Pathophysiology, Functional Implications and Management of Spasticity in Stroke – A review

Akosile, C. O.[1], Fabunmi, A.A.[2]

[1]*Department of Medical Rehabilitation, Faculty of Health Sciences and Technology, Nnamdi Azikiwe University, Awka, Nnewi Campus, Nnewi, Anambra State*

[2]*Department of Physiotherapy, College of Medicine, University of Ibadan and University College Hospital, Ibadan*

Correspondence

Dr. C.O. Akosile, Department of Medican Rehabilitation, Faculty of Health Sciences and Technology, Nnamdi Azikiwe University, Awka, Nnewi Campus, Nnewi, Anambra State, Nigeria • E-mail coakosile@yahoo.com

SUMMARY

The management of spasticity in stroke requires a multidisciplinary approach but more importantly, an understanding of the pathophysiology of its consequences. This paper reviews different definitions from neurophysiology and medical literature which try to place spasticity in stroke in its proper context and describes the current understanding of its pathophysiology and resultant functional implications. It also highlights the current medical, surgical and physical therapy available for its management. It seems spasticity in stroke is best managed using a combination of physiotherapy and Botulinum Toxin- Type A injection, as this is the current trend in research and practice.

KEY WORDS: Spasticity, stroke

INTRODUCTION

Spasticity is a major cause of disability in stroke survivors, causing pain, significant functional problems and likely to lead to complications such as muscle contracture if untreated or badly treated (Barnes, 2001). The term spasticity is used synonymously in medical and physical therapy literature to refer to both the severe hypertonus emerging immediately after a head injury and the slowly-evolving hypertonus following a more focal lesion-like stroke. It has been defined as a motor disorder characterised by velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from the hyperexcitability of the stretch reflex (Brown, 1994; Ivanhoe and Reistetter, 2004).

Bakhta (2000) opined that while this definition is useful for diagnostic purposes, it is rather restrictive in terms of understanding and managing the consequence of inappropriate muscle activity found after stroke. It could not account for the abnormal posture and mass pattern seen or the inappropriate muscle co-contraction and involuntary limb movement associated with exaggerated cutaneous reflexes or efforts (associated reactions), in addition to stretch reflex hyperexcitability which ought to be considered together for success in antispasticity treatment.

True spasticity is apparent when the relaxed spastic muscle is stretched (Rothwell, 1994) and is dependent upon afferent information from feedback following movement of the stretched muscle (Young, 1994). On the other hand, the abnormal posture which is a consequence of increased tonic muscle contraction in the absence of movement is referred to as spastic dystonia (Young, 1994). This is exemplified by the abnormal posture of upper limb flexion and lower limb extension seen in hemiplegia (Young, 1994). It is considered as a form of sustained efferent muscular hyperactivity, dependent upon continuous supraspinal drive to the alpha motor neurone (Sheean, 1998).

PATHOPHYSIOLOGY OF SPASTICITY

Neural Components

The balance interaction between parapyramidal tracts arising in the brainstem is responsible for the supraspinal control of muscle tone. The major tracts are the dorsal reticulospinal tract (DRT), the medial reticulospinal tract (MRT), and the vestibulospinal tract (VST), which are descending pathways synapsing upon the inter-neural network within the spinal cord to exert this influence (Edward, 2002). The DRT has an inhibitory influence, while the MRT and VST have a facilitatory influence on extensor tone. The three together inhibit flexor reflex afferent responsible for flexor spasm (Brown, 1994; Sheean, 1998).

The DRT, unlike the others, is under direct cortical control from the premotor and supplementary motor areas via corticoreticular neurones descending through the internal capsule (Brown, 1994). In stroke, there is damage to this cortical drive due to internal capsule lesion which reduces the activity of the DRT such that the facilitatory effects of MRT and VST become relatively unopposed (Sheean, 1998; Edward, 2002). These impaired modulation of monosynaptic input from primary afferent (Ia) fibres (segmental myotactic reflex), polysynaptic afferent input from cutaneous receptors and golgi tendon organs contribute to alpha motor neurone hyperexcitability of flexor muscles in the upper limb and extensor muscles in the lower limb.

Post-stroke patients often demonstrate an initial flaccidity or hypertonia for a variable period of time before the emergence of hypertonia and excess reflex activity. During this period of shock, the muscles may become toneless and areflexic (Edward, 2002). The delayed appearance of hypertonus is presumed to involve some functional or structural rearrangement within the central nervous system (CNS) (Edward, 2002). These are plastic changes, for which increased receptor activity and collateral sprouting have been implicated.

Nociceptive and motor pathways have also been reported to have considerable influence on each other, emphasizing the clinical importance of pain management in treating spasticity (Barnes, 2001).

