Original Article

Long thoracic nerve release for scapular winging: Clinical study of a continuous series of eight patients

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Scapular winging; Long thoracic nerve; Neurolysis

Summary
Scapular winging secondary to serratus anterior muscle palsy is a rare pathology. It is usually due to a lesion in the thoracic part of the long thoracic nerve following violent upper-limb stretching with compression on the nerve by the anterior branch of thoracodorsal artery at the “crow’s foot landmark” where the artery crosses in front of the nerve. Scapular winging causes upper-limb pain, fatigability or impotence. Diagnosis is clinical and management initially conservative. When functional treatment by physiotherapy fails to bring recovery within 6 months and electromyography (EMG) shows increased distal latencies, neurolysis may be suggested. Muscle transfer and scapula-thoracic arthrodesis are considered as palliative treatments. We report a single-surgeon experience of nine open neurolyses of the thoracic part of the long thoracic nerve in eight patients. At 6 months’ follow-up, no patients showed continuing signs of winged scapula. Control EMG showed significant reduction in distal latency; Constant scores showed improvement, and VAS-assessed pain was considerably reduced. Neurolysis would thus seem to be the first-line surgical attitude of choice in case of compression confirmed on EMG. The present results would need to be confirmed in larger studies with longer follow-up, but this is made difficult by the rarity of this pathology.

Level of evidence: III.

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Introduction

Scapular winging (scapula alata) is usually due to a lesion of the long thoracic nerve that innervates the serratus anterior muscle.
It may also, more rarely, arise from a lesion of the accessory nerve or dorsal nerve of the scapula inducing respectively trapezius and rhomboid muscle palsy. Finally, it may result from fascio-scapulo-humeral muscular dystrophy, or other incidental causes, inducing disordered scapulothoracic rhythm mimicking the same symptomatology (aseptic necrosis of the humeral head, acromial fracture non-union) [1].

Many reports have focused on the various possible lesional etiologies, and most frequently on long thoracic nerve traction and/or compression between its cervical origin at the interscalene or infraclavicular groove and its distal terminal branches in the serratus anterior muscle [2–12].

Whether the original long thoracic nerve lesion was idiopathic or traumatic, most cases show spontaneous recovery. Surgical indications for long thoracic neurolysis or palliative treatment are rare.

Patients present with pain [8,10,13,14], fatigability [13] or upper-limb functional impotence, with scapular winging (Fig. 1) generally appearing secondarily during the 2 weeks following onset [13,15].

Diagnosis is often late, due not to technical difficulty but to the rarity of the entity [16–18], so that time to treatment may be prolonged, with numerous non-contributive examinations. It should be borne in mind in case of pain or prolonged impairment in the scapular region.

Definitive diagnosis is founded on electromyography (EMG), showing lengthened distal latency with respect to the healthy side [16,19,20].

The present hypothesis is that release of the latero-thoracic part of the long thoracic nerve, following Laulan [21], is an effective and reproducible treatment for serratus anterior palsy. We report a continuous single-surgeon experience.

**Material and method**

A continuous single-center single-surgeon series was operated on between 2009 and 2012.

**Table 1** details the present series: nine operations in eight patients (1 bilateral lesion), comprising release of the long thoracic nerve for symptomatic scapular winging resistant to conservative medical treatment and inducing major functional impairment, mainly due to pain.

There were five women and three men (including the bilateral case), with a mean age of 38 years (range, 16 to 48 years). One was a manual worker, one developed scapular winging following spine surgery, two after apparently single trauma, and four after apparently iterative microtrauma. Three had already suffered scapular winging, with spontaneous resolution. Compression was systematically demonstrated on preoperative EMG, showing increased distal latency.

Surgery was under general anesthesia, without curare, to allow for neurostimulation. The patient was positioned in lateral decubitus. Surgical loupes were used systematically. A vertical incision was performed on the median axillary line forward of the anterior edge of the latisimus dorsi muscle. The latisimus dorsi was drawn back to locate the thoracodorsal artery. Nerve function was checked by neurostimulation (serratus anterior contraction with good scapular motion). Neurolysis was then performed along the entire nerve, removing fibrous formations and fascial expansions with coagulation and sectioning of the anterior collateral from the lateral thoracic bundle crossing in front of the nerve. The collateral branches were spared (Fig. 2). Patients were not immobilized postoperatively; only a sling was used, to reduce pain. Immediate active self-rehabilitation was recommended, with unrestricted everyday activity. Resumption of sport and heavy lifting was authorized on a case by case basis according to functional recovery and EMG findings.

