Chapter 4
Trauma and Thoracic Outlet Syndrome

TRAUMA

Trauma to the brachial plexus can be divided into open injuries and closed injuries.[127] Closed injuries are traction injuries and can be divided into supraclavicular, retroclavicular, infraclavicular and mixed injuries. Supraclavicular lesions are more common than retro- and infraclavicular lesions: in a series of 810 cases 75% of the cases were supraclavicular against 25% retro- and infraclavicular.[6,7] Supraclavicular injuries can be subdivided into supraganglionic (preganglionic) and infraganglionic (postganglionic) lesions. This subdivision has important therapeutic consequences, as a supraganglionic lesion, which is a nerve root avulsion, cannot be repaired directly and neurotization techniques or musculotendinous transpositions may be used,[76,128,129,134,165,166,240,246] while the more distal infraganglionic lesions can be restored by local repair.[150] Neurotization is a technique by which an intact neighboring donor nerve is separated from its end organ and then connected directly or by nerve grafts to the distal portion of a non-functioning nerve. In brachial plexus surgery the spinal accessory nerve, rami of the cervical plexus, or intercostal nerves can be transferred to parts of the brachial plexus. Clinically and electrodiagnostically there are some signs and symptoms which may differentiate between supra- and infraganglionic lesions.[28,133,134] Horner's syndrome is suggestive of a nerve root avulsion of Th1. A positive Tinel's sign, which is distal tingling produced by percussing the injured nerves, indicates an infraganglionic lesion. Paralysis of the serratus anterior and rhomboid muscles suggests a supraganglionic lesion. When there is besides limb muscle denervation, paravertebral muscle denervation, a supraganglionic lesion is suspected. Nerve conduction studies may suggest a supraganglionic lesion if motor conduction is absent and sensory conduction is present, while with infraganglionic lesions they are both absent.

Nerve root avulsions occur when there is simultaneously traction of the arm and throwing of the head to the opposite side. By far the most common cause in adults is a motorcycle accident.[6,7,161,164,226] Another important cause is the birth related brachial palsy.[81,224,225] Sunderland[237,241] has described two ways by which nerve roots may be avulsed from the spinal cord: the peripheral and central mechanism. With the peripheral mechanism, lateral traction first causes rupture of the ligamentous attachments of the spinal nerve to the transverse processes and tearing of the dural cone, followed by avulsion of the nerve root. The nerve roots of C8 and Th1 lack the ligamentous attachments of the spinal nerve to the transverse processes, so they have a higher incidence of nerve root avulsions than C5, C6 and C7, which do have a strong attachment to the transverse processes.[99,236] The tearing of the dura causes cerebrospinal fluid to escape into the surrounding tissues, so leaving a traumatic meningocele within two or three weeks.[239] With this mechanism it is conceivable that a dural tear coincides with an intact nerve root, which means that the presence of a traumatic meningocele does not always indicate a nerve root avulsion. The central mechanism causes nerve root avulsions by violent displacements of the spinal cord. With this mechanism the nerve roots can avulse without tearing the dural cone, which means that there is no traumatic meningocele. Although not very common, traumatic meningoceles can exist without nerve root avulsions and nerve root avulsions can occur without traumatic meningoceles.[39,95,130,148,169,225,237,239,266]

Retro- and infraclavicular injuries of the brachial plexus can be caused by shoulder dislocation, fractures of the clavicle or shoulder girdle, and by violent movement of the shoulder causing stretching of the brachial plexus. Clavicular fractures can coincide with brachial plexus compression. This can be instantaneous by a piercing bone fragment, or of delayed onset by malunion, nonunion or extensive callus formation, which requires surgery.[13,18,43,80,103,107,146] The space between the clavicle and first rib in particular may be narrowed by a bone fragment or by extensive callus formation. The prognosis for retro- and infraclavicular lesions is better than for supraclavicular ones.[5,131,134,135,163]

Two classifications are in use to determine the severity of the nerve lesion. Seddon[216] used three types: neurotmesis, axonotmesis and neurapraxia. Neurotmesis means literally cutting of a nerve. All essential structures of the nerve have been destroyed, which makes spontaneous healing impossible. In axonotmesis there is damage to the nerve fibers, which causes a complete loss of neurological function distally. Spontaneous recovery is possible, because the Schwann tubes, endoneurium and perineurium are intact. Neurapraxia (apraxia literally means non-action) describes paralysis without an anatomic lesion. There is a spontaneous and complete recovery. Sunderland introduced a more precise classification, consisting of five degrees of injury.[235] First degree injury (= neurapraxia): a reversible conduction block with an intact anatomical continuity. Second degree injury (= axonotmesis): damage to the axons with distally Wallerian degeneration; complete recovery occurs because of the intact endoneurial tubes. Third degree injury: the continuity of the endoneurial tubes is damaged, which causes incorrect reinnervation. Fourth degree injury: there is a complete disorganization of the nerve, which is still continuous; there can be some recovery, but without a useful degree of function. Fifth degree injury (= neurotmesis): there is a complete interruption of the nerve.

If a nerve is transected, post-traumatic neuromas can develop in infraganglionic, retro- and infraclavicular lesions.[149] A post-traumatic neuroma is a disorganized proliferation of regenerating axons at the proximal stump of a transected nerve. It consists of nerve fascicles with Schwann cells, fibroblasts and axons with myelin.[64] The first published surgical repair of a rupture of the brachial plexus was in 1900 by Thorburn.[206,247] The indication for surgical exploration is absent spontaneous clinical improvement. Surgery within the first six months will give the best prognosis.[15,134] First, an exact intraoperative diagnosis is necessary by exploring the supra- and infraclavicular region of the brachial plexus. Then, depending on the kind of lesion, microsurgical neurolysis, nerve grafting or neurotization can be performed.[15,149] Secondary operations may be necessary and include musculotendinous transfers and arthrodesis.[16]

Imaging

It is important to determine the site of the lesion, as this has significant prognostic and therapeutic consequences. Reliable imaging of the presence or absence of nerve root avulsions is most important. Myelography is very useful to demonstrate traumatic meningoceles in patients with nerve root avulsions.[159,243] It is also possible to see the roots surrounded by contrast.[38,160] CT myelography improved the detection of small traumatic meningoceles.[95,139,154,190,207,264,266,267] If at least 2 mm sections are used, the nerve roots can be well visualized, although the image quality of the lower roots can be less adequate due to artifacts from the shoulders. An important advantage of MR imaging in the detection of nerve root avulsions is its non-invasiveness: no intradural contrast is necessary. There have been several (case) reports about the appearance of nerve root avulsions and traumatic meningoceles on MR imaging.[46,54,74,87,142,144,148,169,177,192,194,200,201,207,254,259,260,266] Traumatic meningoceles are fluid collections extending from the neural foramen which follow the signal intensities of the cerebrospinal fluid in all sequences. MR imaging can depict the traumatic meningoceles very well, even those which do not have a communication with the dural sac, but cannot reliably show all nerve roots.[74,148,169,177,264] Because, as mentioned before, traumatic meningoceles occur without nerve root avulsions and nerve roots can avulse without traumatic meningoceles, it is necessary to image the roots themselves and not only the traumatic meningoceles. CT myelography is considered to be the most reliable investigation for the imaging of the nerve roots.[28,207,266,267] The main advantage of MR imaging in trauma patients is the visualization of the extraforaminal part of the brachial plexus. Thickening of the brachial plexus with and without an increased signal intensity on T2-weighted images can be seen, presumably due to respectively edema[114,200,259,261] and fibrosis.[87,144] Post-traumatic neuromas are seen as well-defined tumors of the brachial plexus with a low signal intensity on T1-weighted images and a high signal intensity on T2-weighted images.[100,177,200,229] MR imaging can demonstrate other causes of brachial plexopathy after trauma, such as a hematoma[21,194,229] and a clavicle fracture[21,200,223,229,259,261,262] with brachial plexus compression. The progress of imaging techniques at this time has not been sufficient to preclude diagnostic surgery to define intraoperatively the exact extent of the lesion.[15]

