Effect of Visio-Motor Ability on Performance

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ABSTRACT

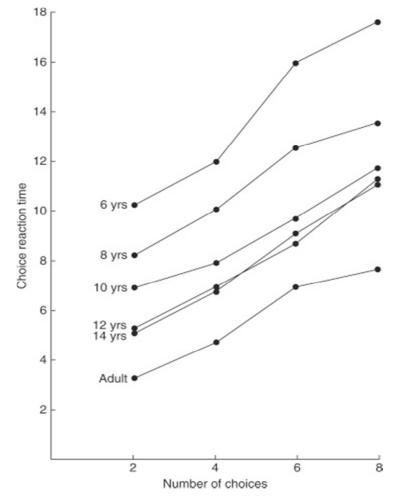
For children with developmental coordination disorder (DCD), the acquisition and performance of everyday visual-motor activities such as buttoning, shoe tying, cutting with scissors or writing, presents a major challenge. Regardless of the activity considered, children with DCD are typically slower and less accurate than their peers. Given the well-acknowledged difficulties of children with DCD, it is surprising to find very few research studies systematically exploring visual-motor skill acquisition and performance in children with DCD Modulation of sensorimotor rhythms (SMR) was suggested as a control signal for brain-computer interfaces (BCI). Yet, there is a population of users estimated between 10 to 50% not able to achieve reliable control and only about 20% of users achieve high (80–100%) performance. Predicting performance prior to BCI use would facilitate selection of the most feasible system for an individual, thus constitute a practical benefit for the user, and increase our knowledge about the correlates of BCI control. Motor ability modulated the impact of task difficulty on visual-motor skill acquisition and task performance. Children with DCD were as fast and as accurate as their peers in their initial performance of the simple, well-learned task (mouse). However, they were slower and less accurate when performing the complex and novel visual-motor task. Over repeated trials, the visual-motor task performance of children with DCD improved on all tasks, even for the simple. With regard to the complex, novel task, once children with DCD understood the features of the task, their performance also improved and approached that of their peers. While children with DCD can generally be characterized as less accurate and slower than their peers, this characterization needs to be specified and qualified; it is probably best not applied to a well-learned task.

KEYWORDS: visiomotor, performance, ability, effect, sensorimotor, control, prediction, machine

Introduction

Motor ability modulated the impact of task difficulty on visual-motor task performance. Children with DCD were as fast and as accurate as their peers in their initial performance of the simple task. However, they were slower and less accurate when performing the complex and novel visual-motor task. Given the well-acknowledged visual-motor difficulties of children with developmental coordination disorder (DCD),[1,2] it is surprising to find few research studies systematically exploring their visual-motor task performance. While children with DCD can generally be characterized as less accurate and slower than their peers, this characterization needs to be specified and qualified; it is probably best not applied to a well-learned, simple task. Visuomotor coordination is amazing. Consider an approaching object like a ball that you would like to catch. This requires a rapid calculation of the path of the ball and being able to extend the arms and position the hands in the appropriate spot at just the right time. The motor command needs to consider not only the timing but also the visuomotor details about joint angles from the eyes to the hand via the neck and head. How is this achieved? The parietal lobe processes visual information that will contribute to the initiation of the motor commands.

In order to arrive at an accurate and precise motor command, a wealth of information needs to be processed about the dynamic and static conditions of the different parts of the body in relation to each other and to the surrounding world. The processing time has been investigated experimentally by having a subject respond to a signal by performing a movement, such as pressing a button when a light goes on.[3,4] The delay is around 0.1–0.2 s and is called the simple reaction time. It represents a minimal delay for a given test situation that cannot be shortened by training.



Discussion

The more complex the situation is, the longer the reaction time. If a choice is involved, such as responding by pressing different buttons to light stimuli of different colors, the time delays become much longer than in the simple reaction time task. The choice reaction task time increases in proportion to the level of complexity and the number of choices.

