SCIENTIFIC ARTICLE

Quantitative Assessment of Scalene Muscle Block for the Diagnosis of Suspected Thoracic Outlet Syndrome

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Purpose To measure changes in upper limb work and power capacity before and after anterior scalene muscle block (ASMB) to suggest thoracic outlet syndrome caused by costoclavicular space compression.

Methods We evaluated 34 patients disabled by symptoms suggesting thoracic outlet syndrome. An ASMB was performed via a supraclavicular injection. The sternocleidomastoid muscle was injected as a control. We captured data obtained from work simulator measurements before and after ASMB. Each patient performed a push−pull test with the forearm at waist level (test 1), an overhead bar push−pull test with the arm elevated (test 2), and the extremity abduction stress test with repetitive hand gripping during static arm elevation (test 3). We measured the work product, time to fatigue, and power generation. Sensory testing was performed after ASMB to rule out improved performance associated with possible sensory nerve block.

Results In contrast to sternocleidomastoid injection controls, symptomatic and functional improvement was noted in all patients (n = 34) after ASMB. Work product measurement improved 93%, 108%, and 104% for tests 1, 2, and 3, respectively. Time to fatigue and power output also increased after the block.

Conclusions Temporary symptomatic improvement after ASMB may be anticipated in patients with TOS. This study documents a significant concurrent increase in upper limb motor function after the block. Increased work and power measurements after ASMB may draw diagnostic inference regarding a dynamic change in the scalene muscle and the costoclavicular space associated with symptomatic thoracic outlet syndrome. *(J Hand Surg Am. 2015;40(11):2255−2261.*

Type of study/level of evidence Diagnostic III.

Key words Thoracic outlet syndrome, diagnosis, scalene muscle block, BTE work simulator.

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THORACIC OUTLET SYNDROME (TOS) results from compression of the subclavian vessels and/or brachial plexus between the clavicle, scalene muscles, and the first rib. The condition is associated with fatigue, pain, and paresthesias in the upper extremity.[1−7] Occasionally, patients report severe headache or chest pain simulating angina associated with cardiac muscle ischemia.[8,9]

Confounding symptoms associated with neurovascular compression may be dynamic and associated

with provocative activity involving postural positioning of the upper limb. Symptoms in patients presenting with neck and chest pain, radiation of pain or numbness into the medial aspect of the upper limb, pulse diminution or pallor associated with arm elevation, and overhead work intolerance suggest TOS.[10] However, these problems may not provide anatomic localization of a specific area of pathology. The symptoms may be transient and difficult to localize using standard imaging techniques.[10,11]

There is no pathognomonic sign or single probative diagnostic laboratory study associated with TOS. Incremental evidence may be obtained from a history of neck trauma, provocative arm posturing or overhead arm use, stressful athletic activity, or the presence of anatomic variations such as cervical ribs.[12] Electrodiagnostic and imaging studies may assist with diagnostic localization.

Temporary relief of symptoms was reported after anterior scalene muscle block (ASMB) by Gage[13] in 1939. Permanent improvement followed scalenectomy. Contemporary authors have injected local anesthetic or paralytic agents into the anterior scalene muscle to induce temporary paralysis.[4,7,10,11,14–16] A weakened scalene muscle loses its ability to elevate the first rib. This may result in more space for the neurovascular elements in the costoclavicular space.

Sanders and Haug[4] and Sanders et al[10] provided a comprehensive review of their experience with ASMB in ascertaining localization of pathology to the scalene muscles and the costoclavicular space. They reported successful postoperative relief of TOS symptoms in patients who responded favorably to the block.

Several authors[4,7,10,13,15] have discussed the diagnostic implications associated with subjective improvement after ASMB.

The purpose of this study was to provide quantifiable physical performance data that would augment and clarify reported symptom changes that may occur after ASMB injection. A data-driven approach may assist with medical treatment decision making. Response to ASMB, associated with quantitative data analysis, may provide an important nexus between disabling symptoms and an anatomic localization of pathology in the costoclavicular space.

MATERIALS AND METHODS

Clinical presentation and diagnosis

We evaluated 34 symptomatic patients (26 women and 8 men; average age, 46 years; range, 20–61 years) over a 3-year period for neck, chest, and upper limb problems. Referrals to this office were requested by community surgeons and neurologists who thought that further study regarding a possible diagnosis of TOS was required. All patients in this series were in good general health but had experienced disabling symptoms for over 1 year. Pain and loss of arm control forced them to discontinue physical work requirements and participation in sports or recreational activities that required throwing or any strenuous use of the upper limb. Factors of disability included changes initiated to avoid increasing pain that would prevent the patient from continuing activities of daily living such as arm elevation required for washing, hair care, dressing, placing objects on high shelves, carrying packages or driving a car, or sitting with the arms in a forward and downward position when using a keyboard.

A focused physical examination determined entry criteria in this series of patients. All 34 patients had arm pain or numbness with provocative positioning,[17] 32 had brachial plexus tenderness, 8 had a positive Wright test,[2] and all 34 had a positive Roos test.[11] Four patients were excluded from this study because our initial evaluation suggested that their primary diagnosis involved cervical degenerative disc disease or shoulder tendinitis.

Electrodiagnostic studies were routinely obtained with the hope of defining diagnostic specificity.[10,18,19] There were no abnormalities associated with ulnar neuropathy in this group. Three patients showed abnormal conduction in the medial antebrachial cutaneous nerve, which suggested a medial brachial plexus cord injury.[20] None of this group had measured clinical muscle atrophy or localizing sensory abnormality.

Disabling symptoms noted in this group of patients included supraclavicular tenderness, arm pain, fatigue with demand for repetitive arm or hand use, pain or weakness with arm elevation, episodes of hand numbness, headache, evidence of impaired circulation and hand blanching with arm elevation, and cyanotic discoloration with depression of the symptomatic limb and shoulder girdle. We used orthopedic evaluation, imaging studies, and electrodiagnostic testing to rule out cervical disc disease, shoulder tendinitis, ulnar neuropathy, and similar neurological conditions. We thought that a presumptive diagnosis of TOS would be strengthened by objective data associated with ASMB.

Study design

An ASMB involves insertion of a 25-gauge needle along the lateral border of the sternomastoid muscle about 2 fingerbreadths above the clavicle,[7,10,14,15] which was approved by our institutional review board. A unilateral ASMB using 3 mL 1% lidocaine was