Follow-up was at 6 weeks and 3, 6 and 12 months post-surgery. Clinical work-up comprised pain assessment on a visual analog scale (VAS) from 0 (no pain) to 10 (worst possible pain), Constant functional score (0 to 100) and examination for scapular winging. Neurological recovery was checked by EMG at a mean 9 months (range, 6–12 months) post-surgery.

Statistical analysis used the Wilcoxon test for matched variables, with the significance threshold set at $P < 0.05$.  

![Figure 1](image1.png) **Figure 1** Patient from back, left scapular winging.

![Figure 2](image2.png) **Figure 2** Long thoracic nerve neurolyzed and after coagulation of anterior collateral of thoracodorsal artery (black).
**Table 1**

<table>
<thead>
<tr>
<th>Patients</th>
<th>Sex</th>
<th>Age</th>
<th>Dominant side</th>
<th>Affected side</th>
<th>Occupation</th>
<th>Sport</th>
<th>Upper-limb history</th>
<th>Etiology</th>
<th>Symptomatology</th>
</tr>
</thead>
<tbody>
<tr>
<td>1a</td>
<td>M</td>
<td>20</td>
<td>R</td>
<td>L</td>
<td>Student</td>
<td>Body-building</td>
<td>R + L shoulder dislocation</td>
<td>Effort in body-building</td>
<td>Pain, limb anteflexion deficit, scapular winging</td>
</tr>
<tr>
<td>1b</td>
<td>M</td>
<td>20</td>
<td>R</td>
<td>R</td>
<td>Student</td>
<td>Body-building</td>
<td>R + L shoulder dislocation</td>
<td>Effort in body-building</td>
<td>Pain, scapular winging</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>20</td>
<td>L</td>
<td>R</td>
<td>Student</td>
<td>Swimming, badminton</td>
<td>—</td>
<td>Effort in body-building</td>
<td>Pain, scapular winging</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>16</td>
<td>R</td>
<td>L</td>
<td>Student</td>
<td>Dance, running</td>
<td>4 LTN palsies, all medical</td>
<td>Effort in body-building</td>
<td>Pain, scapular winging</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>42</td>
<td>R</td>
<td>R</td>
<td>Mechanic</td>
<td>—</td>
<td>—</td>
<td>Paralysis after spine surgery</td>
<td>Pain, scapular winging</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>48</td>
<td>R</td>
<td>R</td>
<td>Civil servant</td>
<td>—</td>
<td>—</td>
<td>?</td>
<td>Pain, limb anteflexion deficit, scapula alata</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>43</td>
<td>R</td>
<td>R</td>
<td>Secretary</td>
<td>Cycling, hiking</td>
<td>2 LTN palsies, all medical</td>
<td>Effort in body-building</td>
<td>Pain, instability, cracking, scapular winging</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>25</td>
<td>R</td>
<td>R</td>
<td>Sales</td>
<td>—</td>
<td>1 LTN palsy, medical</td>
<td>Effort in lifting</td>
<td>Pain, limb anteflexion deficit, scapular winging</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>47</td>
<td>R</td>
<td>R</td>
<td>Invalid</td>
<td>—</td>
<td>Cervicobrachial syndrome</td>
<td>Effort in lifting</td>
<td>Pain, scapular winging</td>
</tr>
</tbody>
</table>

LTN: long thoracic nerve.
Results

Overall results

Results are shown in Table 2. All eight patients were followed up, for a mean 8 months (range, 6–12 months). Patient 1, with bilateral scapular winging, was lost to follow-up with respect to assessment of the second shoulder.

Functional score

Constant scores showed improvement (except for patient 8) from a mean 46.2 preoperatively to 66.7 at end of follow-up ($P=0.0007$). In detail, activity level improved from 6.5 to 13.5, motion from 20.5 to 29.5 and force from 10.8 to 14.25.

