table 5 displays results for the two continuously scored coordination variables, left and right speeded finger test.

## Discussion

### Coordination Scale

Results from this study suggest childhood differences in coordination between those who do and do not develop a schizophrenia-spectrum disorder in adulthood. Coordination deficits appeared specific to the spectrum group, as subjects who eventually developed a schizophrenia-spectrum disorder exhibited significantly poorer premorbid coordination scores compared to those who did not develop a mental illness, and nearly significantly ($p = .08$) poorer premorbid coordination than those who developed a non-psychotic mental illness in adulthood. These results were found while incorporating genetic risk, and held when controlling for demographic variables. Our primary analyses involved a coordination scale consisting of several individual tests of coordination. The aggregate scale provided increased statistical power to detect differences relative to a single item, and yielded an effect size in the “high” range ($d=.62$) when comparing the spectrum group to the no mental illness outcome group.

The findings of elevated coordination deficits among those who eventually developed schizophrenia-spectrum disorders is consistent with the overall findings in this research domain, as well as with the few existing prospective studies. As in the reports of the Israeli High-Risk Survey (36), the Swedish High-Risk Survey (9), and the New York High-Risk

[1]We also ran the multinomial logistic regression considering all three levels of genetic risk which yielded similar findings.

[2]To assess for mediation, given that the odds ratio in logistic regression is a measure of effect size (35), we obtained the estimates for the mediation effects by subtracting the coefficients associated with coordination when the logistic model contained both coordination and genetic risk as predictors in the equation, from the coefficient of the model when coordination was the only predictor in the logistic model. Then we obtained the odds ratios of the coefficients of mediation effects by taking the exponent. For both spectrum compared to no mental illness and spectrum compared to other psychopathology, both odds ratios were very close to 1, indicating almost no effect size (spectrum vs. NMI, mediation effect OR = .99; mediation effect spectrum vs. OPD, OR = .99).

Project (10), motor abnormalities detected in infancy and/or childhood were associated with an increased risk of subsequent schizophrenia-spectrum outcome. Findings from the current study diverge, however, from a landmark study by Walker and colleagues (18) in that the differences in motor functioning observed among infants in their study group were not observable beyond age two. Substantial methodological differences may account for this inconsistency. Walker et al. rated motor function based upon coding of spontaneous behaviors visible on home movies. In contrast, assessment of infant and child coordination in the current study involved formal, highly structured hands-on examinations performed by a pediatric neurologist. We believe that this approach is more likely to detect neurological soft signs. Additionally, the present study included several other methodological advantages over previous studies, including an assessment of coordination blind to risk group and eventual diagnostic outcome; an analysis of a composite scale, as well as individual items; a familial psychiatric risk control group; and a relatively high number of individuals with a schizophrenia-spectrum disorder at follow-up through middle age. The unique methodological advantages incorporated in the present long-term longitudinal prospective study, along with previous studies documenting movement abnormalities beyond infancy (e.g., 6, 16), together increase confidence in the conclusion that coordination deficits frequently antedate the formal diagnosis of schizophrenia-spectrum disorders, perhaps by as much as two decades, and are detectable at a variety of developmental stages.

Results clearly suggest direct effects between outcome and coordination, and outcome and genetic risk. Analyses did not support an interaction between genetic risk and coordination, or a model whereby coordination deficits are mediated by genetic risk. Although important not to over-interpret null findings (especially in light of our imperfect measure of genetic risk), these findings suggest that coordination predicts over and above, and is independent of genetic risk for schizophrenia. Possible neural and environmental explanations as to how coordination deficits might relay to adult schizophrenia outcome are described below.

## Possible Mechanisms

Multiple motor systems, including the corticospinal/pyramidal, supplemental motor, basal ganglionic/extrapyramidal and cerebellar systems and their associated networks likely contribute to motor task performance in our coordination battery (37–38). That being said, motor incoordination is classically attributed to dysfunction of the cerebellum and/or basal ganglia. This neurological understanding, originally derived from clinical-pathological correlations, is now confirmed by functional magnetic resonance imaging and other strategies (e.g., 5, 39–40). Therefore, we consider it likely that dysfunctions in the cerebellum, basal ganglia, or both play a role in the observed coordination deficits in those who developed a schizophrenia-spectrum disorder.

Rather than abnormalities in specific brain structures such as the cerebellum or basal ganglia, several authors argue that disruptions of specific pathways (i.e., fronto-cerebellar dysfunction, striatal pathology) are responsible for neuromotor dysfunction in schizophrenia (12, 41). For example, Mittal and colleagues (40) suggest that, similar to minor physical anamolies, movement dysfunction potentially reflects subcortical brain dysfunction resulting from prenatal insults. This conclusion appears compatible with our own results, documenting coordination deficits well before symptom onset. Thus, it seems reasonable that both positions are true. That is, individuals with schizophrenia may exhibit both intrinsic dysfunction and neuroanatomical atypicality of the cerebellum and/or basal ganglia, and disruption of the patterns of connectivity through which the cerebellum and perhaps the basal ganglia exerts modifying effects on motor output. Regardless of exact mechanisms and timing, the findings from the present study implicate neural substrate involvement (structural, pathways, or both) in schizophrenia, early in the course of illness, prior to overt psychotic symptomatology.

