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Case Reports

Axillary Aneurysm and Brachial Plexus Lesion

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SUMMARY

A case report is described here of an axillary pseudo-aneurysm associated with brachial plexus lesion, caused by a gun shot injury. Clinical Doppler, electrophysiological and neuro-vascular reconstruction in one stage with restoration of substantial functions in the upper limb.

CASE REPORT

A young man of 35, had sustained a a high velocity rifle bullet injury to his right shoulder. The wound of entry was located in the region of the shoulder joint anteriorly. The wound of exit was present in the posterolateral aspect of the upper arm. The ensuing arterial bleeding was controlled with local compression by the patient himself. He noticed a complete loss of muscle power below the level of the shoulder joint. The wounds were superficially stitched in the local hospital. A month later, he noticed a pulsatile lumpiness in the axilla.

Following the appearance of this aneurysm, he developed local pains in the axilla. The hand and the elbow continued to remain anesthetic and paralysed.

Three months after his initial injury and two months after the development of manifest aneurysm, he presented to our Department of Vascular Surgery for further management.

On clinical examination, there was a near total brachial plexus lesion present, involving the C 5, 6, 7, 8 and T 1 roots. This was characterised by total anesthesia of the upper limb below the upper third of the arm. Sensation above this area were blunted upto the prominence of the shoulder joint. The anteromedial part of the shoulder had retained normal sensations.

Below the level of the upper third of the arm, sensations were universally absent (grade 0/5). Deep pressure sensations were appreciated patchily in the upper most arm. Shoulder, elbow, wrist and finger movements were absent (grade 0/5).

Electrophysiological assessment revealed a total posterior cord lesion, a total median nerve and a partial nerve lesion. A supramaximal stimulation at the Erbs point failed to produced any compound muscle action potential (CMAP) in respect of these nerves. Selective stimulation of these nerves in the lower forearm revealed normal CMAPs distally.

An "incompressible" tender and pulsatile aneurysm associated with a bruit, was present in the apex of the axilla, measuring 3 cm in diameter (Figure 1). Brachial and radial pulses were impalpable. No coldness, cyanosis or trophic changes were found in the fingers.

Doppler flow studies confirmed the aneurysm to arise from the second part of the axillary artery with substantial (90%) intrinsic intra-aneurysmal thrombosis. Proximal supraclavicular compression failed to obliterate the aneurysm. Subclavian bruit was audible on Doppler. The upper brachial artery was not audible but the lower brachial artery in the region of the elbow joint was muffled (sluggish information). Brachioradial flow was very poor on Doppler studies. Radial artery revealed extremely poor flow. No digital arterial flow could be detected but the digital venous return was good. The arterial flow in the brachio-radial segment of the artery was totally stopped with gentle manual compression of the lower part of the arm. The venous return in the entire upper limb was normal.

Angiography performed via a right transfemoral Subclavian III catheterisation revealed a large sized almost fully thrombosed aneurysm in the second part of the axillary artery (Figure 2). The distal neck of the aneurysm was located on the inferomedial aspect of the Axillary artery between its second and the third parts. The distal Axillary artery was fully thrombosed and the profunda and the circumflex