Own material

Patients with trauma and abnormal MR imaging

Table 1 describes 31 patients with a history of trauma and abnormalities with MR imaging, examples are shown in Figs. 1-5. In this group, 10 patients (patients Nos. 139, 140, 142, 144, 146, 148, 151, 153, 155 and 159) are described who presented with a flail arm after a severe trauma, especially motorcycle accidents (seven patients). In five patients a 3D-TSE (Figs. 1 and 2) was performed which was surgically correlated in three patients (patients Nos. 142, 144 and 155). In all three patients there were more avulsions found at surgery than meningoceles demonstrated with MR imaging. Other abnormalities seen with MR imaging were thickening of the brachial plexus in six patients (patients Nos. 139, 140, 142, 144, 148 and 153), hematomas in three patients (patients Nos. 142, 151 and 155), and in two patients compression by a clavicle fracture upon the brachial plexus (patients Nos. 146 and 148). In our experience, MR imaging was not able to demonstrate neurotmesis, as shown in patients Nos. 142 (torn upper trunk found at surgery), 144 (intraforaminal neurotmesis of root C5 found at surgery) and 153 (neuromas of the trunks found at surgery, Fig. 3). No surgery was performed in three patients because of spontaneous improvement (patients Nos. 146, 148 and 151). An explanation for the clinical improvement could be the release of compression of the brachial plexus by a healing clavicle fracture (patients Nos. 146 and 148) and a resolving hematoma (patient No. 151, Fig. 4).

In three patients (patients Nos. 150, 154 and 157) with known nerve root avulsions MR imaging was requested to rule out neuroma formation as an explanation for pain. In this group no neuromas could be demonstrated.

In 14 patients (patients Nos. 138, 141, 145, 147, 149, 152, 156, 158, 160, 161, 162, 164, 165 and 167) MR imaging was performed because of a clavicle fracture with symptoms of brachial plexus compression (Fig. 5). In eight patients (patients Nos. 141, 145, 149, 152, 156, 160, 161 and 165) compression of the brachial plexus was seen, whereas in six patients (patients Nos. 138, 147, 158, 162, 164 and 167) the clavicle fracture or callus did not disturb the brachial plexus. Surgical decompression was done in four patients (patients Nos. 149, 156, 161 and 165) and is planned in one patient (patient No. 145) from the group with brachial plexus involvement on

MR imaging. Clinical improvement was seen in all four patients. Only one patient (patient No. 164) from the group where MR imaging did not show brachial plexus compression was operated and this patient remained symptomatic until surgery for a cervical disc protrusion.

The remaining four patients had various pathology: one patient (patient No. 137) with an old stab wound, one patient (patient No. 143) with a coracoid process fracture, one battered child (patient No. 163) and one patient (patient No. 166) with a shoulder luxation.

Patients with trauma and normal MR imaging

Table 2 describes 18 patients with a possible brachial plexus lesion due to trauma and no abnormalities with MR imaging. In most, spontaneous improvement was seen without a definitive diagnosis. Two patients were operated (patient Nos. 168 and 172). Although no abnormalities with MR imaging were detected, neurotmesis of the axillary, radial, musculocutaneous, median and ulnar nerves in patient No. 168 was found, and neurotmesis of the roots C5 and partially of C6 was found in patient No. 172.

Conclusions

MR imaging of the brachial plexus in a patient who presents with a flail arm after an accident has not been very conclusive diagnostically in our experience. Unfortunately, it is not possible to make a distinction between neurotmesis, axonotmesis and neurapraxia. In two cases (patient Nos. 168 and 172) the brachial plexus appeared to be normal with MR imaging, while at surgery neurotmesis was found. MR imaging can demonstrate compression of the brachial plexus by a hematoma or a clavicle fracture. CT myelography is, up until now, the most sensitive investigation to diagnose traumatic nerve root avulsions.[267] MR imaging can, in our experience demonstrate meningoceles well, but it is not always possible to evaluate the nerve roots themselves. However, the use of a 3D-TSE sequence improves the visualization of the nerve roots and this may replace CT myelography in the future.

THORACIC OUTLET SYNDROME

Thoracic outlet syndrome is a very confusing syndrome with controversial therapeutic options. Although the term thoracic outlet syndrome was introduced by Peet[186] in 1956, this syndrome and its different causes have been extensively described before. Wilbourn[273] has written a nice historical overview with three eras of increased surgical interest. The first was the "invariably symptomatic cervical rib syndrome" era, when the presence of a cervical rib was consistently associated with any upper extremity symptoms.[14,62,205] The therapy was removal of the cervical ribs. Adson[2] advised performing a tenotomy of the anterior scalene muscle instead of a cervical rib resection, as he found the same relief with this less difficult procedure. The next era was the “scalenus anticus syndrome” era. In 1935 Ochsner reported patients with a cervical rib syndrome without a cervical rib present.[170] The syndrome was thought to be caused by spasm of the anterior scalene muscle. Elevation of the first rib so caused compression of the subclavian artery and lower trunk of the brachial plexus. The therapy was an anterior scalenotomy. The scalenus anticus syndrome lost popularity because of its low surgical success rate, and because cervical radiculopathy and carpal tunnel syndrome were recognized as different causes of upper extremity symptoms.[274] Other described compression sites were the costoclavicular area with the costoclavicular syndrome[66,67] and the subcoracoid region with the hyperabduction syndrome.[277] Peet[186] introduced the term “thoracic outlet syndrome”, which includes all described compression syndromes and he described a conservative therapeutic regimen. Surgical interest increased again in the early 1960s in the “disputed neurogenic thoracic outlet syndrome” era. The first rib was then thought to be the main cause of compressing the neurovascular structures and Clagett[37] advised removal of the first rib. He recommended a posterior thoracotomy approach which left a large scar. The first rib resection became popular when Roos[209] introduced the transaxillary approach. Roos[211] also introduced the total anterior scalenectomy for a new type of thoracic outlet syndrome with symptoms around the shoulder, due to compression of the ventral rami of the upper roots of the brachial plexus (C5, C6 and C7). The advantage of excising the anterior scalene muscle instead of only transection is that reattachment and extensive scarring is less likely to occur.[215] The most extensive surgery for thoracic outlet syndrome became a combination of transaxillary first rib resection and excision of the anterior and middle scalene muscles.[199]

In 1988 Wilbourn[274] proposed a new classification of thoracic outlet syndrome, in which there is a separation between the vascular and neurologic types. In this classification there are three vascular types (major arterial, minor arterial, and venous) and two neurologic types (true neurogenic and disputed neurogenic).