When young children are tested, their reaction times are much longer than in adults. The simple reaction time for a 6-year-old can be three times that of a 14-year-old,[5,6] and the difference with choice reaction times may be even larger. In everyday life this condition may have severe consequences, such as in an unexpected traffic situation. A 6-year-old will need at least three times more time to interpret what he or she sees. This is the basis for recommending that children below 11 years of age should not ride a bicycle in open traffic. It is noteworthy that this is a gradual maturation process, and training will not shorten the reaction time. During aging the choice reaction time tends to increase again, a factor that can be important when driving a car.[7,8]

The cerebellum is also involved in the coordination of movement. Although it is smaller than the cerebral cortex in volume, it is thought to contain as many nerve cells as the latter. It has a very stereotyped neuronal organization with two types of inputs from mossy and from climbing fibers. These inputs not only carry information from the spinal cord about ongoing movements in all different parts of the body, but they also carry information from the different motor centers about planned movements even before a movement has been executed. The cerebellum also interacts with

practically all parts of the cerebral cortex. This means that it is updated continuously about what goes on in all parts of the body with regard to movement and also about the movements that are planned in the immediate future.[9,10]

Lesions of the cerebellum lead initially to great problems with postural stability and with a lack of accuracy of movements. Most movements can be carried out, but their quality is reduced drastically. The cerebellum was originally thought to be involved exclusively in the coordination of movement. In addition to motor control, evidence has accumulated that the lateral parts of cerebellum may also be involved in different cognitive tasks.

The cerebellum is subdivided into a great number of different microregions that each process different kinds of information. These different regions respond via their output neurons, called Purkinje cells. The cerebellar cortex contains only inhibitory interneurons, and the two input systems, climbing fibers and mossy fibers (via granular cells), provide the excitatory components. All Purkinje cells are inhibitory and project to the cerebellar nuclei, which in turn are excitatory and project to different motor centers in the brainstem and to the cortex via the thalamus.[11,12]

Each granular cell receives very specific input from only a few mossy fibers, which are able to make them fire. The granular cell axons form parallel fibers in the cerebellar cortex, and thousands of parallel fibers impinge on each Purkinje cell that has a very extensive dendritic tree. It is believed that only a small portion of these parallel synapses are functional in any given moment but that they are available to become recruited in learning situations. In contrast, there is only one climbing fiber for each Purkinje cell. The climbing fibers can serve as "error detectors," at least under some conditions. Through the effects of climbing fibers on the Purkinje cells, the synaptic efficacy of the parallel fiber input to the Purkinje cell can be modified (depressed). This change in efficacy can last over many hours and possibly much longer and is referred to as long-term depression (LTD). It is generally thought that this LTD can contribute to motor learning. With regard to basic movements such as posture, walking, and the much-studied eye blink reflex, the cerebellum is clearly involved through each movement phase. It is likely that the fine tuning and modification of movements that are required in different behavioral contexts involve the cerebellum. With regard to the eye blink reflex, motor learning has been demonstrated in terms of associating an unconditioned stimulus with a conditioned one. It requires that the cerebellar cortex be intact, which strongly suggests that the cerebellar cortex contributes importantly to this type of motor learning.[13,14]

There is still, however, much to learn about the actual processing that goes on in the cerebellum, a structure that regulates the quality of motor performance and is involved in some forms of motor learning. A variety of complex motor tasks, including speech and handwriting, can still be performed, but with less accuracy. Thus, the motor programs for these learned tasks cannot be stored exclusively in the cerebellum. When new types of movements are learned, such as riding a bicycle, playing an instrument, or typing at a keyboard, the new stored programs for sequences of motor acts must also involve other structures in the brain, most likely the basal ganglia and the cerebral cortex.

Results

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It is possible that the cerebellum exerts an influence on both spatial orientation and visuomotor coordination. The nature of the deficit displayed by a brain-damaged animal may depend on the extent and regional distribution of the cerebellar pathology. For example, midline as opposed to lateral cerebellar lesions may cause a different profile in navigational abilities. This hypothesis was tested in rats with lesions of cither the midline cerebellum, comprising the vermis and the fastigial nucleus, or the lateral cerebellum, comprising the hemispheres and dentate. Quadrant entries and escape latencies were higher in rats with midline cerebellar lesions but not in rats with lateral cerebellar lesions during visible platform performance. In contrast, the lateral cerebellar group had higher quadrant entries and escape latencies than a sham-operated group during the hidden platform task. These behavioral differences may be explained by the different anatomical connections of the midline as opposed to the lateral cerebellum.[15]

