

REVIEW ARTICLE

PRINCIPLE OF DISSOLUTION AND PRIMITIVE REFLEXES IN ADHD

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Abstract

According to current findings, in the history of neurology proposed by Hughlings Jackson, certain later developed functions during ontogenesis of the central nervous system (CNS) tend to replace the older ones. In this context, recent and historical findings suggest that certain later developed cognitive and motor functions during brain ontogenesis related to higher levels of coordination tend to replace the older ones and their persistence is linked to various neuropsychiatric disorders. Particularly important functional disturbances in ADHD developed early in life likely linked to dissolution process are balance deficits linked to dysfunctions of higher levels of coordination related to neurophysiological and mental functions that typically occur in ADHD. In this context, recent data suggest that one of the important aspects of normal development that may play a role in ADHD is suppression of the so-called primitive reflexes. Taken together these data suggest that ADHD symptoms may present a compensatory process related to interference of more primitive neural mechanism, as related to primitive reflexes, with higher levels of brain functions linked to coordination and balance due to insufficiently developed cognitive and motor integration.

Key words: *ADHD; Developmental disorders; Dissolution; Dissociation; Primitive reflexes*

1. INTRODUCTION

Historical concept of dissolution proposed in the 19th century Hughlings Jackson, who established some basic principles of neurology and psychiatry suggesting that certain later developed functions during ontogenesis of the central nervous system (CNS) tend to replace the older ones when higher stages of development of the CNS has been successfully achieved (Andermann, 1997; Franz & Gillett, 2011; Jacyna, 2011). Jackson proposed a theory of the evolution and dissolution of neural functions based on ontogenetically successive complex neuronal levels that enable performance of more adaptive functions and proposed that disinhibition or release from control present particular characteristic of various neurological disorders (Franz & Gillett, 2011). Although historically old, this principle is still valid and enables to understand basic principles of neurological and psychological functions and their disturbed development related to various neurological and psychiatric disorders.

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2. JACKSON'S PRINCIPLE OF DISSOLUTION

Hughlings Jackson started his scientific work in 1850s and proposed basic concepts of the evolutionary organization of the nervous system for which he proposed three levels: a lower- medulla and spinal cord, a middle- motor areas of the cortex, and a higher- prefrontal areas and suggested that the highest centres create physical basis of mind and consciousness. In his basic and still valid concept Jackson proposed that the highest centres, through the intermediation of the middle and lowest centres re-represent all parts of the organism which enables representations of sensory processes and bodily movements in more complex neural structures that create integration of higher and lower mental functions (Jackson, 1884, 1931; Franz & Gillett, 2011; Jacyna, 2011).

Based on this concept Jackson 1870s described and studied pathological states of mind, the so-called "dreamy states", frequently related to temporal lobe seizures that due to disruption of usual order related to re-representations and specific roles of the highest centres lead to disturbances of consciousness (Jackson, 1884, 1931; Ellenberger, 1970; Meares, 1999; Hogan, & Kaiboriboon, 2003, 2004). Jackson proposed interpretation of the dreamy states as a process of disintegration or "dissolution" and described them as a spectrum of psychiatric symptoms similar to "daydreaming" that is caused by disturbed coordination and disinhibition in neural systems and a release of lower levels of brain functions because of inactivated inhibition of highest functional levels. For theoretical explanation of his clinical data Jackson used the principle of dissolution. In this context the dissolution means "breaking" harmony and continuity of brain functions metaphorically comparable to random "crystallization" from the "solution" from which due to some reasons will become "granulated" or "dissolved" and original continuity is lost. In this context because of loss of control of higher functional levels originally suppressed and regulated more primitive functions are released and are not under control, which causes disturbed functions of consciousness, automatic manifestations due to the dysregulation and exaggeration of more primitive functions (Ellenberger, 1970; Meares, 1999).

Jackson used the principle of dissolution as a description of typical processes that due to functional incompatibility or mutual coordination cause "functional dissociation" leading to more automatic behavior with less voluntary control and less complex than usually is achieved in normally developed or intact brain states because there is a loss of later developed higher nervous functions or developmental deficits that mainly in their sensitive stages of development lead to a dysregulation and exaggeration of functions representing ontogenetically and evolutionary lower level of development (Jackson, 1984; Ellenberger, 1970; Meares, 1999).