Pain

Pain reduction was systematic as of week 6, from a mean score of 5.8 to 1.38 ($P=0.006$), except for patient 8 in whom pain (paresthesia in the C6–C7 territory and lateral cervical pain due to associated cervicobrachial syndrome, known prior to surgery) persisted up to 3 months.

Scapular winging

At 6 months, there were no clinical signs of scapular winging; three patients already showed none at 6 weeks.

EMG

EMG found distal latency elevated to a mean 8.4 ms (range, 5.8–13 ms) preoperatively, falling to 5.5 ms (range, 4–8 ms) postoperatively, with signs of at least partial serratus anterior reinnervation ($P=0.008$).

Complications

Type-1 complex regional pain syndrome appeared in patient 8: over and above the associated cervicobrachial symptoms, she had diffuse upper-limb pain and vasomotor trophic disorder.

Discussion

The etiologies of long thoracic nerve lesions remain poorly known. They have, however, been the focus of numerous studies, totaling almost 200 patients [9] and implicating: positional disorder [1,8,22–24], shoulder trauma [20,24–26] and sport [13,15,27–30]; Vastamäki [9] implicated sport in 35% of cases and iatrogenic lesions in 16%. Neurologic etiologies include poliomyelitis, which can lead to bilateral involvement [25,31], although the most frequent is “brachial neuritis”, also known as amyotrophic neuralgia or paucisymptomatic Parsonage Turner syndrome [31]. In Parsonage and Turner's princeps description, 136 cases included 30 isolated serratus anterior palsies [30]. Other lesions may be of incidental origin: burns [32], radiation therapy sequelae [33], lupus [34], overdose [13], compression by hematoma under anticoagulants [35], or anatomic deformity, notably of one side of the neck [35]. Finally, idiopathic lesions have been implicated at rates varying from 11% for Vastamäki [9] to 10 cases out of 20 for Foo [13].

Overall, most lesions seem to be secondary to traction. Rotation of the head toward the side opposite the shoulder associated to raising the arm doubles the length of the nerve between its two fixed points: the exit from the scalene and fixation by the fascia of the superior muscle digitation [28]. Raising the arm induces posterior, inferior and lateral displacement of the nerve [28]. The association of both movements, notably in sports players, may thus explain microtraumatic etiologies. Vastamäki, on the other hand, implicates rather prolonged effort or violent movement [9].

Natural evolution is usually toward some degree of recovery between 6 and 12 months. Complete recovery appears to be rare, but the clinical series of conservative management have been rather imprecise, given the range of etiologies. Out of 32 iatrogenic lesions, Kaupilla [22] reported only three patients who could be considered cured; the other 29 complained of fatigue on effort (88%) or inability to work with the arms upstretched (54%); 27% had permanent scapular winging and 46% showed scapular winging on effort. In an analysis of the literature, Fery found 30% insufficient results following conservative management, certain authors reporting as many as two-thirds [25].

Thoracolumbar orthoses have been used to maintain scapula-thoracic contact [29,36]: e.g., in 65% of Kaupilla's patients [22], for 3 to 12 hours per day for almost 9 months. Many other authors do not consider them useful [5,10]. Such orthoses, introduced at the turn of the century, have been regularly updated as they are, in general, poorly tolerated and little worn. We have no experience of them, but one study reported use of orthosis in 14 patients and made a complete review of the literature on the various models [37].

Conservative management consists basically in waiting for spontaneous recovery and guarding against pathogenic activity [38]. This is a reasonable attitude, at least during the first 6 months. Functional rehabilitation is offered [39], to conserve shoulder flexibility and scapulothoracic joint integrity; the patient performs arm-raising exercises in a lying position to stabilize the scapula; stretching is not allowed, so as not to worsen the stretched nerve lesion.

In case of suspected traumatic or microtraumatic etiology, when EMG has identified a lesion implicating plexual elongation requiring supraclavicular nerve surgery, and 6 months of physiotherapy has failed to achieve recovery, neurolysis may be beneficial. Diagnosis frequently being late, palliative surgery is often offered after several years without recovery. By then no direct surgical action is possible on the nerve, as neurogenic muscular atrophy has set in, and palliative treatment should be considered to stabilize the scapula on the thorax, using dynamic stabilization by tendon transfer [40,41] or static stabilization by scapulopexy or scapulothoracic arthrodesis [42].