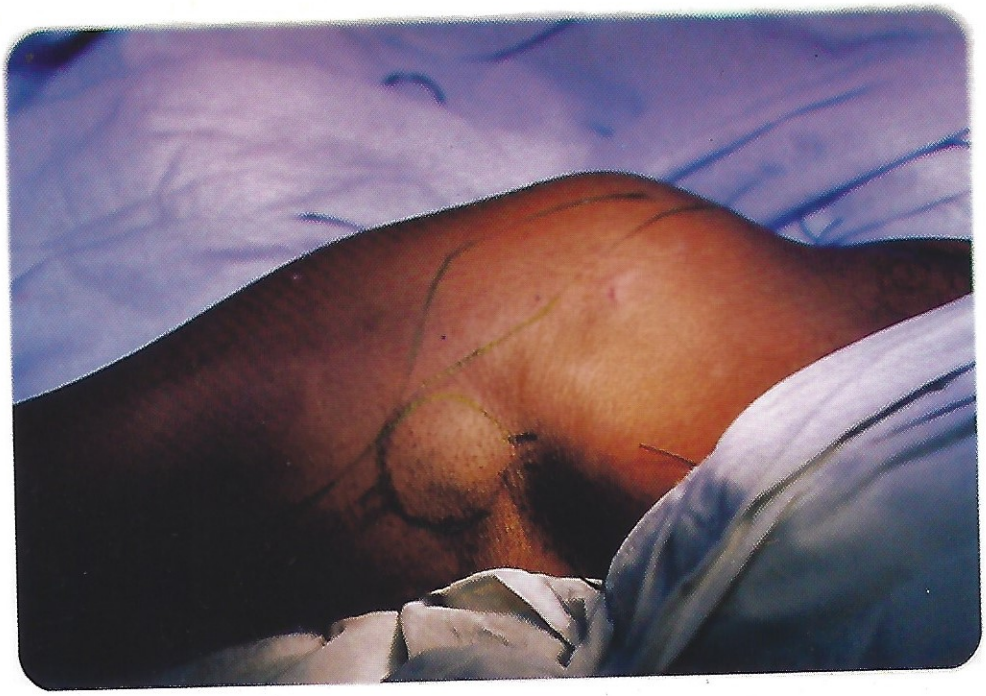

Fig. 1: Axillary aneurysm (surface marked) with proposed alternative incisions.

arteries had collateralized to reform the lower part of the Brachial artery near the middle of the arm, through which a very sluggish flow was seen to the proximal radial and ulnar arteries. The vessels below the wrist were not outlined despite separate injections.

OPERATION

Through a supraclavicular approach, the omohyoid was divided (Figure-3) and an anterior scalenotomy performed. The roots of the brachial plexus (C5,6,7,8, and T 1) were sequentially identified and looped with rubber slings (Figure 4).

Thyrocervical trunk and the Subclavian Artery were secured with rubber slings. The deltopectoral groove was next opened (Figure 5), the cephalic vein displaced and the incision extended to display the Trunks, Divisions and Cord of the plexus. There was considerable adhesions encountered in this area. The distal Subclavian and the proximal axillary arteries were identified and controlled with rubber slings (Figure-6). The posterior cord was found disrupted with a loss of 1 cm nerve segment. The Medial and the Lateral Cords could only be seen in the upper most parts. Gentle traction confirmed continuity above the clavicle. The distal parts of the Cords and their immediate branches had merged into the tough aneurysmal capsule. Gentle dissection of the aneurysmal wall led to identification of the artery proximal and distal to the aneurysm.

At this stage, the long sephanous vein was harvested and the groin wound closed and dressed. The aneurysm was opened and the bleeding aneurysmal ostium overrun with sutures. Similarly other branches were controlled intraaneurysmally (Figures 7 and 8). The proximal artery was perfused with Heparin. The distal segment of the axillary artery was cannulated with an embolectomy catheter which revealed full patency. Reversed arterial flow become established. The distal segment was perfused with Heparin (a total of 3,000 units for both ends). Under a dry field, the aneurysm was totally excised, peeling the walls away from the nerves (Figures 9 and 10). The Medial and the Lateral Cords were identified as intact. Musculocutaneous nerve was intact. Median nerve was found disrupted with loss of 3 cm segment with neuromatous enlargement of the ends. The ulnar nerve was badly lacerated with dense neuromatous enlargement which had become rigidly adherent to the wall of the pseudo- aneurysm. In attempts to excise the neu

