working with neurological patients. Therapists can educate their patients more effectively if they are aware of common factors that can worsen spasticity. Assisting patients in understanding these factors and how these factors can be managed can minimize the impact of spasticity. Case descriptions of physiotherapy (PT) approaches to treating spasticity will provide direct insights on PT management.

Target Population:

Physiotherapists working with the neurological population (stroke, ABI/TBI, MS, CP, SCI)

Description of Supporting Evidence:

Therapists commonly see persons with UMN lesions in their practice. Some of the common signs such as hyperexcitable reflexes and resultant increased muscle tone (spasticity) are common to all UMN lesions. Spasticity, a velocity-dependent increase in resistance to passive stretch felt by the therapist, can contribute towards impaired function and disability. Spasticity can lead to secondary structural adaptation in muscle tissue such as stiffness and contracture. The neural elements and connective tissue changes in UMN lesions result in range of motion limitations affecting both active and passive function. Activities of daily living (ADL) can be affected depending on coping strategies and appropriate interventions.

Spasticity can worsen as a result of number of intrinsic and extrinsic factors. Intrinsic triggers such as bladder and bowel, infection/inflammation, bone injuries, stress, skin problems, pregnancy and menstrual cycle, orthoses, ingrown nails, pain, and postural changes and extrinsic triggers such as ambient temperature and diurnal cycles can worsen spasticity1. Avoiding or limiting the impact of these triggers is a recommended approach to spasticity management.

In addition to managing the impact of these triggers, it is important to provide direct physiotherapy intervention to improve active function. The optimal rehabilitation approach for spasticity management includes a combination of exercise and pharmacotherapy 2. One type of pharmacotherapy is Botulinum toxin type A (BoNTA) intramuscular injections that induce partial and reversible chemodenervation of the involved muscles. BoNTA is a safe and effective treatment for localised spasticity in single or multiple groups of muscles with spasticity. BoNTA works by blocking synaptic transmission at cholinergic terminals of the neuromuscular junctions in the injected muscles without impacting other muscle groups. The local and muscle specific effects of BoNTA are seen within one week of injection and peak effect is seen between 3 to 6 weeks.

BoNTA can directly improve passive mobility, spasms, and pain; however its effect on active limb function depends on how the patient utilizes the available range of motion into ADLs and how therapists can assist them in incorporating the newly available range of motion in active and passive exercise. Thus, BoNTA injections can provide a window of improved mobility where appropriately directed PT interventions can maximize functional gains. BoNTA guidelines advocate exercise and training aimed at targeting specific patient-identified functional goals. However, guidelines remain topical and lack detail regarding the recommendations for timing of exercises, length and duration of interventions to assist physiotherapists in making specific treatment plans to complement BoNTA injections. Spasticity can be seen in both upper and lower limbs and our clinical observations show that spasticity is frequently seen in lower limb muscles such as ankle plantar flexors and toe flexors. As a preliminary step in the direction of developing an understanding of the nature, timing, and duration of physical interventions to complement BoNTA injections, we recently published a case study3 to describe a physiotherapy intervention in a chronic stroke patient following BoNTA injections. We will also present numerous case examples of various UMN lesions from our practice to develop an understanding of the type of PT interventions that maximize functional gains.

Description of Session Format:

Lecture with a 10-15 minute question answer session in the end.

Conclusions and Implications:

As spasticity can have a significant effect on physiotherapy assessment and management of patients with neurological disorders, PTs need to be aware how to assess spasticity and also of the various treatment options. Significant improvements in ADLs, balance, and mobility can be seen with targeted physiotherapy interventions following botulinum toxin injections for spasticity.

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Keywords: Physical therapy, spasticity, neurological rehabilitation, botulinum toxin type-A

P018: Diaphragm Paralysis – it is Not Always Permanent. The Role for Inspiratory Muscle Training

Judy L. Kay, BScPT, Clinical Specialist, Cardiorespiratory

Eastern Health, St. John's, NL

Correspondence: judykay@nl.rogers.com

Learning Objectives and Session Content:

Phrenic nerve injury occurs due to a variety of causal agents, across a wide spectrum of patient populations. The associated diaphragm paralysis may be temporary or permanent. The degree of impairment experienced by individuals can range from a mild sensation of shortness of breath, which primarily occurs when the diaphragm is challenged, to severe dyspnea. Unfortunately for many patients, they are told there is nothing that can be done for their problem and that they need to learn to live with it. By the end of this session, participants will be able to 1. Identify the range of causes of diaphragm paralysis, 2. Complete a physiotherapy specific assessment of diaphragm function and, 3. Prescribe an inspiratory muscle training program that can be completed in either an in or outpatient setting. For this presentation, the case examples involve an ambulatory patient population.

