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Swimmers, in the study by Obayashi, Urabe, Yamanaka, and Okuma (2012), may be prone to hyperkyphosis resulting from the kinds of force generation required by their sport. Professional masters 40 and masters 30 cyclists exhibited a high frequency of thoracic hyperkyphosis in the neutral standing position as compared to sedentary individuals (Muyor, López-Miñarro, Casimiro, & Alacid, 2012). Paddlers also displayed increased thoracic kyphosis as compared to sedentary in the sit-and-reach test (López-Miñarro, Muyor, & Alacid, 2011). Climbers were found to be prone to thoracic hyperkyphosis and lumbar lordosis with a strong correlation between climbing ability/mastery and postural adapations (López-Miñarro et al., 2011).

Generally, greater thoracic kyphosis, upper cross syndrome, and flexed posture or FP (de Groot et al., 2014) have been found in participants of sports that require a bending, trunk flexion dominant posture (López-Miñarro et al., 2011). Compensations of the pelvic tilt (too much anterior or posterior tilt) were found in some cases which is expected due to the body as a kinetic chain. Much of modern western society seems to be desk-bound and spending the majority of the time in spinal flexion. One does not have to be an athlete to face the same sagittal spinal maladaptations.

Thoracic hyperkyphosis and a posterior pelvic tilt may increase spinal load, pressure on intervertebral discs, creep (ligamentous crepitus), and low back pain (Muyor et al., 2012). Greater thoracic kyphosis in younger athletes (not yet reaching full development or maturity) might be due to excessive loading forces in their sport which may in turn damage intervertebral discs (reducing disc height) leading to a reduction in the length of the anterior spinal column (i.e. a "collapsing/caving" in on the anterior side of the spinal column) (López-Miñarro et al., 2011). Excessive kyphosis or lordosis (curving one way or the other past the normal range) affect intervertebral disc pressures, and their compressive/shear type forces (López-Miñarro et al., 2011). Also as a result of FP and thoracic hyperkyphosis, the cervical spine would exhibit more lordosis as the head is jutting out or cranked forward (increased cervical extension).

Hyperkyphosis inhibits breathing as the available anterior or "chest" space is reduced--rib cage position is such that ribs are not allowed their natural motion,and zone of apposition is reduced. The zone of apposition is significant because in normal respiration, it makes up one quarter to one third of the total surface area of the rib cage (Coughlin, Hruska, & Masek, 2005). Thoracic hyperkyphosis and flexed posture in general is also marked by a forward-tilted and protracted scapula which over time may cause impingement due to the reduced subacromial space and restricted shoulder ROM (López-Miñarro et al., 2011). Pelvic tilt compensations (too much anterior or posterior) in response to abberations in the kinetic chain due to the demands of activities (or sedentary "sitting" lifestyle) can affect all the muscles involved in hip flexion/extension (e.g. tight/shortened hip flexors, weak hip extensors such as gluteus maximus).

The aforementioned effects of poor posture, specifically flexed posture (FP), upper cross syndrome, and thoracic hyperkyphosis are just a few examples. Because the body is a kinetic chain, dysfunction in one area can affect everything above, below, and surrounding areas.

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Possible tight/shortened muscles in FP, upper cross syndrome, and thoracic hyperkyphosis are: upper trapezius, levator scapulae, sternocleidomastoid, scalenes, latissimus dorsi, teres major, subscapularis, pectoralis major/minor (Clark, Lucett, & Sutton, 2014).

Possible lengthened muscles in FP, upper cross syndrome, and thoracic hyperkyphosis are: deep cervical flexors, serratus anterior, rhomboids, mid-trapezius, lower trapezius, teres minor, infraspinatus (Clark et al., 2014).

To combat the risk of pathalogical thoracic hyperkyphosis, FP, and the aforementioned compensations, activities/exercises that place the body in extension or emphasize spinal extension/lengthening would be complementary to the flexion-dominant activities/lifestyle. Some muscle actions that oppose the FP and related pathlogies are spinal extension (iliocostalis, longissimus, spinalis-thoracis division, transversospinalis, multifidus), retraction of the scapula (rhomboids, middle trapezius), and scapular depression/downward rotation (rhomboids, lower trapezius), hip extension (gluteus maximus and piriformis), and the abdominals for stabilization and supporting respiratory muscles (rectus abdominis, external oblique, internal oblique, transverse abdominis (Clark et al., 2014; López-Miñarro et al., 2011). The quadratus lumborum is important for lateral stability. Stretching hip flexors (adductor longus, adductor magnus anterior fibers, adductor brevis, gracilis, pectineus, gluteus minimus, tensor fascia latae, psoas, sartorius) to combat the daily "sitting" would also be helpful.

The "how" these tissues should be targeted depends on the individual's issues. I am holding off mentioning exercises, because we were told to discuss that in Module 3. Each person's compensations are different and it is difficult to say whether the tight/shortened muscles are the culprit, or is it that the lengthened muscles are weakened (thus portraying the shortened muscles as "overactive") or are the stabilizers just "weak" or are they just not reactive enough to stabilize a situation (you don't want them to always be "strong" as much as being able to reactively stabilize when called for). When looking at a person, one is seeing (with the naked eye) poor/compensatory movement patterns or asymmetrical movement patterns. Without further testing and diagnostics, one cannot "pinpoint" the exact muscle although one may guess on a muscle "group" that is known to display the observed compensations.

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