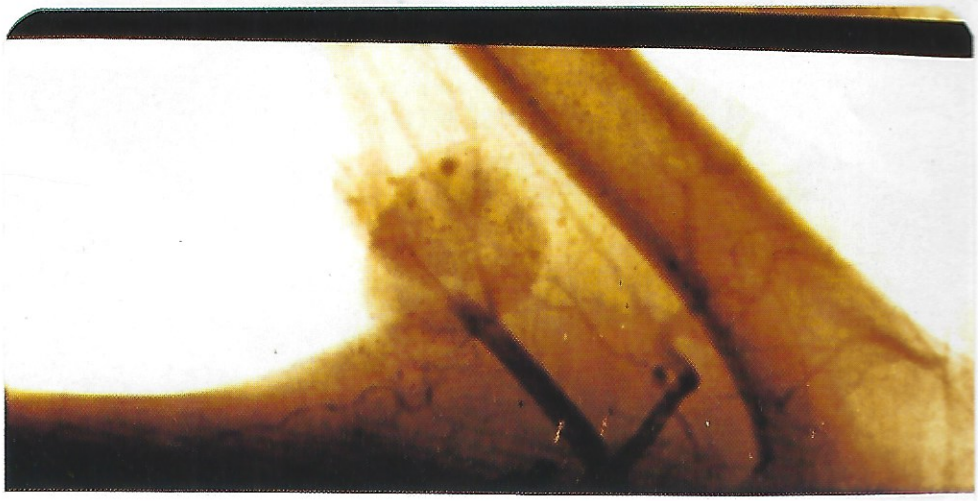

Fig. 2: Partially thrombosed slow-emptying aneurysm.

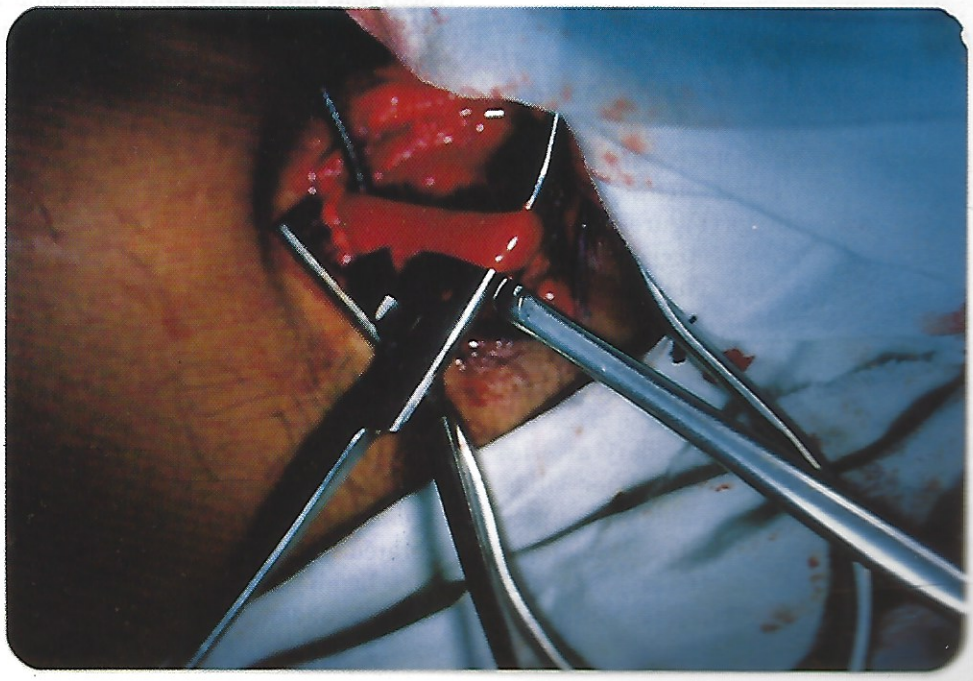

Fig. 3: Omohyoid (supraxlavicular approach).

