Introduction

Today with the specific techniques that a conservative care and manual therapy doctor use, the MUA procedure is gaining ground as one of the more effective procedures for both chronic and certain acute cases of pain from spinal dysfunction where manipulation/conservative care procedures are the treatment of choice but when the patient is minimally responsive to office treatment.

What makes MUA different is the use of Monitored Anesthesia Care (MAC). MAC places the patient in a position to be responsive but less apprehensive upon stretching and deep muscle massage designed to alter fibrotic adhesions. When the adhesions in and around the joints, discs and muscles are altered, a significant improvement in range of motion occurs with a reduction in pain.

MUA is not new or experimental and fits perfectly in today's multi-specialty approaches to care found within the rehabilitation community. MUA has been recorded as far back as 1938 and there is a significant amount of literature supporting MUA as a safe and effective procedure in the treatment of both acute and chronic spinal pain disorders in appropriately selected patients. In short, the patient is selected or qualified for the procedure using standards and protocols established by the American Association of Manipulation Under Anesthesia Providers (AAMUAP).

On the day of the procedure, the patient is given another history and physical by the medical clearance and the OR nursing staff and anesthesiologist. The anesthesia is given by a board certified anesthesiologist only when the patient and MUA certified doctor are ready to begin. The procedure is, in most instances completed in multiple days or what is called serial MUA with 2-3 procedures being the average. A little more movement, range of motion and stretching is accomplished each day to complete the desired effect. Once the procedures are completed, the patient is discharged to post MUA care designed to continue to redefine the mechanical alterations which have taken place and to rehabilitate the weakened areas which were altered by dis-use from injury.

The Scientific Basis for MUA

From a manual therapy perspective, the vertebral motion unit is considered to be from the middle of one vertebrae to the middle of the next to include the vertebral bodies, the disc, facet joints, ligaments, nerve roots and vascular supply. Because of the complexities of the motion unit, many "discogenic" problems are in fact myofascial/facet syndromes (of note: this fact is often considered a major contributor to the 20-40% incidence of failed back surgeries). It is the medial branch of the dorsal primary ramus that is of paramount clinical relevance due to its distribution to the zygapophyseal joints. The medial branches of the dorsal rami supply the zygapophyseal joints above and below its course. Any structure that has a nerve supply is a potential source of pain. The structures in the lumbar spine that receive a nerve supply include the zygapophyseal joints, the ligaments of the posterior holding elements, the paravertebral muscles, the dura mater, the anterior and posterior longitudinal ligaments and the intervertebral discs are potential sources of pain.

It is well known from a biomechanical perspective that to achieve reduction in symptoms and decrease in pain, manipulation/adjustment can be used to recruit the neurological mechanism of collateral inhibition. Collateral inhibition is that part of the arthrokinetic reflex that inhibits the central

transmission of pain through mechanoreceptor collateral fibers, which inhibits the nociceptors in the posterior motor units of the spine and the zygapophuseal capsules. Spinal manipulation is designed to restore biomechanical integrity to areas of articular dyskinesia due to pathomechanical factors, including loss of joint mobility, fibroblastic proliferative changes of the supporting holding elements and soft tissue structures resulting in decreased or lost flexibility/viscoelasticity and the resulting neurological and/or vascular changes.

It has now been well documented by the use of MUA, that this complex mechanism of painful stimuli from mechanical dysfunction of the motion unit frequently mimics discogenic pain especially in failed back surgery where surgical intervention (usually minimally invasive) has left the patient with the same type of pain they had prior to the surgery. When the MUA is performed subsequently, the pain improves considerably.

The Inflammatory Cycle

The facet joints are a primary site for adhesion accumulation. The core of the MUA program is altering fibrotic adhesions caused by inadequate remodeling at the end of the inflammatory cycle. The inflammatory cycle is composed of three phases:

- The initial hypoxia phase where fluids rush in bringing oxygen and swelling.
- The edema and hemorrhage phase when necrotic tissue is being flushed.
- The repair and remodeling phase.

