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REHABILTATION OF ATAXIC GAIT FOLLOWING CEREBELLAR LEISONS: APPLYING THEORY TO PRACTICE

Introduction

Ataxic gait is characterised by a slow walking speed, irregular steps, difficulties with inter and intra limb coordination and reduced postural stability; all resulting in a high risk of falls (Morton and Bastian, 2007). This can have a huge impact on the everyday lives of individuals with ataxia, who have to deal with the frustrations of having relatively normal muscle strength in all four limbs, but lack the coordination and balance to walk safely and independently. Physiotherapy is the main treatment for ataxic gait (Ataxia UK, 2009), but there is very little high-quality research into the effectiveness of treatments (Martin, Tan, Bragge and Bialocerkowi, 2009).

Studies suggest that a range of physiotherapy interventions have positive benefits on gait outcomes. These include dynamic balance training (Armutlu, Karabudak and Nurlu, 2001), customised interventions targeting balance and independence in activities of daily living (Jones, Lewis, Harrison and Wiles, 1996), physiotherapy using the Bobath approach (Gialanella, Bertolinelli, Monguzzi and Santoro, 2005), and customised gait training (Gill-Body, Popat, Parker and Krebs, 1997). However, the participant groups selected in each study were diverse, including patients with progressive ataxia, multiple sclerosis, and acquired brain injuries. This combined with the fact that the evidence comes from small scale studies (Martin, Tan, Bragge and Bialocerkowi, 2009) makes it difficult for physiotherapists to select the most beneficial intervention for an individual patient.

Given the limited research available, clinical reasoning relating to the choice of therapy interventions for individuals with ataxic gait could be enhanced by a greater understanding of

the neural mechanisms of ataxia. This paper aims to review the mechanisms of the key features of ataxic gait and provide theoretically informed suggestions for physical interventions, which are summarised in table 1. There are several causes of ataxic gait; however this review focuses solely on ataxic gait as a consequence of cerebellar lesions.

Overview of cerebellar function in relation to gait

The cerebellum is vital for motor learning (Ioffe, Chernikova and Ustinova, 2007) as well as having an essential role in anticipatory (feed forward) postural control, and coordination of muscle timing and grading (D'Angelo, 2011). In order to influence motor output, the cerebellum must instantaneously process descending information from the cerebral cortex relating to intended movement, together with ascending information relating to the sensory consequences of the movement that occurred (Marsden and Harris, 2011). Marsden and Harris (2011) suggest that individuals with ataxia face many challenges, including coordination difficulties, decreased ability to learn from movement errors, reduced anticipatory postural control and poor balance reactions. Ataxic gait is a compensatory gait pattern that occurs when the cerebellum is unable to fulfil these roles (Thach and Bastian, 2004). The location and severity of the pathology will determine the nature of the particular presentation in each individual. The following section will discuss each component of ataxic gait, explore the neural mechanisms involved and provide theoretically informed suggestions for therapy interventions. This is then summarised in table 1.

Co-ordination difficulties

Difficulties in timing and coordination of inter- and intra- limb muscles is one of the key impairments experienced by individuals with cerebellar lesions (Dietriches, 2008). The

cerebellum has a key role in controlling timing of muscle activity by processing both motor commands and sensory feedback (Marsden and Harrison, 2011). Understanding the cerebellum's functioning at a cellular level can clarify how this occurs (see figure 1).

Insert figure 1 here

Figure 1. The cellular functioning of the cerebellum (Leach and McManus, 2012)

Mossy fibres bring information to the cerebellum from the cerebral cortex, regarding intention to move and from sensory receptors of the body, relaying information about the sensory consequences of the movement which is occurring (Marsden and Harris, 2011), as shown in figure 1. Three to five mossy fibres form excitatory synapses with each granule cell. Golgi cells also form inhibitory synapses with granule cells. The granule cells form parallel fibres which then synapse with thousands of Purkinje cells, the output cells of the cerebellum (Carey, 2011). These Purkinje cells relay information out of the cerebellum via the deep cerebellar or vestibular nuclei to influence either the motor areas of the cerebral cortex, or the descending motor pathways (Bear, Connors and Paradiso, 2007).