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The midline cerebellum has reciprocal connections with the vestibular system, the superior colliculus, and the lateral geniculate nucleus of the thalamus. The fastigial efferent system includes the frontal eye field via thalamic relay nuclei. Connections with the superior colliculus, frontal eye fields, and lateral geniculate are involved in eye movements in primates, but their functions in rodents remain to be determined. Vermal Purkinje cells receive hippocampal input via brain stem nuclei. In turn, fastigial stimulation alters hippocampal and septal neurons via a multisynaptic pathway. These anatomical connections indicate that the midline cerebellum may integrate visual and vestibular signals in such a way as to permit effective visuomotor coordination. Visual ambient guidance is crucially dependent on the superior colliculus. During water maze testing, lesions of the superior colliculus cause visuomotor deficits. Vestibular stimulation caused by a rotating platform impaired spatial orientation in the Morris maze, underlining the importance of vestibular cues during oriented swimming movements. Mice with genetic lesions of the vestibular system sink beneath the water level. This is not the case with midline cerebellar lesions.

Other brain regions important in sensorimotor integration may be involved in different aspects of visual guidance. For example, lesions of the striatum or dopamine depletion in the same area impaired visible platform performance in the Morris maze. Lesioned animals perseverate in wall hugging, but with repeated training abandon this strategy. It remains to be determined whether rats with midline cerebellar lesions can reach normal values with repeated training on the visible platform. Striatal damage causes the release of perseverative, inappropriate strategies. The dorsal striatum receives vestibular input and may process vestibular information during food-motivated maze tasks and during guided swimming movements. Midline cerebellar damage may decrease the animal's ability to use visual and vestibular stimuli during guided swimming movements. Hippocampal pyramidal cells may use the sensorimotor integration provided by the midline cerebellum for navigational place learning.[16]

Lesions of the fastigial nucleus impaired acquisition of the hidden platform task but not visible platform performance. Thus, visible platform performance is affected by midline cerebellar lesions but not by fastigial lesions. These results imply that some degree of sensorimotor integration for swimming toward a visible goal occurs at the level of the vermis and the flocculonodular lobe. The lateral cerebellum receives neocortical input from association parts of the frontal and parietal lobes via rostral pontine nuclei and sends information back to these areas via thalamic nuclei. In addition to VA-VL thalamic nuclei, the cerebellum projects to the dorsomedial and intralaminar nuclei. the former innervating frontal association cortex and the latter widespread areas of the neocortex. The VA-VL thalamic nuclei are involved in cerebellar input to the parietal cortex. In addition to the parieto–ponto–cerebellar pathway, a parieto–rubro–olivo–cerebellar pathway exists. The lateral cerebellum also sends input to the tectum. The parietal cortex is important in navigational abilities. The role of the medial frontal cortex is also important, but is dependent on methodological factors.

It has been proposed that the neocortico–ponto–cerebellar pathway controls the visual guidance of limb and eye movements. This theory may be extended to navigational abilities. Lesions of the lateral cerebellum in rats impaired the hidden platform task but not the visible platform task, an indication of a deficit in spatial orientation and not in sensorimotor guidance. On the basis of this study, it may be hypothesized that the cerebellum is implicated in the cognitive processes required to control whole body movements in space. Fastigial and dentate efferents to dopamine cells in the substantia nigra may also influence the sensorimotor coordination necessary for visual guidance, as the depletion of striatal dopamine by means of 6-hydroxydopamine caused a visuomotor coordination deficit in the Morris Ibotenate-induced lesions of the striatum also caused this deficit. [15,16]

Conclusions

An ability of performing coordinate transformations is implied in several perceptual and motor activities, like visuomotor coordination and predictive behavior. In fact, the CNS seems to be able to

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deal equally well with forward and inverse mappings, although the latter are usually ill-posed and thus are mathematically 'more difficult' than the former. Reaching movements of the arm or phoneme utterance in speech are clear examples. This is a strong bias, in our view, towards planning models which are intrinsically associative and thus bi-directional. The concept has been applied for developing a theory of the dynamic interaction of cortical maps which can solve coordinate transformations in the most general way: providing the built-in regularization mechanisms which allow to deal equally well with forward and inverse mappings.