2. PROCESS OF DISSOLUTION AND PRIMITIVE REFLEXES

As later and recent findings show, main risk factors leading to the process of dissolution are during sensitive and specific developmental stages of brain functions that are also particularly vulnerable to various insults such as brain damages, toxic influences or psychological stress (Teicher, Tomoda, & Andersen, 2006; Fagiolini, Jensen, & Champagne, 2009; Kolb & Gibb, 2011). One of the particularly important postnatal developmental deficits that likely also have various etiological backgrounds influencing higher motor and cognitive functions is persisting of the so-called primitive reflexes (Allen & Capute, 1986; Zafeiriou, 2004; Sanders & Gillig, 2011) as for example Symmetric Tonic Neck Reflex (STNR) Asymmetric Tonic Neck Reflex (ATNR) and some others (Zafeiriou, 2004; Ellis et al., 2012). The primitive (or primary) reflexes (Touwen, 1984; Capute & Accardo, 1991) present specific forms of innate 'behavioral movement patterns' (Niklason, 2012) that are replaced by higher motor and cognitive functions (Allen & Capute, 1986; Zafeiriou, 2004; Sanders & Gillig, 2011) and when they occur in later stages of development they may present a form of 'soft neurological signs' (Polatajko, 1999). According to current findings persisting of these

reflexes is related to certain specific neuropsychiatric or neurological disorders (Keshavan & Yeragani, 1987; Youssef & Waddington, 1988; Zafeiriou, 2004; Links et al., 2010; Nicolson et al., 2011; Sanders & Gillig, 2011). In this context, recent clinical evidence indicates that manifestations of primitive reflexes in later age than is ontogenetically typical are likely linked to a frontal lobe dysfunctions and cortical disinhibition, and may occur in various neuropsychiatric syndromes, for example in schizophrenia or bipolar disorders (Youssef & Waddington, 1988), dementias and Parkinsonism (Links, Merims, Binns, Freedman, & Chow, 2010), delirium (Nicolson, S. E., Chabon, Larsen, Kelly, Potter, & Stern, 2011) and some other neuropsychiatric disorders (Keshavan & Yeragani, 1987; Zafeiriou, 2004; Sanders & Gillig, 2011). These data suggest that persistent (or retained) primary reflexes in general represent evolutionary lower levels of neurophysiological processes that may interfere with processing on higher levels and cause “dissolution”, in the sense of Jackson’s concept, which may be linked to various neuropsychiatric conditions (Franz & Gillett, 2011; Jacyna, 2011).

3. ATTENTION DEFICIT AND HYPERACTIVITY DISORDER (ADHD) AND PRIMITIVE REFLEXES

These findings about primitive reflexes are in agreement with basic neurological concepts discovered by Jackson, who proposed that certain later developed functions during ontogenesis of the central nervous system (CNS) tend to replace the older ones (Andermann, 1997; Franz & Gillett, 2011; Jacyna, 2011). In this context the dissolution may occur when higher stages of the CNS development were not successfully achieved or are dysfunctional due to damages. As a consequence the lower neural functions may be disinhibited and their release from control may lead to dysregulation of later developed adaptive functions and lead to dissolution (Franz & Gillett, 2011).

Typical consequences of dissolution lead to discoordination on various functional levels and specific disturbances developed early in life. Currently there is mild evidence about presence of retained (not suppressed) primitive reflexes in ADHD and until this time just two studies have been published (Taylor, Houghton, & Chapman, 2004; Konicarova & Bob, 2012). These preliminary findings in ADHD children are in agreement with few reported studies in children with dyslexia documenting higher level of retained primary reflexes (McPhillips, Hepper, & Mulhern, 2000; McPhillips & Jordan-Black, 2007).

These preliminary but conceptually important findings about retained primitive reflexes in ADHD could be in principle related reported balance deficits that frequently occur in ADHD children and could be linked to dysfunctions of higher levels of coordination related to neurophysiological and mental functions (Buderath et al., 2009; D’Agati et al., 2010; Ghanizadeh, 2011). Recent findings show that high proportion of children with ADHD exhibit altered balance and motor abnormalities (Buderath et al., 2009; D’Agati et al., 2010; Ghanizadeh, 2011). According to brain imaging studies these balance deficits likely are linked to prefrontal cortex deficits that influence attention and executive functions (Arnsten, 2009; Shaw & Rabin, 2009; Makris et al., 2009) and also may have cerebellar origin. In addition ADHD children in many cases exhibit atrophy in cerebellar regions associated with balance and gait control and these balance and motor dysfunctions are most likely linked to inhibitory deficits due to cerebellar abnormalities (Berquin et al., 1998; Baillieux et al., 2008; Buderath et al., 2009; O’Halloran et al., 2012).