The “major arterial vascular thoracic outlet syndrome” is rare and is caused by a cervical rib which compresses the subclavian artery.

The “minor arterial vascular thoracic outlet syndrome” is more common. With hyperabduction and exorotation of the arm positional ischemia occurs. A cervical rib is usually not present.

The “venous vascular thoracic outlet syndrome” causes spontaneous thrombosis of the subclavian vein.

The “true neurogenic thoracic outlet syndrome” is a typical clinical syndrome most often found in young women and is associated with an elongated transverse process of C7 or a cervical rib, from which a fibrous band extends to the first rib. The C8 and

Th1 ventral rami of the roots or the lower trunk are stretched over this fibrous band.[82,90,274] The subclavian artery can also be elevated and compressed by this band.[17] The transverse process of the seventh cervical vertebra is normally not larger than that of the first thoracic vertebra. If the transverse process of C7 extends beyond that of Th1, it can be seen as an abortive attempt to form a cervical rib.[210] The characteristic clinical picture is that of wasting and weakness of especially the lateral thenar muscles, usually combined with the medial forearm muscles. Other symptoms are pain, paresthesias and sensory loss along the medial aspect of the arm, forearm and hand. Nerve conduction studies are often typical and helpful in excluding a carpal tunnel syndrome and ulnar nerve entrapment at the elbow.[83,278] The therapy is surgical division of this fibrous band.[82,90]

The "disputed neurogenic thoracic outlet syndrome" includes all disorders in which there is suspected brachial plexus compression without the typical clinical, and radiologic findings as seen in the true neurogenic thoracic outlet syndrome.[49,274] Many symptoms might be present: pain, numbness and paresthesias in the lower trunk distribution, shoulder pain and back discomfort. The electrophysiologic studies can be normal.[218] Surgical treatment consists of first rib resections or scalenectomies, which can be complicated by serious brachial plexus injuries.[34,271] Because the clinical picture is unclear and there are no reliable diagnostic tests, this type is overdiagnosed and overtreated.[272] Before treatment it is important to exclude other treatable diagnoses, such as carpal tunnel syndrome and cervical radiculopathy.

There has been one report of an athlete with the typical clinical and electrophysiologic findings of a true neurogenic thoracic outlet syndrome but without a cervical rib or an elongated transverse process of C7. At surgery compression of the lower trunk by a hypertrophied anterior scalene muscle was found.[110]

Imaging

Conventional radiographs can show bony abnormalities, which include cervical ribs, elongated transverse processes of C7, and exostoses or old fractures of the first rib and clavicle. Conventional angiography is useful in detecting the major arterial vascular thoracic outlet syndrome, but might be replaced by MR angiography.[172] CT can add only little information regarding osseous abnormalities. Interposition of the transverse process of C7 between the anterior and middle scalene muscles can be shown, as well as the contact between a cervical rib and the brachial plexus. However this is not a very useful sign, as it can occur on both the symptomatic and asymptomatic sides. Fibrous bands cannot be detected on CT scans.[22] With MR imaging distortion of the brachial plexus can be seen. There is only one article which studied the value of MR imaging in thoracic outlet syndromes.[180] This article described that a mid- to low-intensity band, which was called a MRI band, could distort the brachial plexus. A sensitivity of 79% and a specificity of 87.5% was found when the distortion of the brachial plexus on MR imaging was correlated with the clinical symptoms. However, in the same study a MRI band was also found in three asymptomatic volunteers which was associated with brachial plexus distortion in one, so that the value of these findings remains unclear. These good results have been criticized by Cherington[35], who disagreed with the MR imaging abnormalities. In conclusion thoracic outlet syndrome is a very controversial subject in clinical medicine as well as in diagnostic radiology.

Own material

Table 3 describes 23 patients with a variety of clinical symptoms consistent with thoracic outlet syndrome, examples are shown in Figs. 6-8. Two patients (patients Nos. 189 and 205), with an elongated transverse process of C7, presented with the typical true neurogenic thoracic outlet syndrome. In both patients a fibrous band, which caused neural compression, was found. In patient No. 205 (Fig. 6) a subtle angulation of the ventral ramus of root C8 was found in comparison with the other normal side. MR imaging of patient No. 189 showed no abnormalities. All other patients, except for patient No. 201, had a variety of symptoms consistent with disputed neurogenic thoracic outlet syndrome. From this group surgery was performed in 10 patients. Clinical improvement was seen in seven patients (patients Nos. 186, 192, 196, 198, 199, 203 and 204), while three patients (patients Nos. 191, 207 and 208) remained symptomatic. All of these patients showed no abnormalities with MR imaging.

Three patients with cervical ribs were imaged, two were symptomatic (patients Nos. 190 and 202) and one asymptomatic (patient No. 201, Fig. 7). In patients Nos. 190 and 201 angulation of the brachial plexus was seen, indicating that angulation does not necessarily correlate with symptoms.

Two patients (patients Nos. 196 and 204) were imaged after a resection of a cervical rib and an anterior scalenotomy. In patient No. 204 (Fig. 8) the anterior scalene muscle appeared to be very thick, despite prior surgery. After a new anterior scalenotomy and a first rib resection the symptoms have disappeared.

Conclusions

Thoracic outlet syndrome is clinically very difficult to diagnose. MR imaging does not seem to be very helpful in making this diagnosis. We have not been able to visualize fibrous bands. Angulation of a ventral ramus of nerve root C8 was found in one of the two patients with the true neurogenic thoracic outlet syndrome. However, angulation of the nerves does not necessarily coincide with symptoms, as was demonstrated in a patient with a cervical rib who was asymptomatic.