Non-neural Components

Some authors have suggested that an increase in the mechanical stiffness of the muscle is responsible for spastic hypertonia (Dietz, 1992; Brown 1994; Ada et al, 1998). It is thought that the stiffness is mediated by permanent structural changes in the mechanical properties of muscle or connective tissues which may be variable in character (Katz and Rymer, 1989; Carey and Burghardt, 1993). It is important to note that this mechanical contribution to hypertonia would not arise without the damage to the central nervous system (CNS), producing the reduced activity and/or stereotypical postures associated with upper motor neurone lesions (Brown, 1994).

Disuse atrophy is another phenomenon occurring in patients with hypertonus, even in the presence of sustained muscle activity. This is due to the disruption of central and segmental synaptic drive onto spinal inter-neurones (Rothwell, 1994; Gordon and Mao, 1994). Patients who are unable to sustain muscle activity against gravity will be unable to maintain the muscle characteristics associated with postural control. Atrophy of the slow twitch fibres responsible for function then results with a consequent increase in the dominance of fast twitch fibres (Edward, 2002). In stroke and in the presence of hypertonia, there can be a gradual change in fibre-type composition with increased numbers of slow muscle fibres (Dietz, 1992), which may be the result of a muscle fibre transformation following continuous activity in hypertonus (Dattola et al, 1993). Slow-twitch fibres develop larger forces at lower firing rates and are recruited first. An increase in their proportion may thus contribute to a gradual increase in hypertonia. This selective atrophy of fast twitch muscle fibres is also assumed to contribute to a reduction in voluntary power in hemiparesis (Vrbova et al., 1995).

FUNCTIONAL IMPLICATION OF SPASTICITY

Certain pathological activities associated with hypertonus will help in discussing the functional implication of spasticity. These are:

Positive Support Reaction: This rigid extension of the leg with subsequent inability to balance with normal alignment of the trunk and pelvis (Edward, 2002) is thought to be due to a proprioceptive stimulus evoked by the stretch of the intrinsic muscle of the foot and an exteroceptive stimulus evoked by the contact of the pad of the foot with the ground (Bobath, 1990). Clinically, plantarflexor hypertonia associated with the inversion of the foot is a primary feature of this reaction. There is also a shortening of the intrinsic foot musculature due to the inability to transfer weight across the full surface of the foot and loss of range in the plantar fascia. Shortening of the tricep surae may also result due to the inability to attain a plantigrade during the stance phase of walking, which further

exacerbates the inability to transfer weight or to adapt to irregularities in ground surface (Dietz, 1992).

Consequently, the post-stroke individual tries to maintain balance by a compensatory hip flexion with a retraction of the pelvis, due to the backward displacement of weight by the pressure from the ball of the foot (Edward, 2002). The knee extension obtained with this is not due to appropriate quadriceps activity as the quadriceps may even be wasting. Hyperextension of the knee may occur due to the abnormal alignment of the pelvis over the foot, an impaired interaction between the hamstrings and quadriceps muscle groups and a shortening of the gastrocnemius (Edward, 2002). The hip flexors, adductors and medial rotators may be shortened as a result of the flexed, retracted position of the hip and pelvis, invariably producing a mechanical obstruction to correct hip alignment in the stance phase (Edward, 2002). The knee is prevented from being released by the pressure exerted by the foot pushing into plantarflexion. The extended leg is hitched forward in order to step through in moving the foot away from the floor (Dietz, 1992). This compensatory strategy coupled with associated flexion reaction of the upper limb is responsible for the typical hemiplegic gait. The described lower limb activity demands greater effort, particularly from the latissimus dorsi, resulting in the shortening of the trunk side flexors and hypertonicity of the upper limb flexors (Dvir et al, 1996).

The ability to stand up and sit down is also affected. The stiff extended leg prevents the post-stroke individual from standing, pushing him back into the chair. The inability to flex the knee also makes the attempt at sitting largely unsuccessful and the individual ends up falling into the chair. Hemiplegics tend to stand up and sit down on their sound leg due to the inability to support their weight on their paretic leg (Edward, 2002).

Complications like contracture at the ankle affects function. For example, an equinus deformity will interfere with donning footwear and the use of a wheelchair footplate.

Associated Reaction: These are thought to be pathological movements indicating a potential for hypertonus development or accentuating prevailing spastic synergy (Bobath, 1990). They are initiated with attempted movement or at the preparatory stage of movement (Dickstein et al, 1995) and may also occur with involuntary action such as yawning, coughing and sneezing or when dysphasic patients try to communicate (Edward, 2002). The appearance and severity of the associated reactions with hypertonia may be due to underlying low tone or lack of

stability of proximal key points. Sustained associated reactions may lead to decreasing functional level in that movement. For example, repeated flexion of the arm may lead to a gradual loss of range and ultimately contracture (Davies, 1990; Dvir and Panturin, 1993). The involuntary elbow flexion (accompanying the stepping through attempt at the lower limb in some patients) is thought to interfere with walking and standing balance (Bhakta, 2000).