4. CONCLUSIONS AND FUTURE DIRECTIONS

Taken together recent data suggest that ADHD symptoms may present a process related to interference of more primitive neural mechanism such as primitive reflexes with higher levels of brain functions due to insufficiently developed cognitive and motor integration related to disturbed balance in ADHD. The specific deficits and symptoms that occur in

ADHD may be particularly influenced by disproportional development in which higher certain motor and cognitive functions have been developed normally according to usual morphogenetic plans and certain others not which likely is compensated by persisting functions representing ontogenetically and evolutionary lower level of development.

As a consequence, in the case of ADHD these retained reflexes and discoordination related to disturbed balance may explain attentional dysregulation that frequently occurs as a response to various stimuli during behavioral and cognitive tasks that is likely linked to a conflict between higher and lower level of cognitive and motor functions during brain processing. In the sense of Jacksonian theory these retained primary reflexes may cause “dissolution” (or disintegration) of mental functions that may be linked to dissociative symptoms frequently observed in children with ADHD in its various symptomatic forms (Endo, Sugiyama, & Someya, 2006; Johnson, Robertson, Kelly, Silk, Barry, Daibhis, Watchorn, Keavey, Fitzgerald, Gallagher, Gill, & Bellgrove, 2007).

In the context of recent findings continuing research and increase number of studies focused on relationships of ADHD symptoms with retained primitive reflexes, disturbed balance, traumatic stress and dissociative symptoms might show interesting and useful data. This continuing research specifically would need to increase number of participants in the research studies including follow up studies that could help to understand certain developmental aspects of ADHD in children and define implications of these data for clinical and educational practice.

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REFERENCES

- Allen, M. C., Capute A. J. (1986). The evolution of primitive reflexes in extremely premature infants. *Pediatric Research*, 20, 1284-9.
- Andermann, A., A. (1997). Hughlings Jackson's deductive science of the nervous system: a product of his thought collective and formative years. *Neurology*, 48, 471-81.
- Arnsten, A.F. (2009). Toward a new understanding of attention-deficit hyperactivity disorder pathophysiology: an important role for prefrontal cortex dysfunction. *CNS Drugs*, 23 (Suppl 1) 33-41.
- Baillieux, H., De Smet, H.J., Paquier, P.F., De Deyn, P.P., & Marien, P. (2008). Cerebellar neurocognition: insights into the bottom of the brain. *Clinical Neurology and Neurosurgery*, 110(8), 763-73.
- Berquin, P.C., Giedd, J.N., Jacobsen, L.K., Hamburger, S.D., Krain, A.L., Rapoport, J.L., Castellanos, F.X. (1998). Cerebellum in attention-deficit hyperactivity disorder: a morphometric MRI study. *Neurology*, 50(4), 1087-93.
- Buderath, P., Gärtner, K., Frings, M., Christiansen, H., Schoch, B., Konczak, J., Gizewski, E.R., Hebebrand, J., & Timmann, D. (2009). Postural and gait performance in children with attention deficit/ hyperactivity disorder. *Gait & Posture*, 29(2), 249-54.
- Capute, A.J., & Accardo, P.J. (1991). *Developmental disabilities in infancy and childhood*. Baltimore, MD: Paul Brooks.
- D'Agati, E., Casarelli, L., Pitzianti, M.B., & Pasini, A. (2010). Overflow movements and white matter abnormalities in ADHD. *Progress in Neuropsychopharmacol & Biological Psychiatry*, 34(3), 441-5.
- Ellenberger, H.F. (1970). *The Discovery of the Unconscious: The History and Evolution of Dynamic Psychiatry*. New York: Basic.
- Ellis, M.D., Drogos, J., Carmona, C., Keller, T., & Dewald, J.P. (2012). Neck rotation modulates flexion synergy torques indicating an ipsilateral reticulospinal source for impairment in stroke. *Journal of Neurophysiology*, 108(11), 3096-104.
- Endo, T., Sugiyama, T., & Someya, T. (2006). Attention-deficit/ hyperactivity disorder and dissociative disorder among abused children. *Psychiatry and Clinical Neurosciences*, 60, 434-8.