Recent research conducted by D'Angelo and colleagues (2010) suggests that synthesis of sensory information from the body (including somatosensory, proprioceptive, visual and vestibular information), with information about intention to move (from the cerebral cortex), occurs throughout the cerebellum. This occurs first at the mossy fibre-granule cell synapses. Additionally, D'Angelo et al (2010) found that the Golgi cells can have an inhibitory effect on the granule cells and this can influence timing of their firing. Thus, the simultaneous

synthesising, controlling and learning from inputs from multiple sources can influence the output of the Purkinje cells, which in turn will influence the planning, timing and coordination of muscle activity.

This complex function of the cerebellum is required for many human activities including walking, which requires simultaneous control of multiple muscle groups around each joint, coordination of multiple joints within each limb and coordination of inter limb activity. Thus, it is not surprising that individuals with impaired cerebellar function find walking highly challenging, and develop less efficient, compensatory strategies, for example hyperextending their knees to increase knee joint stability. Therapy therefore needs to target the underlying postural instability rather than the compensation, in order to remediate the gait abnormality. It has been found in the upper limb that individuals with ataxia have better control of isolated joint activity, as compared to multi-joint activity (Zachowski, Thach and Bastian, 2002). Therefore physiotherapists may choose to limit the number of joints the individual has to control simultaneously, for example targeting trunk control in perched sitting, while the hip, knee and ankle joints are in a stable position. As trunk control develops, so postural control can be further challenged by progressing to standing. An additional strategy may include the provision of external aids to provide stability, for example, stabilising the ankle with splints might allow greater efficiency of hip and knee movement.

Difficulty adapting to movement error

Individuals with cerebellar lesions present with difficulties in adapting their movements to new environments or unexpected disturbances (Iig et al, 2008) for example, if the floor becomes slippery, or people are moving around them in unpredictable patterns. This can be

explained by exploring the role of the second group of fibres, called climbing fibres, which bring information to the cerebellum for processing. These climbing fibres arise from the inferior olive of the medulla (Bear, Connors and Paradiso, 2007). The nuclei within the inferior olive process information from proprioceptive, somatosensory, nociceptive, visual and vestibular systems, with each neuronal axon splitting to form on average seven climbing fibres (Sugihara, Wu and Shinoda, 2001). The pivotal work of Marr (1969) and Albus (1971) suggested that these climbing fibres produce error messages, indicating differences between the cortically intended movement, and sensory information representing the movement that occurred (Bear, Connors and Paradiso, 2007). More recently, it was discovered, that climbing fibres fire strongly when there is unexpected somatosensory, vestibular or visual information (Gibson, Horn and Pong, 2004), and the Purkinje cells then produce the error message (Carey, 2011). This type of stimulus could be externally generated, for example sensory information from a sudden change in the environmental stimuli (a perturbation), or internally generated, for example the proprioception of joints moving in an unexpected way.

This ability to respond to unexpected sensory information is important for immediate adaptation when the intended movement has been unsuccessful (Medina, 2011). It is therefore essential during gait, where motor adaptation to changing conditions is required. Consequently, individuals with cerebellar lesions will have difficulty walking in challenging or dynamic environments which require instantaneous adaptation. Therefore they may benefit from graded practice of walking in more challenging environments. More severely affected individuals may need additional support such as walking aids or physical support when there are exposed to environmental challenges, or advice to set up safe walking environments and avoidance of the challenges.

Difficulties with motor learning

The relationship between climbing fibres, parallel fibres and Purkinje cells has a role in motor learning, as well as the short term adaptation of movements. Each climbing fibre entering the cerebellum winds around one Purkinje cell, synapsing with it hundreds of times. Firing of a climbing fibre will automatically activate the Purkinje cell in a complex spike. Activation of many parallel fibres on the same Purkinje cell will also make it fire but in a simple spike (Bear, Connors and Paradiso, 2007). Simultaneous climbing fibre and parallel fibre activity causes weakening of the parallel fibre-Purkinje cell synapse due to an influx of calcium into the Purkinje cell, making it less receptive to neurotransmitters released by parallel fibres. Repeated occurrences lead to long term depression (LTD), with a weakening of that neural pathway (Broussard and Kassardijan, 2004). This is important in error based learning, as pathways controlling movements which have resulted in abnormal sensory feedback (error signal) will become weakened, and therefore less likely to fire in the future. Recent research by Carey (2011) suggests that with differing intensities of activation, climbing fibres can also induce long term potentiation (LTP), and therefore strengthening at this synapse. Medina (2011) argues that the full mechanisms of motor adaptation and learning at the Purkinje cell level are not yet fully understood. However, the importance of this level in utilising abnormal sensory information to adapt to and learn from movement is clear.