Starting from the abstract description of a class of motor tasks in terms of attractor fields, we have proposed that the synthesis of the corresponding motor plans may be 'naturally' performed by exploiting the distributed nature, the topologic organization and the dynamic behavior of spatial representations characteristic of cortical maps.

In particular, we have proposed that a basic mechanism of propagation of a peak of activation, driven by an external target field may account for both the formation of trajectories in distal space and the inverse kinematic problem.

An application to the generation of the sequence of vocal tract configurations corresponding to a continuous sequence of vowels has shown that the proposed model may account for redundancy resolution, and may accommodate different speaking rates and levels of stress.[17]

References

- 1. Smits-Engelsman, B. C. M., Niemeijer, A. S., & van Galen, G. P. (2001). Fine motor deficiencies in children diagnosed as DCD based on poor grapho-motor ability. Human Movement Science, 20, 161-182.
- Smits-Engelsman, B. C. M., Wilson, P. H., Westenberg, Y., & Duysens, J. (2003). Fine motor deficiencies in children with developmental coordination disorder and learning disabilities: An underlying open-loop control deficit. Human Movement Science, 22, 495-513.
- 3. Stephenson, E. A., & Chesson, R. A. (2008). 'Always the guiding hand': Parents' accounts of the long-term implications of developmental co-ordination disorder for their children and families. Child: Care, Health and Development, 34, 335-343.
- 4. Sugden, D. A. (2006). Leeds consensus statement: Developmental coordination disorder as a specific learning difficulty. Leeds: DCD-UK/Dyscovery Centre.
- 5. Summers, J., Larkin, D., & Dewey, D. (2008). Activities of daily living in children with developmental coordination disorder: Dressing, personal hygiene, and eating skills, Human Movement Science, 27, 215-229.
- 6. Szklut, S. E. & Breath, D. M. (2001). Learning Disabilities. In D. A. Umphred (Ed.), Neurological Rehabilitation (4 th edition) (pp. 308-350).
- 7. St-Louis, Missouri: Mosby Inc. Thomas & Thomas, 2008. Tseng, M., Howe, T., Chuang, I., & Hsieh, C. (2007). Co-occurrence of problems in activity level, attention, psychosocial adjustment, reading and writing in children with developmental coordination disorder. International Journal of Rehabilitation Research, 30, 327-332.
- 8. Wang, T. N., Tseng, M. H., Wilson, B. N., Hu, F. C. (2009). Functional performance of children with developmental coordination disorder at home and at school. Developmental Medicine & Child Neurology, 51, 817-25.
- 9. Wechsler, D. (1991). WISC-III: Wechsler intelligence scale for children. San Antonio, TX: Psychological Corporation, Harcourt Brace Jovanovich. 113
- 10. Wickstrom, R. L. (1983). Fundamental Motor Patterns (3 rd edition). Philadelphia: Lea & Febiger. Wilson,

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- 11. B. N., Dewey, D., & Campbell, A. (1998). DCD Questionnaire. Wilson, B., Kaplan, B., Crawford, S., Campbell, A., & Dewey, D. (2000). Reliability and validity of a parent questionnaire on childhood motor skills. American Journal of Occupational Therapy, 54, 484-493.
- 12. Wilson, P. H., & McKenzie, B. E. (1998). Information processing deficits associated with developmental coordination disorder: A meta-analysis of research findings. The Journal of Child Psychology and Psychiatry, 39, 829-840.
- 13. Wolpert, D. M. & Flanagan, J. R. (2001). Motor prediction. Current Biology, 18, R729-R732.
- 14. Wolpert, D. M., & Flanagan, J. R. (2010). Primer: Motor learning. Current Biology, 20, R467-R472.
- 15. Wolpert, D. M., Ghahramani, Z., & Flanagan, J. R. (2001). Perspectives and problems in motor learning. Trends in Cognitive Science, 5, 487-494.
- 16. Zipp, G. P. & Gentile, A. M. (2010). Practice Schedule and the Learning of Motor Skills in Children and Adults: Teaching Implications. Journal of College Teaching and Learning, 7, 35-42.
- 17. Zwicker, J. G., Missiuna, C., Harris, S. R., & Boyd, L. A. (2011). Brain activation associated with motor skill practice in children with Developmental Coordination Disorder: an fMRI study. International Journal of Developmental Neuroscience, 29, 145-152.