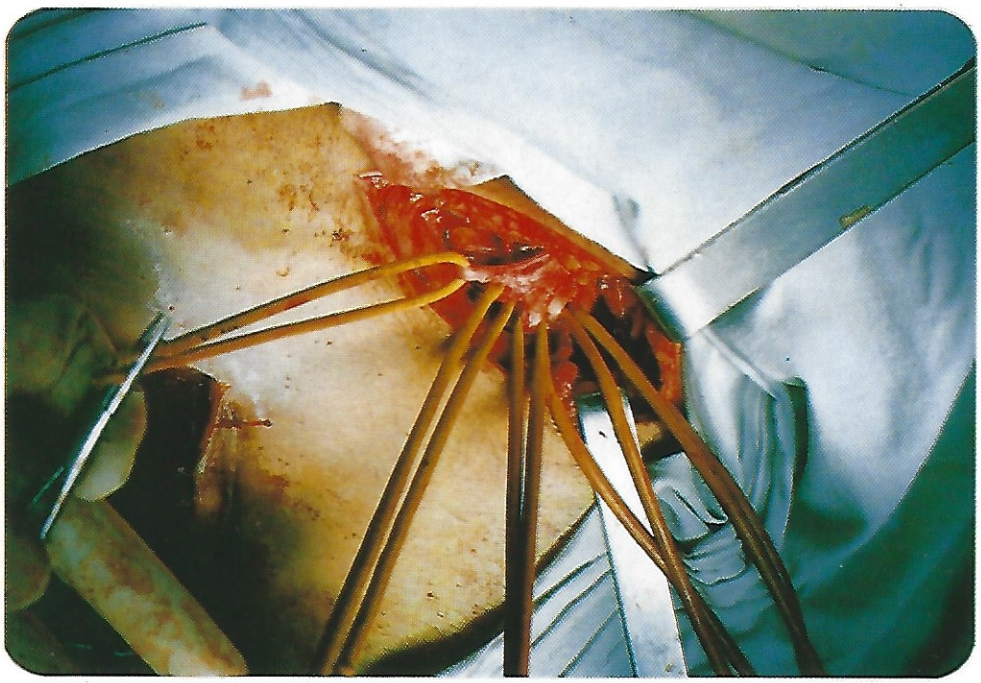

Fig. 4: Isolated supraclavicular roots of brachial plexus.

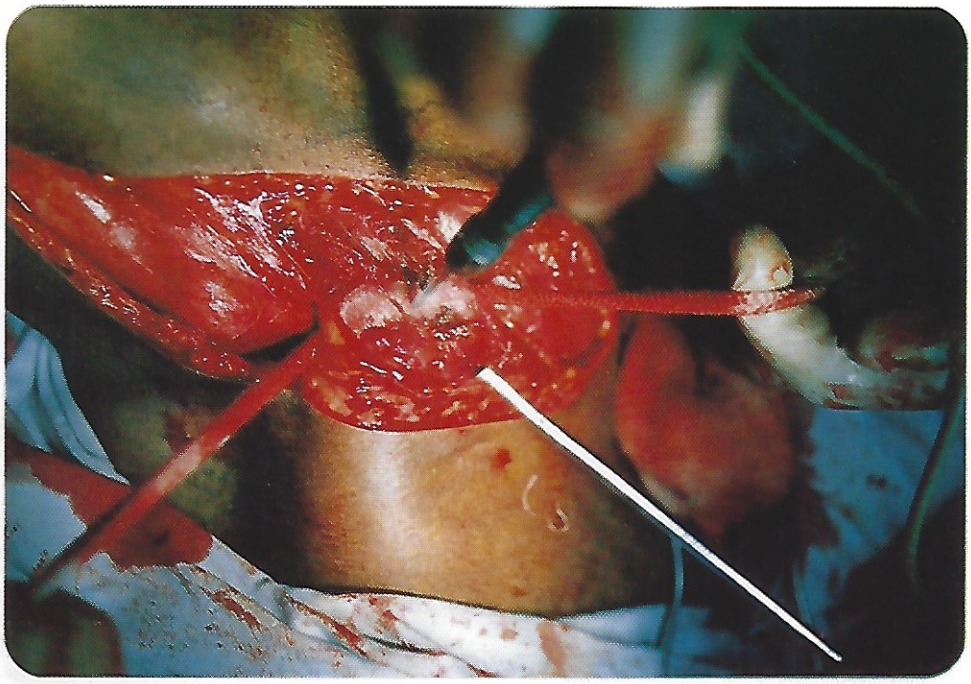

Fig. 5: Deltopectoral exposure (pectoral tenotomy).