Individuals with cerebellar lesions will therefore have impairments in this ability to respond to and learn from unexpected sensory information. Given that trial and error practice is normally used in the development of motor skills (Schmidt and Lee, 2005), these impairments will have significant impacts on motor learning. Use of the medial temporal

lobe learning system that does not appear to require cerebellar input (Bear, Connors and Paradiso, 2007) may be a more successful mechanism for learning in these individuals. This form of learning, known as declarative learning (Saywell and Taylor, 2008), would involve stepwise repetition of the required movement, with verbal prompts from the therapist to ensure that individuals are consciously aware of what they are doing. However, Saywell and Taylor (2008) report that learning through this method, as opposed to through trial and error, requires higher numbers of repetitions and is more easily lost. Physiotherapists must therefore consider that goals will take longer to achieve and individuals will need to continue to practise the skills they have learnt in order to ensure they do not deteriorate.

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Difficulties consolidating movement skills

In addition to difficulties with learning skills through trial and error, individuals with cerebellar ataxia are also likely to have difficulty consolidating their skills, to allow them to be used automatically (Iig, Golia, Tider and Giese, 2007). This difficulty could be explained by examining how Purkinje cells drive plasticity in the deep cerebellar and vestibular nuclei (D'Angelo, 2011) where consolidation of motor learning is thought to occur (Wulft et al, 2009). Park et al (2010) demonstrated this through performing functional MRI (fMRI) scans of healthy individuals during motor learning tasks. They found that the cerebellar cortex (i.e. granule layer) was more active in the initial stages of learning, coinciding with a high level of cortical activity, and that the deep cerebellar nuclei became active as skills became more automatic. Furthermore, although debated in the literature, Schoch, Dimitrova, Gisewski and Timmann (2006) suggested that the inability to consolidate skills could explain why individuals with damage to deep cerebellar nuclei have worse functional outcomes (including gait) than those without this involvement.

This has significant implications in gait rehabilitation for individuals with ataxia, as the cerebellar lesion could impact their ability to consolidate motor learning in order to make gait an automatic process. Thus, high intensity practice of necessary skills is required, with increased repetitions and minimal breaks between practice sessions. In individuals with more severe damage it may be necessary for them to always rely on cortical control of gait, where they have to actively think about each step, rather than it occurring automatically. This was highlighted by Mihara et al (2007) who found greater activity in the motor cortex during steady state walking in individuals with cerebellar damage compared to healthy individuals. This suggests that physiotherapists may need to teach patients to consciously attend to their walking and the particular components that are required to achieve an optimal gait pattern. Additionally, this lack of automatic control means that individuals with ataxia will present with high variability in their motor performance, especially if they are tired, unwell or distracted. They will therefore require education and strategies to manage their daily routines, and to cope for fluctuations in their mobility.

Insert table 1 here

Table 1. The characteristics of ataxic gait, neural mechanisms and potential rehabilitation strategies

Difficulties associated with specific cerebellar lesions

Whilst the cerebellum has a uniform cytoarchitecture and method of processing information, its different lobes (figure 2) receive input from different parts of the body or cerebral cortex, and outputs affect different pathways. Lesions in specific parts of the cerebellum will

therefore have different effects on gait patterns (Iig et al, 2008). These are summarised in table 2. However, focal cerebellar damage is rare in humans because even if a specific lesion occurs, swelling, pressure and circulatory disturbances are common (Dietrichs, 2008). Therefore, although it is important that physiotherapists understand the role of the different regions of the cerebellum in order to better target treatment, it is likely that symptoms observed in clinical practice will arise from damage to more than one region.