Phase I (the acute inflammatory phase) lasts up to 72 hours and is characterized by a humeral response (coagulation, fibrinolysis, kinin, compliment, phagocytosis and chemotaxis), and cellular response (mast cell degranulation, and prostaglandin release).

Phase II (repair phase) lasts from 48 hours to six weeks and is characterized by synthesis and deposition of collagen. Collagen will contract between three weeks and fourteen weeks post injury but may take up to six months.

Phase III (remodeling phase) lasts from three weeks to twelve months or more. It is during this period in which collagen is remodeled to increase its functional capacity. Maximum strength of collagen is dependent upon forces exerted on it during this phase (stretching during the MUA procedure has a direct effect on collagen remodeling).

Role of Adhesions

Adhesions form in the joints, joint capsules, muscle fibers and all areas of stress. These adhesions cause decreased movement from articular fixation and shortening of muscle fiber length from disuse. It is also important to note that it has been shown in the literature that intradural adhesions have been shown at the microscopic level to disrupt neurovascular bundles containing branches of the sinuvertebral nerve where they course between the adherent dura and the posterior longitudinal ligament. All of these are mechanisms and sources of "discogenic" type of pain and has been shown to reduce the patient's pain when addressed. From an MUA perspective, it has been shown that the stretching that is accomplished

during the procedure has an altering effect on the adhesions in all of these structures and is the major reason why the patient has reduced pain post MUA.

One additional mechanism of pain and fibrotic adhesion formation that must be mentioned here is that caused from muscle splinting or protective rigidity. Usually after an injury, there is a protective muscular rigidity that occurs to prevent further damage and pain. This can usually be overcome with conservative care. This muscle contraction is defined as a shortening of the muscles or other tissues that cross a joint and restrict normal movement. When a muscle becomes chronically fatigued it will remain in a contracted state and develop adhesions within the tissue. It will be weak but appear strong. One type of contracture we deal with is myostatic where there is no specific pathology but is caused by disuse from lack of exercise or injury. Then there is scar tissue formation post surgery, from repetitive stress injuries and from repetitive overexertion injuries. These fibrotic adhesions dramatically reduce range of motion, cause fixation and reduce function by shortening the normal muscle fiber length.

Connective Tissue

There are three types of connective tissue we concern ourselves with in MUA.

• Organized: Tendons, ligaments, etc.

• Dense: Fascialplanes, capsules, adhesions

• Areolar: loose connective tissue between muscle fibers

If motion is maintained during the healing of trauma, connective tissue of the areolar type develops during remodeling. If the injured area is immobilized, dense contracted fibro-adhesions develop during remodeling. Immobilization of an injured area develops dense fibrous tissue 2 weeks after injury.

It is easier to prevent tightness and restriction from fibro-adhesion formation by frequent repeated activity, then to correct it after it had developed. Gross evidence indicates that fibro-adhesions begin to develop in less than 24 hours after micro/macro trauma with restriction beginning in less than 3 days.

Response to Stretching a Relaxed Muscle

Since 70% of the MUA technique involves stretching relaxed muscles, we need to understand the reactions and response to these stretches. Stretching the muscles, joints and joint capsules creates the biophysiological atmosphere for change to occur when static flexibility becomes dynamic flexibility. During stretching, we overcome definable barriers of elasticity which determine the boundaries which a muscle fiber has anatomically been limited to. Our primary interest is in creating a remodeling effect in muscles which are shortened from disuse, or from adhesion accumulation or both. Adhesions form in muscles fibers and cause joint restriction. If fixation from joint adhesion causes restriction, adhesions in muscle occurs as well.