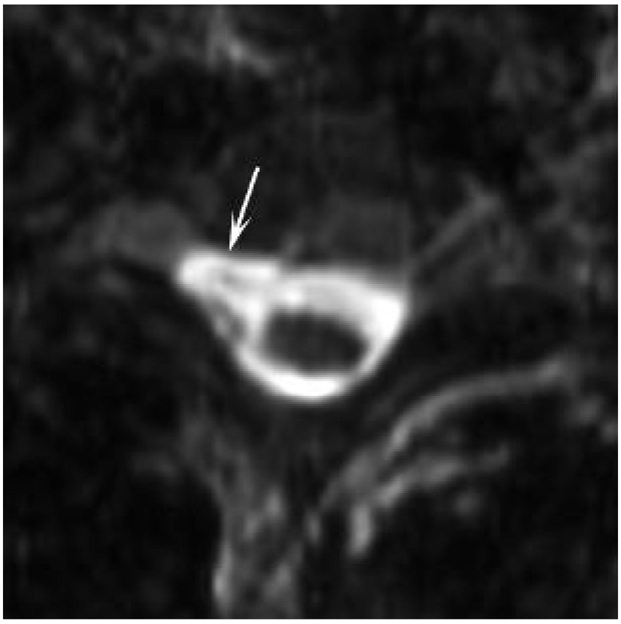

Fig. 1A

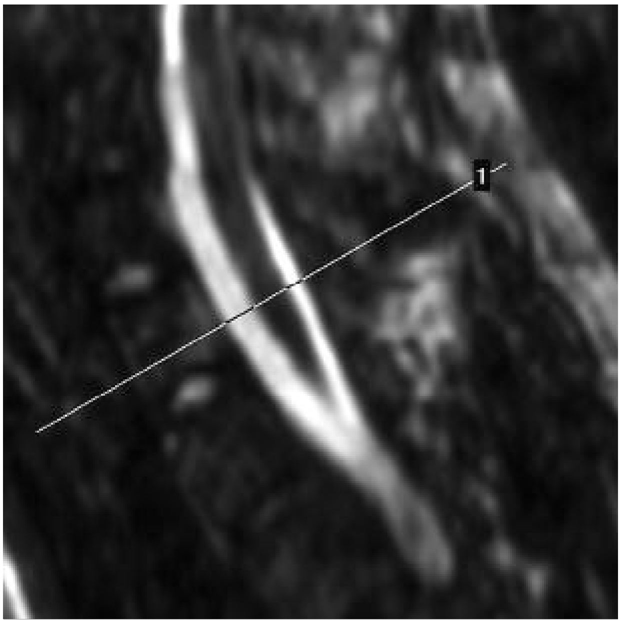

Fig. 1B

Fig. 1. Patient No. 146: traumatic meningocele C6-7 after a motorcycle accident five weeks ago.

A. and **B.** Oblique axial reconstruction of a 3D-TSE volume acquisition (**A**) with the sagittal scanogram (**B**) shows a meningocele (arrow).

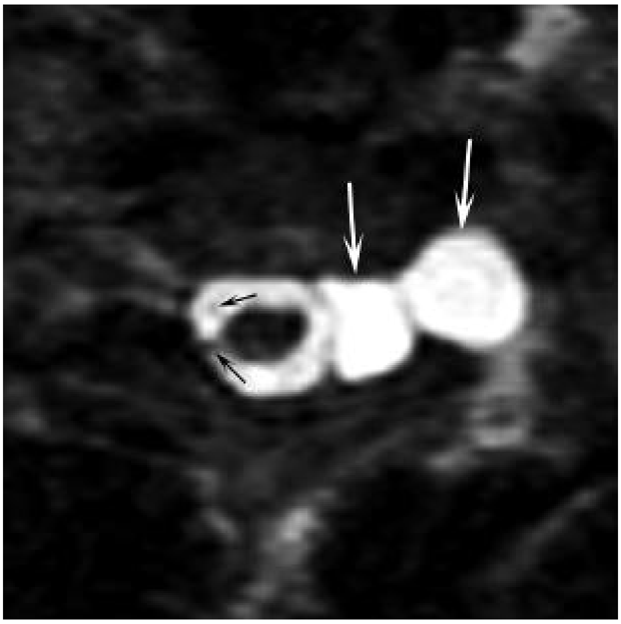

Fig. 2

Fig. 2. Patient No. 154: nerve root avulsion and traumatic meningocele due to a motorcycle accident 20 years ago. Axial TSE image shows a traumatic meningocele (white arrows) on the left with a nerve root avulsion of Th1. Note the intact nerve roots on the right (black arrows).

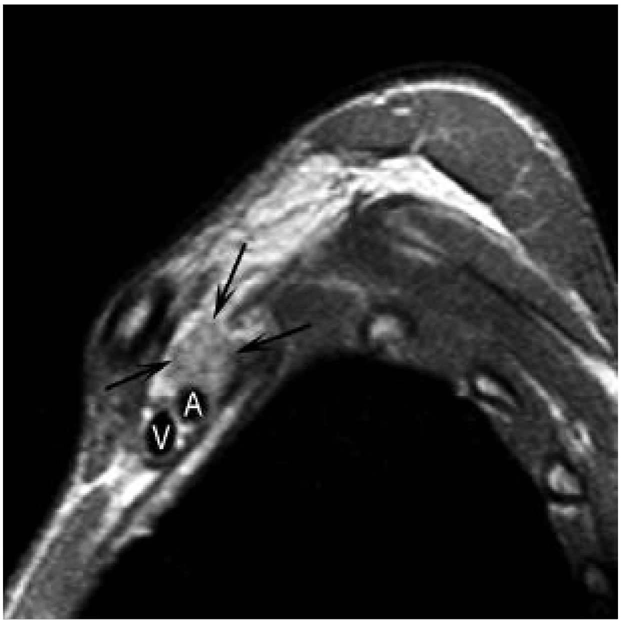

Fig. 3A

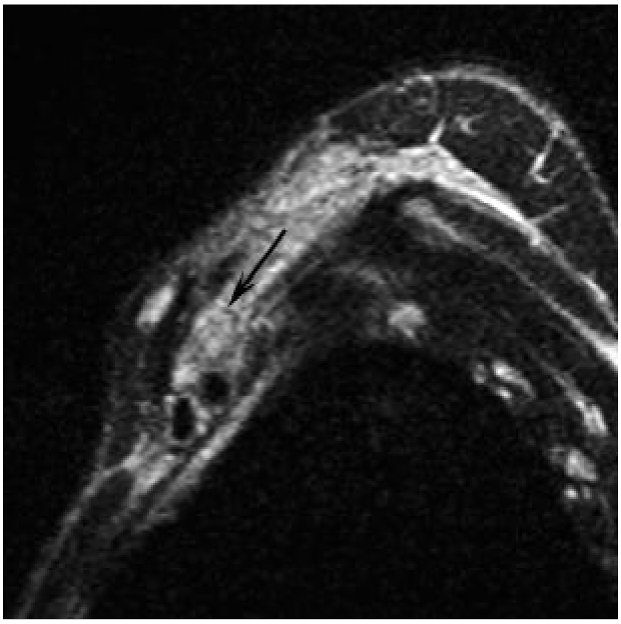

Fig. 3B

Fig. 3C

Fig. 3. Patient No. 153: swollen brachial plexus in a patient with a paralyzed arm 12 days after a bike accident.

A and **B.** Sagittal proton-density (**A**) and T2-weighted (**B**) image at the level of the divisions show a swollen slightly hyperintense brachial plexus (arrows) consistent with edema. A = subclavian artery, V = subclavian vein.

C. Coronal T1-weighted image shows a diffusely swollen brachial plexus, particularly marked in the trunks and divisions (long arrows). The short arrow points to the dorsal scapular artery, which passes through the brachial plexus. A = subclavian artery.
Surgery performed 12 weeks later showed rupture of the trunks with neuroma formation.

