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SOFT NEUROLOGICAL SIGNS AND SCHIZOPHRENIA – A LOOKING GLASS INTO CORE PATHOLOGY?

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Cvetić et al (2009) raise extremely interesting points in chosing to investigate soft neurological signs, which have arguably been forgotten by many psychiatrists. These abnormalities are found in 50-65% of patients with schizophrenia, and yet neurological examinations seem to be so rarely performed clinically (Heinrichs & Buchanan 1988). Perhaps it is time for us to consider again how important these signs might be as a diagnostic marker.

In support of this suggestion is evidence showing that neurological abnormalities of motor function might actually be quite central to the disease process. Ridler et al. (Ridler 2006) have recently found that people with schizophrenia showed both delayed motor development in early childhood (implying abnormalities of development of motor systems) and impaired adult executive function. Additionally, normal subjects showed a positive correlation between motor development and executive function (Ridler 2006). This seems to imply that infant motor function and adult executive function are closely linked, and that developmental abnormalities of motor function might be extremely important to the pathogenesis of schizophrenia, rather than an idiosyncracy of these patients. Indeed, the idea that deficits in early formation of motor systems might actually cause schizophrenia has received some support (Cannon 2002, Erlenmeyer-Kimling 2000, Walker 1994, Jones 1994).

Evidence from neuroimaging further supports the idea that abnormalities of motor cortical and subcortical regions of the brain are central to schizophrenia. Ridler et al. (2006) examined schizophrenic patients and showed a discontinuity between Infant Motor Development (IMD) and both premotor gray matter volume and fronto-parietal white matter volumes in adulthood. They argue that, since neuronal connectivity deficits are known to be important in the pathogenesis of schizophrenia, this implies that the premotor cortex is essentially disconnected in these patients, and cannot play its normal role in motor function (Ridler 2006). It is also argued that, since the motor cortex (with its links to the cerebellum) is important in adult executive function, this disconnectivity might also explain executive deficits in schizophrenic adults (Heyder 2004, Erlenmeyer-Kimling 2000).

This evidence strongly suggests that abnormal motor development plays a central role in causing some of the main deficits observed in adult schizophrenia. Importantly, it is fairly easy to see how underlying deficits in motor function might produce not only executive deficits, but also Soft Neurological Signs, which we observe in schizophrenia. For example, premotor cortical disconnection might account for deficiencies in sensorimotor integration – for example in the "opposition index finger-thumb" test, dysdiadochokinesia, and finger-nose test, and in sequencing of complex motor acts – for example in the "fist-ring", "fist-brink-palm" tests.

If these soft neurological signs are actually good indicators of the 'core pathology' of the condition, then perhaps we are undervaluing them. Examination of these signs might provide us with an invaluable method for diagnosing schizophrenia, as opposed to other disorders (which may result in similar symptoms) such as paranoid personality disorder or delusional disorder. Only recently, Thomann et al. (2009) found that, even in first episodes of schizophrenia, soft neurological signs were significantly increased compared to controls. Chan et al. (2009) have also recently supported the idea that soft neurological signs appear in a majority of the schizophrenia patient population.

One would expect that there would be different markers on neuro-imaging which would correlate with different soft neurological signs and different groups of symptoms of schizophrenia. This is illustrated by Janssen et al. (Janssen 2009)

who have shown in adolescent first psychotic episode patients that 'increased severity of sensory integration neurological soft signs correlate with smaller right and left thalamus volume, while increased severity of sequencing of complex motor acts neurological soft signs correlate with smaller right caudate volume' (Janssen 2009). In Janssen's study the PANSS negative symptom score was included as a covariate because it was positively associated with the sensory integration subscale score, the sequencing of complex motor acts subscale score and the other signs subscale score of the Neurological Evaluation Scale. An association between negative symptom severity and severity of neurological soft signs had been previously reported (Mayoral 2008). Hence the finding reported by Janssen of a correlation between increased severity of neurological soft signs and with smaller right and left thalamus volume and smaller right caudate volume fits well with Cvetić et al's clinical observations (Cvetić 2009).

It is worth considering whether it might be useful to neurologically examine patients with psychotic symptoms. We might be able to use Soft Neurological Signs as a 'looking glass' into the core pathology of schizophrenia.

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