

[IMAGE]

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Is a Paraspinal Compartment Syndrome Possible?

By Warren Hammer, MS, DC, DABCO

When we think of a compartment syndrome, most of us think of a runner with severe anterior compartment leg pain after running some distance. If the case is severe enough, a fasciotomy is performed to reduce the pressure on the muscle due to compromised circulation.

A compartment syndrome has been defined as "a condition in which increased pressure within a limited space compromises the circulation and function of the tissues within that space."¹ In a compartment syndrome, there is an enclosed noncompliant envelope (fascia) that raises the compartment pressure to a degree sufficient to reduce the arteriovenous circulation, resulting in an ischemic sequelae.²

Peck et al.,³ based on the anatomy of the posterior erector spinae muscles and their fascial compartments, feel that lower back pain may occur for reasons similar to anterior tibial compartment syndrome. They injected saline into the cervical, thoracic, lumbar and sacral regions of the erector spinae muscles in six fresh cadavers (less than five hours postmortem). In all cases, there was an increase in pressure due to the increased volume. In all cases, a fasciotomy resulted in an immediate decrease in pressure.

In two cases, large amounts of saline were rapidly injected into the cervical region. In both cases, a marked reduction of the cervical lordosis occurred, and the sternocleidomastoid muscles (SCM) became stiff and "board-like" to palpation. After an hour, the cervical lordosis gradually reappeared and the SCM became soft to palpation.

One of the cardinal features of compartment syndrome (anterior tibial syndrome) is a woody hardness or palpable rigidity in which the muscles lose the ability to contract. Therefore, palpable rigidity is not necessarily a muscle spasm. Since a spasm of the erector spinae should extend the spinal column, it seems odd that a spasm would flex the lumbar spine and eliminate the lumbar lordosis.

The erector spinae muscles are bilaterally paired, curved tubes, enclosed within fascial envelopes lying parallel to the spine. A compartment syndrome results from increased intracompartmental pressures, producing a "Bourdon tube effect": straightening of a curved tube by increasing intratubal pressure (the

palpable muscle rigidity straightening the spine and lessening the lordosis).³

Carr et al.⁴ gave a case report in which they diagnosed a patient as having a paraspinal compartment syndrome.⁴ The clinical signs of the patient were muscle tenderness and tenseness; inability to actively contract the muscles without severe pain; and pain on passive muscle stretch. Their anatomic study proved the presence of a closed paraspinal muscle space enveloped by fascia and osseous elements. They concluded that a subset of patients have a chronic (and occasionally an acute) compartment syndrome.

Konno et al.,⁵ listed their criteria for diagnosing chronic compartment syndrome of the lumbar back muscles:

1. no symptoms at rest;
2. normal range of motion;
3. exercise-related low back pain;
4. improvement by extension; 5. no neurologic deficits in the lower extremities.

They stated that this diagnosis should be considered and specifically ruled out when elderly patients complain of exercise-related low back pain. They believe that measuring intramuscular pressure provides an objective guideline for assessing the contribution of muscle pain to low back pain. They agree with Peck³ when they state that as spinal alignment changes from lordosis to kyphosis, the intramuscular pressure increases and blood flow decreases.

Konno et al.,⁵ also felt that the intramuscular pressure in lumbar muscles depended primarily on spinal alignment, i.e., flexion to extension. They postulate that even a local change in spinal alignment, as occurs with compression fracture or degenerative spondylolisthesis, will increase intramuscular pressure and decrease blood flow.

Increased erector spinae pressure has also been demonstrated in vivo in normal and spinal patients in the flexed position while standing and with loading.⁵ The pressure was significantly higher in patients with osteoporosis, degenerative spondylolisthesis and previous lumbar spine surgery than in the control subjects in flexed positions while standing and with loading.

As the reader of this column is aware, I have been treating the posterior fascial layers of numerous back patients. I believe that there may be different levels of compartment-like patients who will benefit from

fascial release techniques. I also believe that restricted fascia besides the vascular effect adversely affects the load transfer from the lower extremity through the pelvis to the upper extremity. There are numerous local fascial restrictions in the back that are related to the etiology of spinal pain.

Recent findings⁶ that have finally connected the posterior fascial layers from the sacrum to the cervical spinal areas, especially with regards to the transmission of tension, have opened up a whole area of discussion, not only on the cause of spinal pain but even the validity of orthopedic testing, which has blamed the nervous system instead of tension produced by the fascia. This topic will be discussed in a future article.

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[IMAGE]

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