Insert figure 2 here

Figure 2. The lobes of the cerebellum (Leach and McManus, 2012)

The spinocerebellum: poor anticipatory postural control, increased postural sway and abnormal balance responses

The spinocerebellum receives a large volume of sensory information from the body, as well as information from the motor cortex (D'Angello, 2011). Its efferent neurons pass into the brainstem nuclei and output from these nuclei influences three different lower order motor pathways: the vestibulospinal, reticulospinal and rubrospinal pathways (Porter, 1993). These pathways all have a role in the regulation of postural tone, automatic postural adjustments, and regulation of agonist-antagonist muscle activity. The spinocerebellum is therefore important in controlling coordination for automatic balance reactions and smooth movement. Individuals with damage to this part of the cerebellum present with central hypotonia, postural tremor, increased postural sway, hypermetric balance reactions and limited preparatory postural control (Morton and Bastian 2007). Clinically, this means that they

present with poor stability and reduced functional balance (Ioffe, Chernikova and Ustinova, 2008).). Additionally, damage to this area can lead to poor intra-limb coordination (Thach and Bastian, 2004).

Martin, Tan, Bragge and Bialocerkowi (2009) suggest that specific strength and balance training customised to individual difficulties can be an effective treatment intervention. Additionally, teaching individuals to prepare themselves for movement by using environmental cues, such as aligning themselves with a point on the wall may be beneficial (Saywell and Taylor, 2008). Teaching patients to increase their base of support as a compensation for their lack of stability, or slow their movements down, may also be of benefit. For those with severe damage who are unable to achieve sufficient stability in standing and stepping, walking aids may be necessary.

Spinocerebellum: Difficulties with automatic gait control

Difficulty regulating step timing is common in ataxic gait. This may be due to damage to the cerebellar locomotor region (CLR), which in cats, has been shown to be essential in controlling rhythmical gait (Armstrong 1988). The CLR acts as a pacemaker for the mesencephalic locomotor region (MLR) which in turn stimulates central pattern generators (CPGs) in the spinal cord to produce automatic stepping (Armstrong, 1998). Cats with the MLR intact and the CLR removed demonstrated reciprocal stepping but with poor timing and inequalities in step (Armstrong, 1998). Although CPG activity is thought to exist in humans (Duysens and Crommert, 1998), it is known that a higher level of cortical control over this activity is needed for the challenges of human bipedal gait (Reisman, Bastian and Morton, 2010). Therefore the research in cats cannot be directly translated to humans. However, in fMRI scans of people during mental imagery of walking and running Jahn et al (2008) found

an area of high activity within the spinocerebellum, correlating with the CLR in cats. Therefore, expecting individuals with damage to this region to gain a symmetrical, rhythmical gait pattern without cognitive attendance to it may be unrealistic. Instead concentrating on achieving safe walking, regardless of the pattern, and without a secondary task, may be more realistic.

The vestibulocerebellum: difficulty with balance and eye control

The vestibulocerebellum receives input from the vestibular system through mossy fibres and visual information through climbing fibres. Its output fibres project to the vestibular nuclei and eye muscles (Broussard and Kassardijan, 2004). It has a role in balance, as well as being important in regulating the vestibular ocular reflex (VOR), which allows gaze stability with head movement (Porter, 1993). Thus, a useful strategy for individuals with damage to the vestibulocerebellum may be to improve head stability. Alternatively, an individual who has learnt to walk through stabilising their head position but needs to progress, may benefit from being challenged with VOR retraining, for example, by focusing on an object as they are walking and their head position changes.

The cerebrocerebellum: difficulties with adapting gait

The cerebrocerebellum is part of a motor loop between the cerebral cortex and cerebellum and is important in controlling planned, voluntary movements (Bear, Connors and Paradiso, 2007). D'Angelo (2011) described the cerebellum's role in the loop as refining and modifying the motor command sent by the motor cortex, adding precision, timing and control from past movement experience. Furthermore, it can direct conscious attention to the movement when required (Thach and Bastian, 2004). Cats with lesions in this area presented

with foot placement difficulties in challenging conditions but under standard conditions, their gait appeared normal (Reisman, Bastian and Morton, 2010). The study described earlier by Iig et al (2008) found that individuals with cerebrocerebellar tumours also had difficulties in foot placement. However, these individuals scored highly on standardised gait and balance outcome measures used by physiotherapy departments, as difficulties resulting from their lesion only become evident in challenging environments, for example, stepping over objects. This suggests that therapists need to assess individuals with cerebellar lesions when walking in real life environments to gain an accurate picture of the level of disability. Therapy needs to target the factors that are challenging for the individuals using specific strategies to overcome them. For those with more severe neurological damage, living environments may need to be made obstacle-free, and the use of a walking aid or wheelchair for outdoor use may be required.