Neurolysis through a latero-thoracic approach is relatively non-invasive, effective, reproducible and enduring, unlike dynamic or static stabilization techniques. Its thus appears as the surgical treatment of first intention in case EMG findings of compression with increased distal latency,
<table>
<thead>
<tr>
<th>Patient</th>
<th>Preoperative constant score</th>
<th>Postoperative constant score</th>
<th>Preoperative VAS</th>
<th>Postoperative VAS</th>
<th>Preoperative EMG (months)</th>
<th>Postoperative EMG</th>
<th>Time to resolution of pain</th>
<th>Time to resolution of scapular winging</th>
</tr>
</thead>
<tbody>
<tr>
<td>1a</td>
<td>52</td>
<td>89</td>
<td>4</td>
<td>0</td>
<td>6.1</td>
<td>4.6 to 6 months</td>
<td>6 weeks</td>
<td>6 weeks</td>
</tr>
<tr>
<td>1b</td>
<td>50</td>
<td>Lost to FU</td>
<td>5</td>
<td>Lost to FU</td>
<td>5.8</td>
<td>Lost to FU</td>
<td>Lost to FU</td>
<td>Lost to FU</td>
</tr>
<tr>
<td>2</td>
<td>68</td>
<td>92</td>
<td>7</td>
<td>1</td>
<td>9.9</td>
<td>5.8 to 8 months</td>
<td>6 weeks</td>
<td>6 weeks</td>
</tr>
<tr>
<td>3</td>
<td>48</td>
<td>68</td>
<td>7</td>
<td>2</td>
<td>7.2</td>
<td>4.8 to 6 months</td>
<td>6 weeks</td>
<td>6 weeks</td>
</tr>
<tr>
<td>4</td>
<td>42</td>
<td>79</td>
<td>5</td>
<td>0</td>
<td>13</td>
<td>8 to 10 months</td>
<td>6 weeks</td>
<td>6 months</td>
</tr>
<tr>
<td>5</td>
<td>36</td>
<td>64</td>
<td>5</td>
<td>1</td>
<td>8.9</td>
<td>5.5 to 6 months</td>
<td>6 weeks</td>
<td>6 weeks</td>
</tr>
<tr>
<td>6</td>
<td>44</td>
<td>52</td>
<td>6</td>
<td>0</td>
<td>6</td>
<td>4.8 to 7 months</td>
<td>3 months</td>
<td>6 months</td>
</tr>
<tr>
<td>7</td>
<td>46</td>
<td>62</td>
<td>5</td>
<td>1</td>
<td>8.3</td>
<td>4 to 12 months</td>
<td>6 weeks</td>
<td>4 months</td>
</tr>
<tr>
<td>8</td>
<td>30</td>
<td>28</td>
<td>8</td>
<td>6</td>
<td>10</td>
<td>5.2 to 12 months</td>
<td>Persistence</td>
<td>3 months</td>
</tr>
</tbody>
</table>

VAS: visual analog scale; EMG: electromyography.
which found both diagnosis and indication; results are good, as in the present series and as shown by Laulan [14], with 100% recovery. These results of course should be borne out by larger studies, to achieve greater statistical power; but in this rare pathology it is difficult to find a sufficient number of cases.

Conclusion

Serratus anterior muscle palsy is a rare pathology, generally following trauma by violent stretching of the upper-limb and hence of the long thoracic nerve. Diagnosis may be late, due not to its difficulty but to the rarity of the entity; time to treatment is thus often long. It should be borne in mind in case of pain or prolonged unexplained functional impairment in the scapular region, so as to avoid being reduced to palliative measures.

Increased distal latency on EMG confirms diagnosis, indicating neurolysis of the thoracic part of the long thoracic nerve.

Disclosure of interest

P. Clavert is a consultant for Mitek et Tornier, SA.

Concerning the present article: None.

The authors declare that they have no conflicts of interest concerning this article.

References