Fig. 4A

Fig. 4B

Fig. 4C

Fig. 4D

Fig. 4. Patient No. 151: hematoma in the interscalene triangle in a patient with a paralyzed arm five weeks after a skiing accident.

A. Sagittal T1-weighted image shows a hematoma in the interscalene triangle (long white arrow). There is also a large hematothorax (short white arrow). The black arrow points to the anterior scalene muscle, m = middle scalene muscle.

B. Sagittal T1-weighted image lateral to **A** shows that the hematoma (long arrow) is located between the anterior (a) and middle (m) scalene muscles. Short arrow points to the hematothorax.

C. Sagittal T1-weighted image at the level of the cords of the brachial plexus shows that the cords (black arrows) are swollen, white arrow points to the axillary artery.

D. Coronal T1-weighted image of the hematoma (long arrow), short arrow points to the hematothorax.

Fig. 5A

Fig. 5B

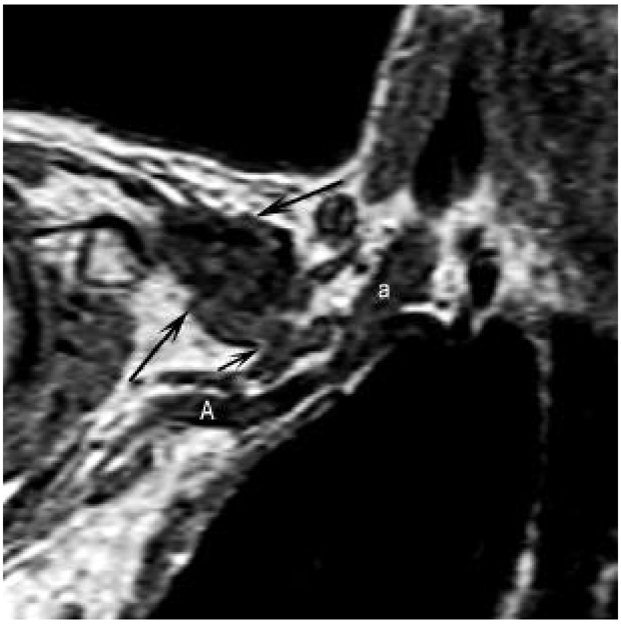

Fig. 5C

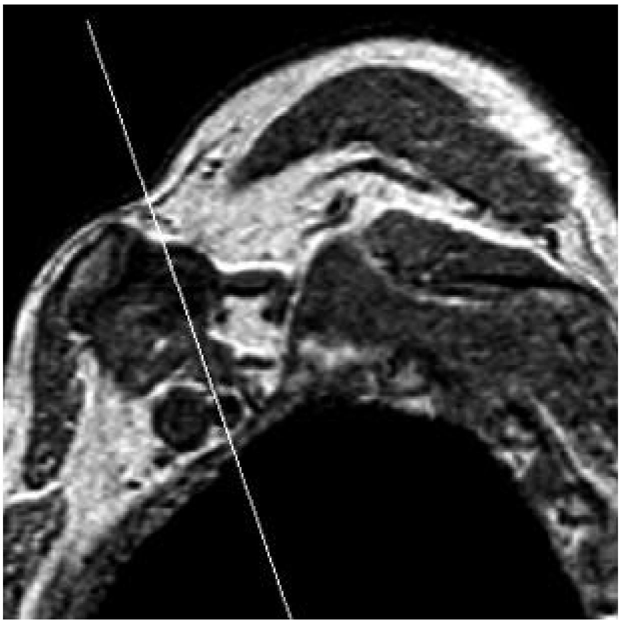

Fig. 5D

Fig. 5. Patient No. 149: clavicle fracture with compression of the brachial plexus.

A. Sagittal image from a T1-weighted 3D volume acquisition shows compression of the brachial plexus (long arrow) by callus (short arrows). A = axillary artery, V = axillary vein.

B. Coronal T1-weighted image demonstrates the relation between the brachial plexus and the callus (arrow).

C. and **D.** Oblique coronal reconstruction of a T1-weighted 3D volume acquisition (**C**) with the sagittal scanogram (**D**) shows the compression of the brachial plexus (short arrow) by the callus (long arrows) better. A = axillary artery, a = anterior scalene muscle.

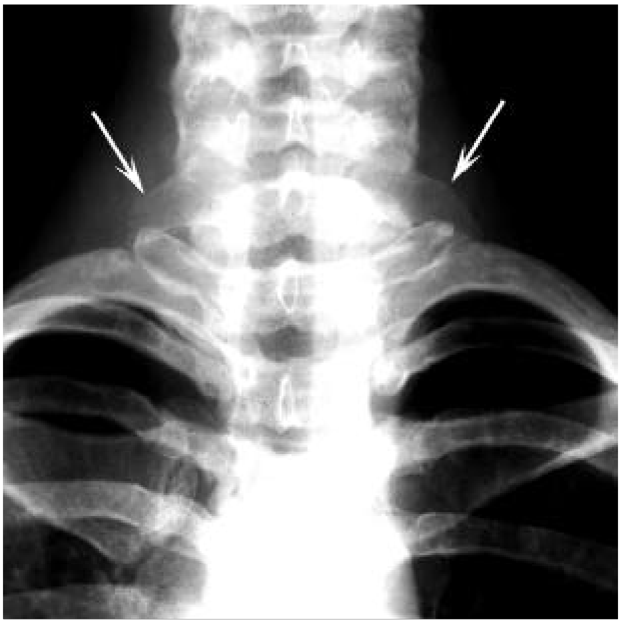

Fig. 6A

Fig. 6. Patient No. 205: left-sided true neurogenic thoracic outlet syndrome in a patient with an elongated transverse process of C7.

A. Conventional radiograph shows the bilateral elongated transverse processes of C7 (arrows).
B. Coronal T1-weighted image demonstrates a normal right ventral ramus of root C8 (arrow). C7, Th1 = vertebral body C7, Th1.
C. Coronal T1-weighted image shows that the left ventral ramus of root C8 is slightly more curved (arrow). Th1 = vertebral body Th1.
D. and **E.** Oblique coronal reconstruction of a T1-weighted 3D volume acquisition (**D**) with the axial scanogram (**E**) shows both ventral rami of root C8 in one plane. Note the angulation on the left side (short arrow) compared to right side (long arrow). C7, Th1 = vertebral body C7, Th1.
At surgery compression of the left ventral ramus of root C8 by a fibrous band was found.