Inappropriate co-activation of agonist and antagonist muscles: This can impede normal limb movement and function. For example, the coactivation of the triceps and biceps may affect placement function, while the co-contraction of the forearm flexor and extensor muscles may prevent voluntary finger extension and relaxation of grip (Bhakta, 2000).

Inappropriate muscular activity: This may lead to painful deformity and interference with function. For example, painful toe flexion and difficulty with walking and running may result from the inappropriate activity of the intrinsic foot muscles and long toe flexors. Extensor hallucis longus overactivity may cause involuntary big toe hyperextension and difficulty in donning footwear (Bhakta, 2000).

MEDICAL MANAGEMENT OF SPASTICITY

The available drugs for spasticity management are either systemic or focal in the nature of their administration. They are:

Oral baclofen: The most widely-used anti-spastic agent. It is a structural analogue of gamma-amino butyric acid (GABA) and binds at the Gaba B-receptor. Its recommended dosage is around 40 -100mg daily, given in divided doses due to its relatively short half-life. Significant side effects include drowsiness, fatigability and muscle weakness (even of the unaffected muscles), which may increase disability. The side effects limit the role of oral baclofen in stroke and it is not advisable as a first line anti-spasticity (Bhakta, 2000; Barnes, 2001). Headache, ataxia, insomnia and sudden withdrawal, seizures, hallucinations and psychosis are other side effects (Terrence and Fromn, 1981).

Tizanidine: A central a-2 adrenergic agonist with similar side effects (drowsiness, fatigability and muscle weakness) to baclofen, though to a lower extent (Barnes, 2001). Its effects are thought to be mediated via neurones in the locus ceruleons and inhibitory spinal inter-neorones. It appears that tizanidine also has an anti-nociceptive effect.

It is however not recommended for routine use in stroke due to limited evidence of its effectiveness (Bhakta, 2000). Monitoring of liver function is essential as it leads to incidence of abnormal liver function. (Barnes, 2001).

Dantrolen Sodium: This acts peripherally and probably suppresses the release of calcium ions from the sarcoplasmic reticulum producing a dissociation of excitation/ contraction and coupling. Side effects are similar to those of baclofen. It also causes abnormality of liver function.

Diazepam: An effective anti-spastic agent with very limited clinical implications. Anti-spastic dosages produce significant drowsiness and fatigue. Diazepam affects walking and increases the risk of cognitive dysfunction and should not be used for the routine management of spasticity in stroke (Bhakta, 2000; Barnes, 2001).

FOCAL TREATMENT

Intrathecal Baclofen Infusion: This is done via pumps and is effective in refractory lower limb spasticity where several muscle groups in both legs are affected (Bhakta, 2000). There is a need to exercise caution in its use in stroke due to the risk of weakening muscles on the normal side. Meythaler et al (1999) reported tone reduction on the affected side and muscle strength preservation on the normal side with continuous intrathecal baclofen infusion. Francisco and Boake (2003) also reported improvement in subjects' walking speed, functional mobility rating, and spasticity with 9 months of combined physical therapy and intrathecal baclofen.

Nerve Blocks: This refers to the blockade of percutaneous nerves and/ or motor points using phenol or alcohol. Phenol nerve blocks have been found effective in managing abnormal arm and leg posture in hemiparesis over 6 months. In equinovarous, deformity and inappropriate knee flexion, lasting from a few months to several years have also been reported (Petrilla and Knoploch, 1988). The risk of painful and persistent dysaesthesia following the injection of mixed motor and sensory nerves exists (Skeil and Barnes, 1994). Phenol nerve block is no longer recommended, though the risk of sensory disturbance may be reduced by phenol motor point block ((Bhakta, 2000; Skeil and Barnes, 1994). Fifty per cent alcohol has been used as an alternative to phenol but has been less effective.