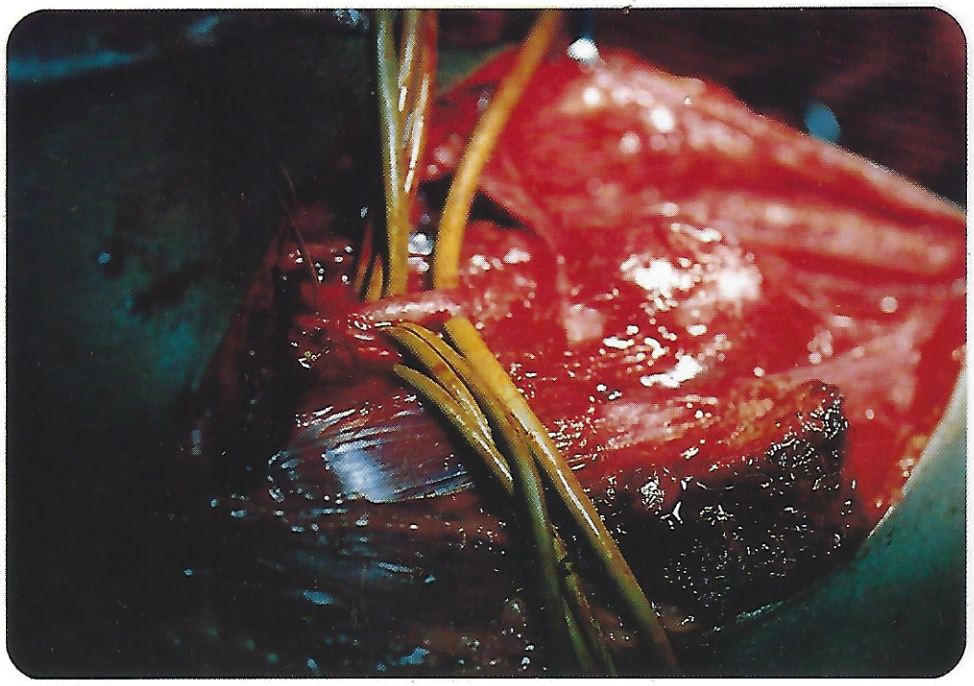

Fig. 6: Axillary artery slung proximal to aneurysm.