Relevance to the Physiotherapy Profession:

Effective ventilation is essential to daily function. Unfortunately, there are many different causal agents and circumstances that damage or interfere with the phrenic nerve along its course.

From its origin at the C3 to C5 spinal level to its insertion across the diaphragm surface, the phrenic nerve exposure heightens its potential for interference. As a result, respiratory impairment may co-exist with a mechanical injury of the neck, a trunk insult from blunt force trauma in a motor vehicle accident or a local or systemic inflammatory process, to name a few examples. Patients may not achieve their maximum functional recovery without optimizing the recruitment and strength of the muscles of inspiration in general, and the diaphragm in particular.

Target Population:

The information and intervention reviewed are relevant to physiotherapists working in a wide variety of settings, including inpatient, ambulatory care and rehabilitation units. The case examples are from an adult population but are applicable for physiotherapists working with older pediatric patients.

Description of Supporting Evidence:

Diaphragm paralysis has been classified into five major categories: traumatic, compression related, inflammatory, neuropathic, and idiopathic.[1] Evidence indicates that spontaneous recovery of some diaphragm function can occur in 43% of patients after 12 months and 52% of patients after 24 months.[2] The gold standard for diagnosing diaphragm dysfunction is a sniff test in fluoroscopy, to demonstrate paradoxical diaphragm movement. Different parameters have been used to measure recovery. While some define a functional respiratory recovery as an increase in forced vital capacity greater than 400 ml, a measurement of maximum inspiratory pressure (MIP) has more commonly been used to establish a baseline and to track outcomes of strength training. Inspiratory muscle training (IMT) has been shown to improve ventilatory capacity in many neurological and non-neurological disorders with impaired inspiratory muscle function.[3]Threshold muscle trainers are oral devises that are calibrated and able to be set prescriptively to challenge and strengthen the muscles of inspiration. Directed effort enables increased isolation of the diaphragm. When a prime muscle for a task is impaired, the brain tends to learn and recall command programs that result in muscle coordination patterns generated by lower sensorimotor circuitry. It uses whatever muscle groups that will compensate for the lost input; use muscles that are functionally "good-enough", not necessarily those that are optimal.[4,5] Retraining of the diaphragm can occur with attention to first gaining motor control with appropriate sequence of mechanics, while eliminating the extraneous activity. Then effective strengthening can be achieved.

Description of Session Format:

The session will involve a variety of media from didactic to patient videos and case studies. A workshop-type approach will be used for a portion of the presentation as demonstration and audience participation are included to assist knowledge translation and skill acquisition.

Conclusions and Implications:

Diaphragm paralysis can occur in a variety of settings from surgical and medical respiratory situations to rehabilitation and outpatient populations. Depending on the causal agent or circumstances this may be a temporary or permanent state. When this primary muscle of inspiration becomes impaired, other muscles increase activation to accommodate for the loss. When there is recovery of phrenic nerve function, the diaphragm recruitment doesn't automatically dominate again. It requires deliberate, directed activation to gain motor control then strength. Even with a permanent hemi diaphragm paralysis, the remaining diaphragm fibers may be trained to increase ventilation capacity. Often diaphragm dysfunction has gone either undiagnosed, or if confirmed on a single sniff test, has been untreated with the presumption no change can occur. As noted, if there has been incomplete injury, phrenic nerve recovery, may evolve over a 2 year period. Targeted, progressive muscle training, of the diaphragm in particular, can be prescribed effectively for a wide cross section of patients. The patients discussed in this presentation were referred in an outpatient setting. They had been living with varying levels of dyspnea and limited function due to differing degrees of diaphragm dysfunction. IMT is a very important adjunct to the overall management of patients with diaphragm dysfunction in a variety of contexts. Physiotherapists have the relevant knowledge base and technical skill sets to prescribe IMT and achieve change where none was thought possible.

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Keywords: Diaphragm paralysis, phrenic nerve injury, shortness of breath, inspiratory muscle training

P020: Advances in Compression Bandaging for Chronic Edema/Lymphedema

Martina Reddick, RN

Private consultant, Tors Cove, NL

Correspondence: martinareddick@hotmail.com

Learning Objectives and Session Content:

Lymphedema is a progressive chronic condition requiring life-long management. Although the incidence is unknown, it affects a significant number of Canadians. There are many causes of chronic edema/lymphedema however, the most common cause in the western world, for the development of lymphedema is believed to be a consequence of cancer surgery and/or radiation treatment. Other causes include filariasis, venous disease, renal disease and trauma. Management of this chronic condition requires a multidisciplinary approach. Physiotherapists and other health care professionals have a responsibility to identify those at risk and recognize the early signs as early intervention leads to improved outcomes.

Learning Objectives and Session Content

This workshop will highlight advances in lymphedema management in Canada. Best practice emphasizes the essential role of physiotherapists, occupational therapists physicians, and nurses in caring for individuals coping with chronic conditions.