Botulinum Toxin Type A (BT-A): Injection of BT-A into spastic muscles produced chemodenervation by preventing the release of acetylcholine at the neuromuscular junction. BT-A acts peripherally to reduce muscle contraction caused by the hyperexcitable alpha motor neuron pool. The relaxation period lasts about 3 months with loss of effects occurring through axonal sprouting proximal to the affected nerve terminal and the formation of neuromuscular junctions (Bhakta, 2000). The advantage of BT-A over other anti-spasticity drug treatments includes the ability to target specific muscle groups, lack of sensory disturbance, patient tolerability, and ease of administration. Muscles have to be properly chosen and BT-A doses individualised for an optimal functional outcome (Cardoso et al, 2007). A medium BT-A dosage (320 UI spread over 2-5 muscles) have been found to be both safe and effective in producing long-lasting improvement of spastic foot dysfunction in post-stroke individuals (Mancini et al, 2005). Different authors have advocated combining BT-A injection with physical therapy modalities such as exercise therapy, functional neuromuscular stimulation or robotic training for a more potent effect (Bhakta 2000; Cardoso et al, 2007; Levy et al, 2007).

SURGICAL TREATMENT

This includes procedures that interfere with the neuronal pathways and procedures that correct musculoskeletal deformity (Bhakta, 2000). Selective tibial neurotomy improves a range of active ankle dorsiflexion in patients with calf spasticity. Surgical intervention can be divided into peripheral ablative procedures such as rhizotomy or peripheral neurotomy or more central ablative procedures such as cordectomy, myelotomy, and stereotactic procedures. Other techniques include cerebellar or spinal stimulators (Smyth and Peacock, 2000; Barnes, 2001). Surgical sectioning of tendons and muscles combined with post-operative serial splintage is done for patients with persistent deformity (Achilles tendon lengthening for equines deformity at the ankle), functional lengthening of forearm finger flexors, elbow flexor release and tenodesis facilitate arm placement and grip in patients with a potential for functional voluntary movement.

PHYSIOTHERAPY TREATMENT

Physiotherapy treatments of hypertonus are based on many assumptions and beliefs which are largely unsubstantiated (Edward, 2002). The general aim however is to improve motor performance and functional ability (Bhakta, 2000). The treatments target both the neural and non-neural components of spasticity. Functionally-based therapies integrate the repetition of everyday tasks. Basically,

physiotherapy modalities attempt to normalize the spasticity as much as possible through retraining, reproducing, patterning or positioning the muscles or groups of muscles to mimic normal movements. These techniques attempt to bring the muscle to a state of normal stretch without causing the stretch reflex to react abnormally or co-contraction of agonist muscles.

Contentious issues in what should be the major therapy focus include whether weakness is apparent or real, aerobic or resisted exercises are beneficial or deleterious in spastic hemiplegics and if tone should be compromised for function or vice-versa (Bhakta, 2000). Reports from different authors have shown that muscle weakness truly exists in upper motor neurone lesions with other features like loss of dexterity, muscle atrophy and potential for contracture development (Rothwell, 1994; Gordon and Mao, 1994; Young, 1994, Edward 2002).

Facilitation techniques like the Bobath and the proprioceptive neuromuscular facilitation (PNF), electrical stimulation, and aerobic and resisted exercise trainings have been employed for tone modulation, body conditioning, muscles strengthening and enhancement of ambulation functions after stroke. Research evidence (Wang, 1994; Hesse et al, 1995; Potempa et al, 1996; Miller and Light, 1997; Sharp and Brower, 1997; Pomeroy and Tallis, 2000; Kawahira et al, 2004; Eich et al, 2004; Macko et al, 2005; Lennon et al 2006; Akosile, 2007) supports the inclusion of these modalities. Davidson and Walters (2000) have argued for the consideration of functional gains over movement quality issues, which may include tonus increase and exacerbation of associated reactions. Edward (2002) had opined that while regular mobilization, muscle stretching and serial casting may be effective for increasing and maintaining range of movement, lasting changes would come with the functional use of the affected limb. The use of orthosis is recommended for improving walk function. The effectiveness of ankle-foot orthosis in enhancing walking capacity is documented (Mojica et al, 1988; Hesse et al, 1999).

Concurrent sensory stimulation using heat and cold can help with short-term reduction in spasticity and serve as useful adjuncts to other physiotherapy treatments (Bhakta, 2000). Lycra orthosis are used for postural management of the upper limb and may provide additional deep pressure and warmth to the tissue (Gracies et al 1997 cited by Bhakta). Proper posture and limb positioning improves comfort and reduces spasticity and forms part of both the PNF and Bobath techniques. Proper positioning is important whether the patient is lying in bed or sitting in a chair or wheelchair. Support should be provided as necessary to ensure patient comfort and appropriate position and posture. Positioning should allow for the patient's maximum ability to interact with his or her environment.

CONCLUSION

The best approach in the management of spasticity in stroke seems to be a combination of BT-A injection with one or more of the available physiotherapy interventions. The focus of the management should however go beyond the reduction of muscle tone and strengthening of weak muscles to getting the patient to functionally use the limb for different activities. This in the long run may even provide the desired tone modulation and muscle strength improvement.

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