- Fagiolini, M., Jensen, C.L., & Champagne, F.A. (2009) Epigenetic influences on brain development and plasticity. *Current Opinion in Neurobiology*, 19, 207-12.
- Franz, E. A., & Gillett, G. (2011) John Hughlings Jackson's evolutionary neurology: a unifying framework for cognitive neuroscience. *Brain*, 134, 3114-20.
- Ghanizadeh, A. (2011). Predictors of postural stability in children with ADHD. *Journal of Attention Disorders*, 15(7), 604-10.
- Jackson, J.H. (1884). The Croonian Lectures on Evolution and Dissolution of the Nervous System. *British Medical Journal*, 1, 591-3, 660-3, 703-7.
- Jackson, J.H. (1931). *Selected writings of John Hughlings Jackson. Vol 1. On epilepsy and epileptiform convulsions*. Taylor J, editor. London: Hodder and Stoughton.
- Jacyna, L. S. (2011) Process and progress: John Hughlings Jackson's philosophy of science. *Brain*, 134, 3121-6.
- Johnson, K. A., Robertson, I. H., Kelly, S. P., Silk, T. J., Barry, E., Dáibhis, A., Watchorn, A., Keavey, M., Fitzgerald, M., Gallagher, L., Gill, M., & Bellgrove, M.A. (2007). Dissociation in performance of children with ADHD and high-functioning autism on a task of sustained attention. *Neuropsychologia*, 45, 2234-45.
- Keshavan, M. S., & Yeragani, V. K. (1987) Primitive reflexes in psychiatry. *Lancet*, 1, 1264.
- Kolb, B., & Gibb, R. (2011). Brain plasticity and behaviour in the developing brain. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, 20, 265-76.
- Konicarova, J., & Bob, P. (2012). Retained Primitive Reflexes and ADHD in Children. *Activitas Nervosa Superior*, 54, 135-138.
- Links, K. A., Merims, D., Binns, M. A., Freedman, M., & Chow, T.W. (2010) Prevalence of primitive reflexes and Parkinsonian signs in dementia. *Canadian Journal of Neurological Sciences*, 37, 601-7.
- Makris, N., Biederman, J., Monuteaux, M.C., & Seidman, L.J. (2009). Towards conceptualizing a neural systems-based anatomy of attention-deficit/ hyperactivity disorder. *Developmental Neuroscience*, 31(1-2), 36-49.
- Meares, R. (1999). The Contribution of Hughlings Jackson to an Understanding of Dissociation. *American Journal of Psychiatry*, 156, 1850-1855.
- McPhillips, M., & Jordan-Black, J. A. (2007). Primary reflex persistence in children with reading difficulties (dyslexia): A cross-sectional study. *Neuropsychologia*, 45, 748-754.
- McPhillips, M., Hepper, P. G., & Mulhern, G. (2000). Effects of replicating primary-reflex movements on specific reading difficulties in children: a randomised, double-blind, controlled trial. *Lancet*, 355, 537-541.
- Nicolson, S. E., Chabon, B., Larsen, K. A., Kelly, S.E., Potter, A. W., & Stern, T. A. (2011). Primitive reflexes associated with delirium: a prospective trial. *Psychosomatics*, 52, 507-12.
- O'Halloran, C.J., Kinsella, G.J., Storey, E. (2012). The cerebellum and neuropsychological functioning: a critical review. *Journal of Clinical and Experimental Neuropsychology*, 34(1), 35-56.
- Polatajko, H.J. (1999) Developmental Coordination Disorder (DCD): alias, the clumsy child syndrome. In K. Whitmore, H. Hart, & G. Willems (Eds.), *A neurodevelopmental approach to specific learning disorders*. London: Mac Keith Press, pp. 119-133.
- Sanders, R. D., & Gillig, P. M. (2011). Reflexes in psychiatry. *Innovations in Clinical Neuroscience*, 8, 24-9.
- Shaw, P., & Rabin, C. (2009). New insights into attention-deficit/ hyperactivity disorder using structural neuroimaging. *Current Psychiatry Reports*, 11(5), 393-8.
- Taylor, M., Houghton, S., & Chapman, E. (2004). Primitive reflexes and attention deficit/ hyperactivity disorder: Developmental origins of classroom dysfunction. *International Journal of Special Education*, 19, 23-37.
- Teicher, M. H., Tomoda, A., & Andersen, S. L. (2006). Neurobiological consequences of early stress and childhood maltreatment: are results from human and animal studies comparable? *Annals of the New York Academy of Sciences*, 1071, 313-323.
- Touwen, B.C.L. (1984). Primitive reflexes-conceptual or semantic problem. In H.F.R. Prechtl (Ed.), *Continuity of neural functions from prenatal to postnatal life*. Oxford, Great Britain: Spastics International Medical Publications.
- Youssef, H. A., & Waddington, J. L. (1988). Primitive (developmental) reflexes and diffuse cerebral dysfunction in schizophrenia and bipolar affective disorder: overrepresentation in patients with tardive dyskinesia. *Biological Psychiatry*, 23, 791-6.
- Zafeiriou, D. I. (2004). Primitive reflexes and postural reactions in the neurodevelopmental examination. *Pediatric Neurology*, 31, 1-8.