Fig. 6B

Fig. 6C

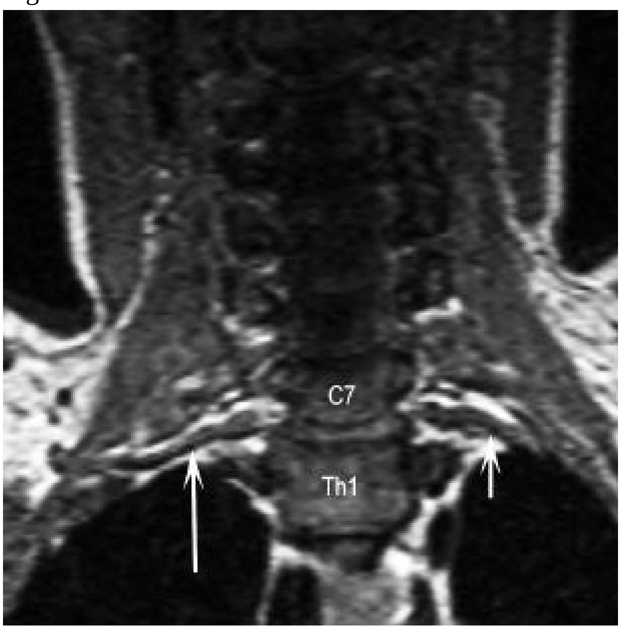

Fig. 6D

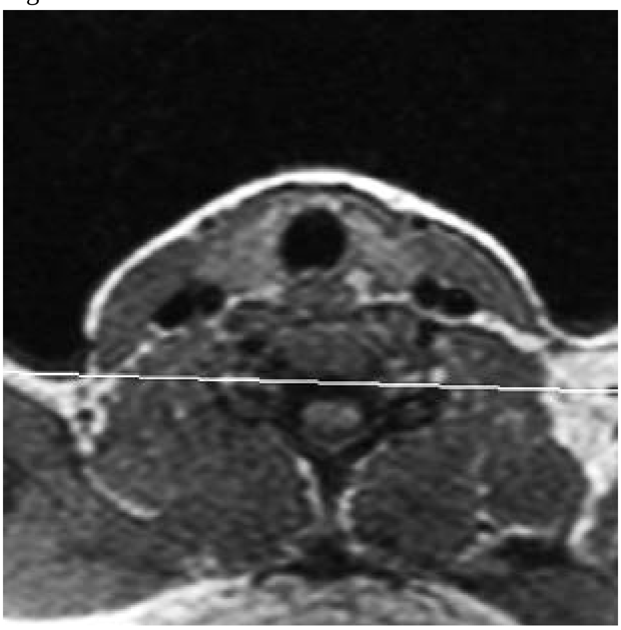

Fig. 6E

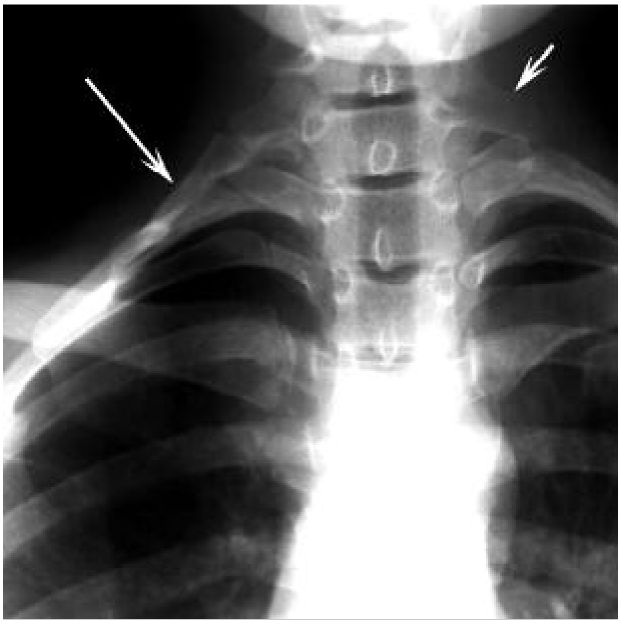

Fig. 7A

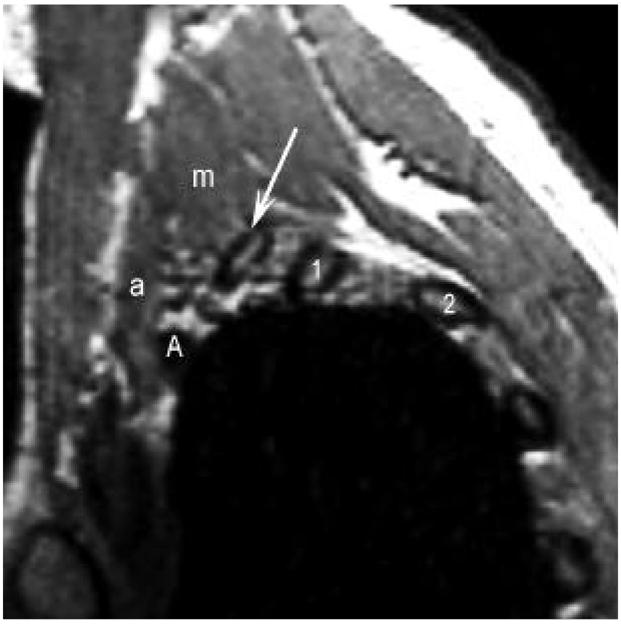

Fig. 7B

Fig. 7C

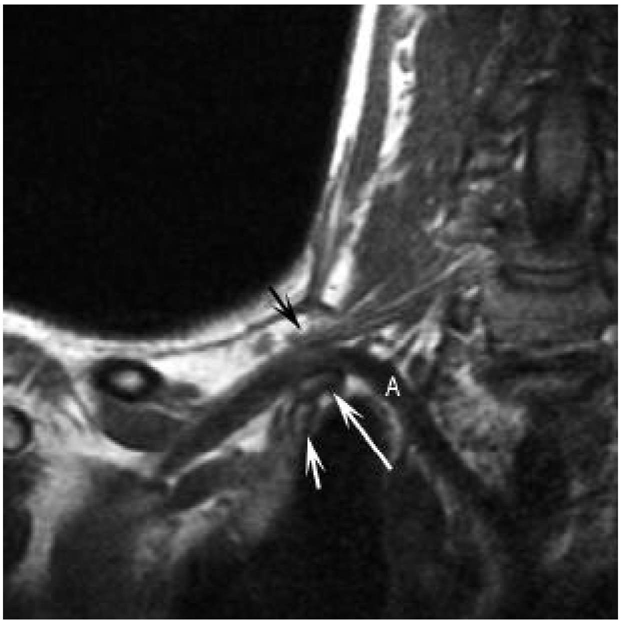

Fig. 7D

Fig. 7. Patient No. 201: asymptomatic cervical rib.

A. Conventional radiograph shows the right cervical rib (long arrow); note the elongated left transverse processes of C7 (short arrow).

B. Sagittal image from a T1-weighted 3D volume acquisition shows the insertion of the middle scalene muscle (m) upon the cervical rib (arrow). A = subclavian artery, a = anterior scalene muscle, 1 = first rib, 2 = second rib.

C. Coronal T1-weighted image shows the articulation of the cervical rib (arrow) with the transverse process of C7.

D. Coronal T1-weighted image more anteriorly than in **C** shows a slight angulation of the brachial plexus (black arrow) and subclavian artery (A) due to the cervical rib (long white arrow) which articulates with the first rib (short white arrow).