Collectively, given the likely role of the cerebellum and other related circuits in coordination deficits, our findings might be viewed in the context of Andreasen's unitary model of “dysmetria” (42). Andreasen and colleagues posit that dysfunction of the cerebellum and cortico-cerebellarthalamo-cortical circuits (CCTCC) might be a unifying explanation for diverse motor, cognitive, and psychiatric symptoms of the illness. Recent studies suggest that cerebellar dysfunction might underlie some of the core features of the disease (e.g., cognitive abnormalities) (43), and our findings provide further support that such cerebellar dysfunction precedes illness onset. Andreasen and colleagues suggest that the presence of coordination deficits indicate underlying abnormalities in basic cognitive processes (e.g., perception, associations) that could lead to misinterpretation of external and internal stimuli. Misinterpretation might account for schizophrenia symptoms, ultimately taking the form of psychotic processes (e.g., hallucinations, delusions, thought disorder, negative symptoms).

Previous studies from the current project have reported other neurodevelopmental markers and precursors to spectrum disorders that provide additional information about regions of possible disruption as well as timing of possible insults (e.g., increased MPAs, ocular dis-alignment, atypical laterality) (22, 31–32). Collectively, these findings, along with the findings from the current study, support dysfunction of the CCTCC and perhaps other neural networks and processes (e.g., hemispheric asymmetry) early in life (first and second trimester), well before the onset of more downstream hallmark symptoms (i.e., delusion and hallucinations). It should be mentioned, however, that additional studies are needed to pinpoint specific regions or pathways, as well as timing of disruption and developmental processes, responsible for the diverse symptomatic manifestations of schizophrenia using more advanced techniques over time (43).

From a diathesis-stress perspective (emphasizing environmental stress), research suggests that poor coordination is associated with a number of social, academic, and emotional consequences (44). Additionally, evidence exists to support detrimental effects of coordination abnormalities over time (e.g., 45). Beyond the neurodevelopmental implications of our findings discussed above, it is reasonable to speculate that poor coordination in childhood engenders at least some taxing psychosocial encounters that may contribute to stress within a diathesis-stress framework. It is also reasonable to speculate that these stressful events further exacerbate pre-existing coordination deficits as well as neurological vulnerabilities, resulting in a self-sustaining, iterative, and perhaps progressively detrimental process between coordination and stress.

## Conclusions

This study suffers from some notable limitations. Despite a relatively large number of individuals who developed a schizophrenia-spectrum disorder, the raw number of people in this group limits statistical power for some analyses. This is particularly true of the number of individuals who developed a spectrum disorder who were not in the high-risk group, and might also contribute to only a trend level difference between the schizophrenia-spectrum and other psychopathology outcome group. Another concern shared by all high-risk research is the issue of generalizability to those individuals who develop a spectrum disorder but who do not have a parent with schizophrenia. It is likely, however, that genetic influences play a role in most cases of schizophrenia, even if the parents fail to manifest the disorder phenotypically (46). It should also be mentioned that there was only one neurologist performing the neurological exam, preventing an evaluation of interrater reliability. The neurologist was, however, highly trained and functioning under strict research procedures and conditions.

Despite these limitations, given the strengths and uniqueness of this study, the findings advance the understanding of the development of schizophrenia in several ways. Detecting

coordination deficits prospectively adds considerable support to the notion that coordination dysfunction precede schizophrenia and may be a meaningful expressions of an underlying biomarker. Applying what is known about the mechanisms of coordination deficits to the etiology of schizophrenia offers possible clues into early neural deficits mediating the development of the disorder.

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## Table 1

Primary diagnosis by age, sex, and genetic risk status of subjects.

| | Age | | Sex | | Genetic Risk | | | |
|---|---|---|---|---|---|---|---|---|
| | **Mean** | **SD** | **Male** | **Female** | **HR** | **OR** | **LR** | **Total** |
| **Schizophrenia-spectrum** | | | | | | | | |
| Schizophrenia | 11.5 | .77 | 10 | 8 | 15 | 2 | 1 | **18** |
| Any psychosis or delusional disorder | 11.7 | .83 | 5 | 3 | 4 | 3 | 1 | **8** |
| Schizotypal PD | 11.6 | .57 | 0 | 4 | 1 | 3 | 0 | **4** |
| Paranoid PD | 11.3 | .61 | 0 | 2 | 2 | 0 | 0 | **2** |
| **Total Schizophrenia-spectrum** | 11.6 | .73 | 15 | 17 | 22 | 8 | 2 | **32** |
| **Other Disorders** | | | | | | | | |
| Non-psychotic mood or anxiety disorder | 11.7 | .63 | 12 | 15 | 12 | 11 | 4 | **27** |
| Non-psychotic alcohol/drug abuse | 11.9 | .63 | 23 | 11 | 9 | 17 | 8 | **34** |
| Non-spectrum personality disorders | 11.6 | .83 | 6 | 12 | 7 | 6 | 5 | **18** |
| **Total Other Disorders** | 11.8 | .68 | 41 | 38 | 28 | 34 | 17 | **79** |
| **No mental illness** | | | | | | | | |
| **Total No diagnosis** | 11.7 | .64 | 64 | 69 | 44 | 42 | 47 | **133** |
| **All Participants** | **11.7** | **.67** | **120** | **124** | **94** | **84** | **66** | **244** |