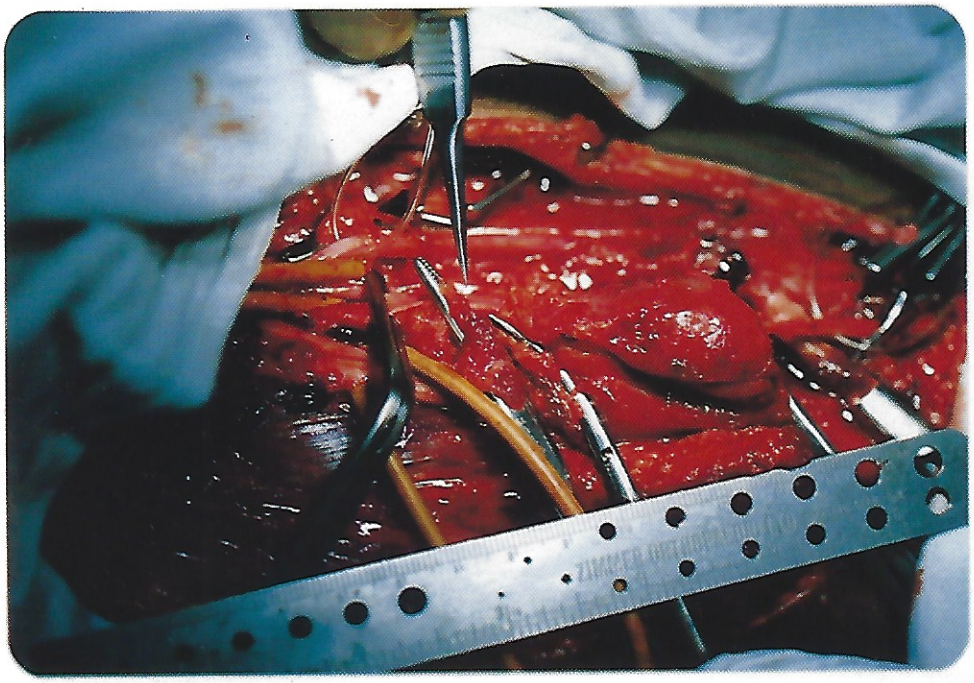

Fig. 7: Axillary aneurysmotomy under vascular control.

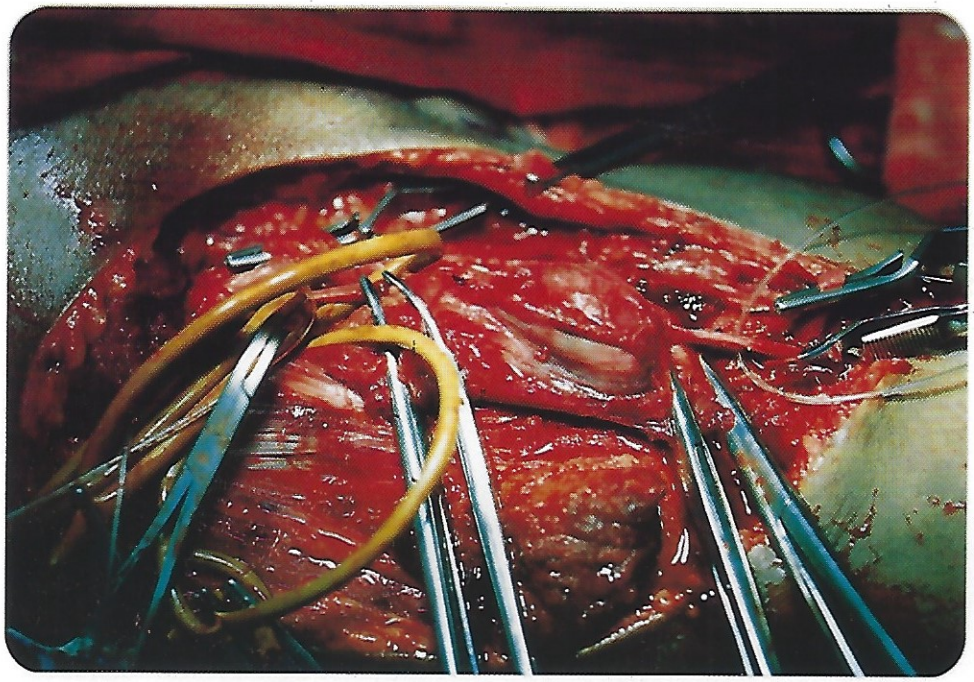

Fig. 8: Intra-aneurysmal haemostasis.

Fig. 9: Adhesions between intact nerves and aneurysmal capsule.