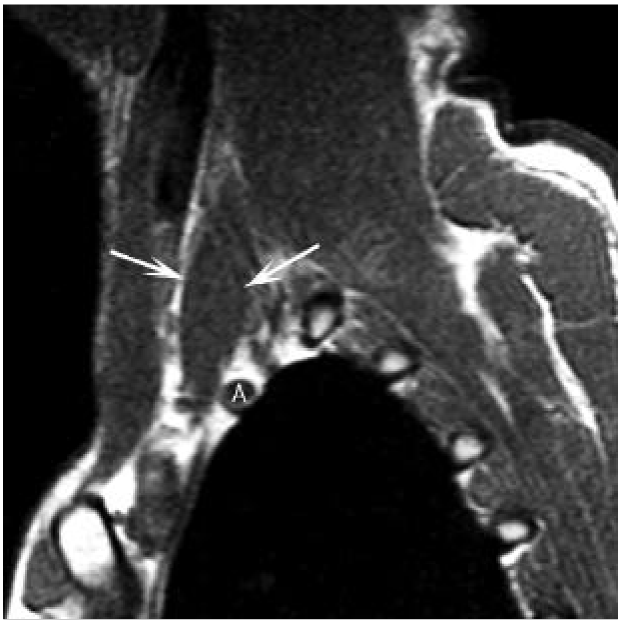

Fig. 8A

Fig. 8B

Fig. 8. Patient No. 204: in this patient a cervical rib was removed and a scalenotomy performed on both sides; the left side remained symptomatic.

A. Sagittal T1-weighted image shows a large anterior scalene muscle (arrows), despite the prior scalenotomy. A = subclavian artery.

B. Coronal T1-weighted image demonstrates the difference in size between the small right anterior scalene muscle (short arrow) after a successful anterior scalenotomy, and the enlarged left anterior scalene muscle (long arrow). After a second anterior scalenotomy and a first rib resection the symptoms of the left side disappeared.

Table 1

Trauma and abnormal MR imaging

pt no	m/f	age	presenting symptoms	MR imaging findings	diagnosis/follow-up
137	m	31	supraclavicular stab wound 4 years ago, pain arm	fibrosis adjacent to the brachial plexus	no therapy, symptoms have disappeared
138	f	39	clavicle fracture 3 years ago, 1st and cervical rib resection 1 year ago, progressive trophy intrinsic hand muscles and paresthesias hand	no brachial plexus compression	hyperabduction trauma during surgery
139	m	20	motorcycle accident 7 months ago, paralysis arm	slightly thickened brachial plexus	surgery 3 months later: avulsion of roots C6 and C7
140	m	20	motorcycle accident 5 weeks ago, paralysis arm, acute surgery 5 weeks ago showed at least avulsions of 3 roots	3D-TSE not assessable due to movement artifacts, thickened trunks, CT myelography: avulsions without meningoceles C5-C7, avulsion with meningocele C8, Th1 not assessable due to streak artifacts of the shoulders	no further surgery
141	m	73	clavicle fracture and partial clavicle resection 6 years ago, pain in neck and shoulder	callus with brachial plexus compression	pain treatment
142	m	29	motorcycle accident 3 weeks ago, paralysis arm	3D-TSE: meningocele C7-Th1, hematoma around the brachial plexus with swollen trunks	surgery 3 months later: avulsions roots C7, C8, Th1, torn upper trunk
143	m	27	coracoid process fracture 7 years ago treated with osteosynthesis which was removed 2 years ago, shoulder remains painful	no brachial plexus compression	cervicothoracic facetsyndrome C3-Th3, pain treatment
144	m	26	motorcycle accident 10 days ago, paralysis arm	3D-TSE: meningoceles C6-7, C7-Th1, thickened brachial plexus	surgery 3 months later: avulsions of C6-Th1, intraforaminal neurotmesis C5 with neuroma formation
145	f	24	clavicle fracture 3 times, last fracture 6 years ago, malunion, paresthesias arm	callus with brachial plexus compression	scheduled for lateral clavicle resection
146	m	19	motorcycle accident 5 weeks ago, clavicle fracture, paralysis arm	3D-TSE: meningoceles C5-6, C6-7, clavicle fracture with compression of brachial plexus	2 months later: spontaneous improvement, except for the triceps muscle; hypesthesia C7, C8 and Th1
147	m	34	clavicle fracture and distal clavicle resection 3 years ago, pain in shoulder and paresthesias hand, normal EMG	no brachial plexus compression	no diagnosis, physiotherapy
148	f	42	hit by car 2 months ago, paralysis arm and clavicle fracture	clavicle fracture with compression of brachial plexus, brachial plexus slightly thickened	no surgery because of spontaneous improvement of C5, C6 and C7 functions
149	m	48	clavicle fracture 2 months ago, severe paresis arm and hand muscles	clavicle fracture with compression of brachial plexus	surgery: decompression of the brachial plexus, weakness disappeared postoperatively
150	m	34	motorcycle accident 16 years ago, paralyzed arm, pain in hand	3D-TSE: meningoceles C6-7, C7- Th1, Th1-Th2, no neuroma	reflex sympathetic dystrophy
151	m	31	skiing accident 5 weeks ago, clavicle and 1st rib fracture, paresis arm	hematoma in interscalene triangle	spontaneous improvement

Table 1 *Continued.*

Trauma and abnormal MR imaging

pt no	m/f	age	presenting symptoms	MR imaging findings	diagnosis/follow-up
152	f	51	clavicle fracture followed by resection 1st rib and part clavicle 2 years ago, pain arm and hand	callus and fibrosis with brachial plexus compression	no surgery
153	m	20	bike accident 12 days ago, paralyzed arm	diffusely swollen brachial plexus	surgery 3 months later: neuromas of the trunks
154	f	39	motorcycle accident 20 years ago, paralyzed arm, pain in arm and hand	3D-TSE: meningoceles C6-7, C7- Th1, Th1-Th2, no neuroma	reflex sympathetic dystrophy
155	m	28	motorcycle accident 3 weeks ago, paralyzed arm, acute surgery: rupture brachial plexus and subclavian artery reconstruction	3D-TSE: meningoceles C5-6, C6-7, C7-Th1 and Th1-2, multiple hematomas around the brachial plexus	surgery: avulsions of all roots
156	m	49	clavicle fracture 16 months ago, paresthesias in hand, after 1st rib resection still paresthesias and pain in hand	callus with brachial plexus compression	surgery: brachial plexus decompression, post-operatively the pain has disappeared, paresthesias still present
157	m	40	old trauma, neuroma formation?	meningoceles C5-6, C6-7, C7-Th1, no neuroma	no surgery
158	m	23	clavicle fracture 2 years ago, malunion, pain in shoulder	no brachial plexus compression	pain treatment
159	m	23	motorcycle accident 10 months ago, paresis arm with partially functioning hand musculature	3D-TSE: meningoceles C5-6, C6-7, C7-Th1, old clavicle and 1st rib fracture without brachial plexus compression	no surgery
160	f	58	clavicle fracture 2 months ago, pain in shoulder, arm and hand	clavicle fracture with compression of brachial plexus	no surgery, spontaneous improvement
161	f	56	clavicle fracture 24 years ago, non-union, pain in hand	callus with brachial plexus compression	surgery: decompression brachial plexus, post-operatively the pain has disappeared
162	m	53	old clavicle fracture, thoracic pain	no brachial plexus compression	no therapy
163	f	2	battered child, paralysis proximal arm muscles	normal brachial plexus, myelum contusion	not known
164	m	37	clavicle fracture 3½ months ago, paresthesias arm	no brachial plexus compression	surgery: repositioning fractured parts clavicle, symptoms remained, 8 months later anterior and medius scalenectomy, symptoms remained, 3 months later surgery for protrusion C5-6, post-operatively asymptomatic
165	m	46	clavicle fracture 10 months ago, non-union, pain in shoulder and arm	clavicle fracture with compression of brachial plexus	surgery, postoperatively asymptomatic
166	m	62	shoulder luxation and lesser tubercle fracture, weakness arm	normal brachial plexus, fluid around humeral head	spontaneous improvement
167	m	28	car accident 2 years ago followed by osteotomy clavicle, pain in shoulder and arm	no brachial plexus compression	spontaneous improvement