Muscle Anatomy and the Physiology of Stretch

The myofibril contains the contractile unit within the micro-myofibers of the striated muscle called the sarcomere. It is within the sarcomere Z band to Z band that the myofilaments Actin and Myosin contract. It is the body's ability to elongate the muscles fiber at the myofilament level that makes it possible to accomplish the return to normal flexibility that occurs during the MUA procedure. A sarcomere may shorten to 50% of its normal length during contraction and 120% of its length during maximal stretching. When contraction occurs, there is change in the permeability of the myofilament due to stretch reflex and neurological stimulus. Myosin attaches to Actin and there is movement inward

or outward to perform the desired function. This Myosin-Actin sliding action is accomplished by a cross bridging chemical attraction. We believe that when the adhesions form within the muscles fibers, this normal sliding effect does not occur or is restricted. **Stretching creates a natural homeostasis and alters the adhesion** (Straight Linear Force Stretching).

By using twilight sedation, the patient is placed in a relaxed physiological state that allows for the stretching of the myofibrils Actin and Myosin that leads to the elongation of the muscle. The stretch is performed to move the muscles for the "disuse" elastic phase, into the "disuse" plastic deformans phase. The normal muscle has an "elastic" range that is part of the normal muscle length when performing a function. With an injured area that has had disuse, the normal elastic range is greatly reduced and the muscle is shortened. Straight linear force produces the desired stretch into plastic deformans and is permanent. Stretching is done with constant linear force to elongate the muscle and to allow relaxation from the Golgi tendon response to ballistic stretching. To complete the elongation of the muscle takes several days and is the most important reason for post MUA Therapy.

Neurology and Anesthesia for MUA

Ultra short acting anesthetics such as Brevitol and Diprivan (Propofol) act on the CNS to depress the reaction time to contract a muscle and/or respond to perceived pain. The most recent studies show that these anesthetics effect the reticular formation which act on the Gamma motor neurons. Gamma motor neurons innervate the contractile portions of the muscle spindle. It is thought that by depressing the response of internuncial neurons within the spinal cord, the normal transmission to alpha motor neurons is slowed which has a direct effect on the muscle activity during secondary contraction from pain. It is recognized that when the pathology of pain is effecting the parpaspinal muscles there will be a reflex reaction of muscle spasm to protect the area from further damage via stimulation from alpha motorneurons in the anterior horn. Pain perception comes from abnormal mechanical response to stimulation of the type I, II & IV mechanoreceptors. By temporarily depressing this activity within the spinal cord via administration of sedative/hypnotics, one neural pathway for facilitation of paraspinal muscle spasm and pain is removed. Propofol and versed are true sedative/hypnotics. The patient perceives pain, but ligamentus and articular function remain intact but the patient does not remember.

Multi-Specialty Approach to Chronic Spinal Pain

The prospective MUA patient is one who has undergone complete work ups including imaging studies and examination for their spinal pain including but not limited to orthopedics, neurology, pain management, physical therapy and chiropractic. These patients have often been through multiple modalities of treatment seeking to relieve their pain and restore normal function from pain medication/injections, physical therapy, spinal manipulation, massage, acupuncture etc. In this small population of patients that are non or minimally responsive to standard conservative care, MUA becomes the treatment of choice with a high degree of success in these patients.

Providing this type of care where manual therapy can be performed under the care of a board certified anesthesiologist allows the manual therapy care doctor to engage and impact restrictive scar tissue that simply cannot be done in the office. This is the striking difference that MUA can offer to patients with chronic pain and what makes the difference where other treatments have had limited success. When performed in a controlled environment with certified MUA practitioners, MUA is a safe, effective and a

highly documented therapy for well selected candidates whose condition is known to be appropriately treated with manual therapy.

Here at Klass Chiropractic, P.C., we follow all standards and protocols in patient selection and care as deemed by the AAMUAP. If you have any questions regarding the material you have just read, you can contact me personally at 516.330.8810. If you would like to explore MUA further you can get more information about Dr. Gordon and the Gordon MUA Technique at http://www.gordonmua.com/.

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