**Table 2**

The final nine tasks selected for the current study as measures of coordination.

| | Motor Coordination Task |
|---|---|
| 1. | Left diadochokinesia |
| 2. | Right diadochokinesia |
| 3. | Left finger opposition test |
| 4. | Right finger opposition test |
| 5. | Left speeded finger opposition test |
| 6. | Right speeded finger opposition test |
| 7. | Right index finger and right foot tap |
| 8. | Right and left index finger and right foot tap |
| 9. | Right hand left hand opens closes |

## Table 3

Multinomial logistic regression predicting adult diagnostic outcome.

| Predictor: Coordination only | | | | | | |
|---|---|---|---|---|---|---|
| **Groups** | **Predictor** | **df** | **Wald $\chi^2$** | **B** | **Odds Ratio** | **C.I.** |
| OPD vs. Spectrum | Coordination | 1 | 3.85 * | −.06 | .95 | .90 – 1.00 |
| NMI vs. Spectrum | Coordination | 1 | 10.95 ** | −.09 | .91 | .86 – .96 |
| **Predictors: Genetic risk and coordination** | | | | | | |
| OPD vs. Spectrum | Risk | 1 | | −1.34 | .26 | .11 – .64 |
| | Coordination | 1 | 2.88 * | −.05 | .95 | .90 – 1.00 |
| NMI vs. Spectrum | Risk | 1 | 10.64 ** | −1.41 | .25 | .11 – .57 |
| | Coordination | 1 | 8.82 ** | −.09 | .92 | .87 – .97 |

* $p < .10$

** $p < .05$

*** $p < .01$

**Table 4**

Coordination item score by diagnostic outcome.

| Coordination Variable | Schizophrenia Spectrum Group | | | | | | Other Psychopathology Group | | | | | | No Mental Illness Group | | | | | | Odds Ratio: Spectrum v.NMI | 95% CI |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Normal | | Suggestive | | Definitive | | Normal | | Suggestive | | Definitive | | Normal | | Suggestive | | Definitive | | | |
| | n | % | n | % | n | % | n | % | n | % | n | % | n | % | n | % | n | % | | |
| Diadochokinesia (right)[a] | 15 | 50 | 13 | 43 | 2 | 7 | 54 | 69 | 21 | 27 | 3 | 4 | 99 | 76 | 27 | 21 | 4 | 3 | 3.7 | 1.6 – 8.4 |
| Diadochokinesia (left)[a] | 7 | 23 | 13 | 43 | 10 | 33 | 32 | 41 | 28 | 36 | 18 | 23 | 56 | 43 | 60 | 46 | 14 | 11 | 2.5 | 1.0 – 6.2 |
| Finger-opposition (right) | 19 | 59 | 7 | 22 | 6 | 19 | 48 | 61 | 25 | 32 | 6 | 8 | 92 | 69 | 30 | 23 | 11 | 8 | 1.5 | .7 – 3.4 |
| Finger-opposition (left) | 17 | 53 | 9 | 28 | 6 | 19 | 45 | 57 | 26 | 33 | 8 | 10 | 90 | 68 | 33 | 25 | 10 | 8 | 1.8 | .8 – 4.0 |
| Right index finger and right foot tap | 23 | 72 | 6 | 19 | 3 | 9 | 62 | 79 | 14 | 18 | 3 | 4 | 111 | 84 | 18 | 14 | 4 | 3 | 2.0 | .8 – 4.8 |
| Right and left index finger and right foot tap[a] | 13 | 41 | 6 | 19 | 13 | 41 | 42 | 53 | 16 | 20 | 21 | 27 | 69 | 52 | 41 | 31 | 23 | 17 | 2.5 | 1.1 – 5.5 |
| Right hand left hand opens-closes[a] | 14 | 44 | 16 | 50 | 2 | 6 | 47 | 60 | 29 | 37 | 2 | 3 | 84 | 63 | 48 | 36 | 1 | 1 | 2.2 | 1.0 – 4.8 |

[a]Spectrum and No Mental Illness groups significantly differ at the $p<.05$ level.

## Table 5

Left and right speeded finger tests speed by outcome group.

| Coordination Variable | | Schizophrenia Spectrum Group (N=32) | Other Psychopathology Group (N=79) | No Mental Illness Group (N=133) |
|---|---|---|---|---|
| **Left Speeded Finger** [a,b] | Mean Time (sec) | 17.0 | 14.8 | 13.8 |
| | SD | 5.6 | 4.0 | 3.7 |
| **Right Speeded Finger** [b] | Mean Time (sec) | 16.3 | 14.4 | 14.3 |
| | SD | 5.5 | 3.6 | 4.3 |

[a] Spectrum and Other Psychopathology Group significantly differ at the p<.05 level

[b] Spectrum and No Mental Illness Group significantly differ at the p<.05 level