Table 2

Trauma and normal MR imaging

pt no	m/f	age	presenting symptoms	3D-TSE performed	diagnosis/follow-up
168	m	29	motorcycle accident 2 months ago, paralysis arm	yes	surgery 9 months later: neurotmesis axillary, radial, musculocutaneous, median and ulnar nerves
169	m	25	motorcycle accident 4 months ago, paralysis arm	yes	spontaneous but not complete recovery
170	m	32	neck trauma 6 months ago, weakness shoulder muscles	no	entrapment suprascapular nerve
171	m	33	trauma 1 year ago, sensory deficit Th2	no	no diagnosis
172	m	29	trauma 2 weeks ago, paralysis shoulder and upper arm	yes	surgery 9 months later: neurotmesis of C5 and partially of C6
173	m	39	arthroscopy shoulder 4 months ago, weakness and paresthesias 4th and 5th finger, EMG: lesion C6, C7 and C8	no	traction lesion medial cord, spontaneous improvement
174	m	14	contusion shoulder	no	physiotherapy
175	f	22	motorcycle accident 1 year ago, paralysis arm, normal EMG	yes	psychotherapy
176	m	36	motorcycle accident 10 days ago, weakness abductors and exorotators shoulder	yes	no surgery, revalidation
177	m	68	polytrauma 5 months ago, weakness supraspinous muscle and small hand muscles	no	spontaneous improvement
178	f	85	fall on shoulder, paresis arm	no	spontaneous improvement
179	f	27	trauma 4 weeks ago, paralysis arm, EMG: possible neuropraxia	no	spontaneous improvement
180	m	73	trauma in the past, pain and paresthesias 3rd, 4th and 5th finger	no	spontaneous improvement
181	m	32	fall of rock 4 weeks ago, surgery of the skull, 5 days later paresthesias arm and hand, EMG: C5-6 lesion	no	neuropraxia, revalidation
182	m	52	car accident 4 months ago, paresthesias arm	no	whiplash
183	m	57	after fall 10 months ago weakness in shoulder and upper arm	no	cervical spinal canal stenosis
184	m	30	after trauma 1 year ago pain and weakness in shoulder and arm	no	no diagnosis, no therapy
185	m	36	motorcycle accident 2 months ago, weakness in shoulder and upper arm	no	spontaneous improvement

Table 3

Thoracic outlet syndrome and cervical ribs

pt no	m/f	age	presenting symptoms	MR imaging findings	diagnosis/follow-up
186	m	28	thoracic outlet syndrome	normal	anterior and medius scalenectomy, clinical improvement
187	m	48	pain in neck and arm	normal	thoracic outlet syndrome, no surgery
188	m	48	atrophy intrinsic hand muscles for 3 years, EMG suggests lower brachial plexus lesion	elongated transverse process of C7 on radiograph, no brachial plexus compression	not known
189	f	22	paresthesias in hand and weakness in 1st 2nd and 3rd fingers, EMG suggests C8 and Th1 lesion	elongated transverse process of C7 on radiograph, no brachial plexus compression	surgery: fibrous band with compression C8 and Th1
190	f	41	cervical rib and paresthesias in hand	cervical rib with slight angulation C8	not known
191	m	41	pain in neck and shoulder	normal	anterior and medius scalenectomy, no clinical improvement
192	f	37	thoracic outlet syndrome on both sides	elongated left transverse process of C7 on radiograph, no brachial plexus compression	anterior and medius scalenectomy on both sides and removal left transverse process of C7, left side asymptomatic, right side pain in shoulder
193	m	38	paresthesias in hand	normal	thoracic outlet syndrome, no surgery
194	f	32	costoclavicular syndrome	normal	carpal tunnel syndrome or thoracic outlet syndrome
195	f	39	pain in arm and paresthesias in hand	normal	thoracic outlet syndrome, no surgery
196	f	39	removal cervical rib and scalenotomy on both sides 2 years ago, left arm and hand remain painful	small remnant anterior scalene muscle, no brachial plexus compression	1st rib resection on the left side, pain has disappeared
197	f	47	surgery for thoracic outlet syndrome 2 years ago, surgical diagnosis was a neurofibroma, 1 year later tenotomy of pectoralis minor muscle, still paresthesias in arm and hand	elongated transverse process of C7 on radiograph, pectoralis minor and anterior scalene muscle atrophic, no brachial plexus compression	spontaneous improvement
198	f	31	thoracic outlet syndrome	normal	anterior and medius scalenectomy, clinical improvement
199	f	41	pain in shoulder and paresthesias hand	normal	anterior and medius scalenectomy, pain in shoulder remains, hand asymptomatic
200	f	39	paresthesias in arm and hand	small cervical ribs on radiograph, no brachial plexus compression	no surgery
201	f	19	asymptomatic supraclavicular palpable mass	cervical rib with slight compression subclavian artery and slight angulation brachial plexus	no therapy
202	f	32	cervical ribs on both sides, paresthesias and weakness left arm	cervical ribs, relation brachial plexus and cervical ribs not well seen due to little fat	no surgery
203	m	45	thoracic outlet syndrome	normal	anterior and medius scalenectomy, clinical improvement
204	m	38	removal cervical rib and scalenotomy on both sides (right side 3 years ago, left side 1 year ago), paresthesias unchanged on the left side	normal left anterior scalene muscle, atrophic right anterior scalene muscle	1st rib resection and anterior scalenotomy on left side, symptoms have disappeared
205	f	16	atrophy intrinsic hand muscles, EMG: C8 lesion	elongated transverse process of C7 on radiograph, angulation of C8	surgery: fibrous band with compression of C8
206	m	50	costoclavicular compression	normal	not known
207	f	48	paresthesias hand	normal	anterior and medius scalenectomy, clinical improvement, 9 months later recurrence of symptoms
208	f	41	thoracic outlet syndrome	normal	anterior and medius scalenectomy, no clinical improvement, 4 months later after transposition ulnar nerve asymptomatic