Insert table 2 here

Table 2. Symptoms of lesions in specific areas of the cerebellum and potential rehabilitation strategies

Predictors of recovery

Recovery following cerebellar damage is slow and often incomplete (Deluca et al, 2011). It has been suggested that individuals with focal lesions have a better recovery than those with diffuse lesions (Marsden and Harris, 2011). Iig, Golia, Tider and Ggiese (2007) proposed that after cerebellar damage walking is no longer automatic and, as every step would be a

conscious movement, pathways through the cerebrocerebellum would be active. Therefore, if there is damage throughout the cerebellum, individuals would not only experience the difficulties resulting from the damaged spinocerebellum, but would also have difficulties compensating due to the cerebrocerebellar damage.

Further research into how recovery of function following cerebellar lesions occurs would assist physiotherapists in their clinical reasoning regarding their treatment and management of the individual.

Conclusion

A sound understanding of how the cerebellum functions, both in health and disease, can aid physiotherapists to target their assessment, treatment, management and goal setting with individuals who have had lesions in this area of the brain. The essential role of the cerebellum in automatic gait, postural control, coordination and motor planning and adaptation explains why significant cerebellar damage can be devastating to functional walking. Appreciating the central role of the cerebellum in motor learning and in learning through error, informs our understanding of why individuals with cerebellar lesions take longer to relearn the skills of walking and require more repetition than individuals with other CNS lesions. It may also be necessary to use compensatory aids and strategies for those with more severe cerebellar damage. More research is needed to determine the efficacy of commonly used physiotherapy and rehabilitation techniques and strategies in order to ensure that individuals with cerebellar lesions achieve the best possible outcomes from their therapy.

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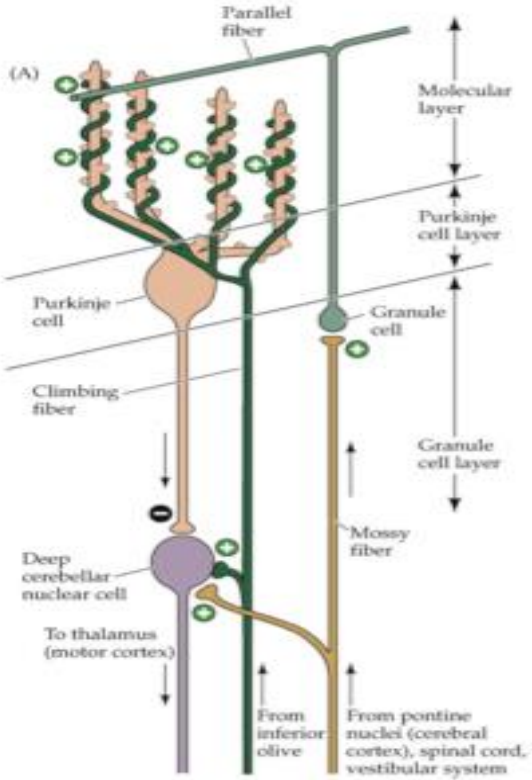

Figure 1. The cellular functioning of the cerebellum (Leach and McManus, 2012)

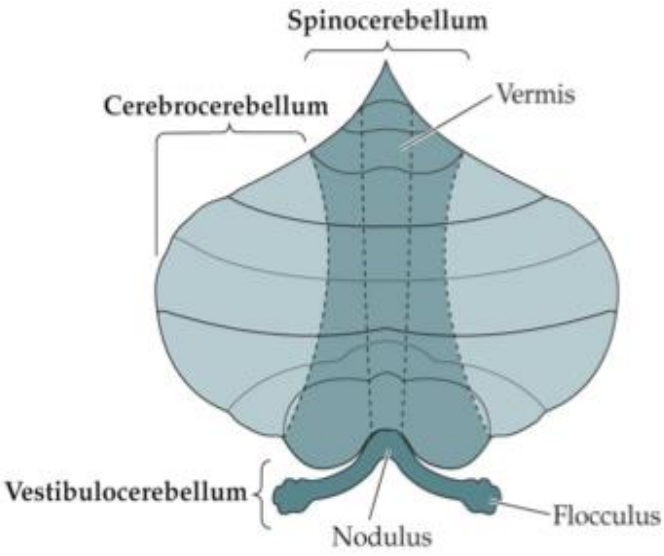

Figure 2: The lobes of the cerebellum (Leach and McManus, 2012)