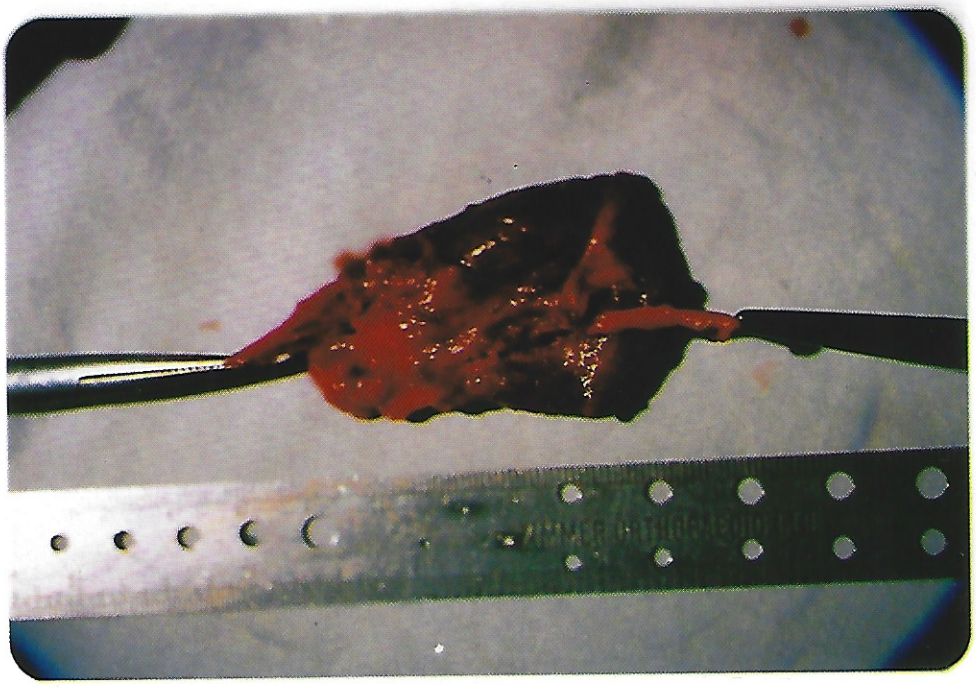

Fig. 10: Excised aneurysm showing arterial connections.

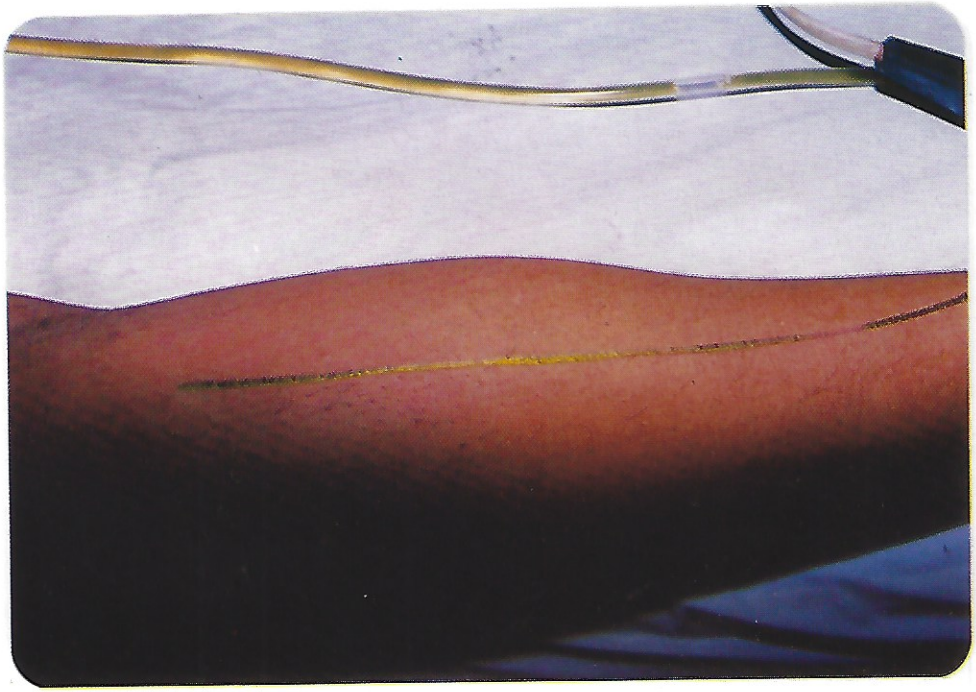

Fig. 11: Surface marking of calf for donor sural nerve.

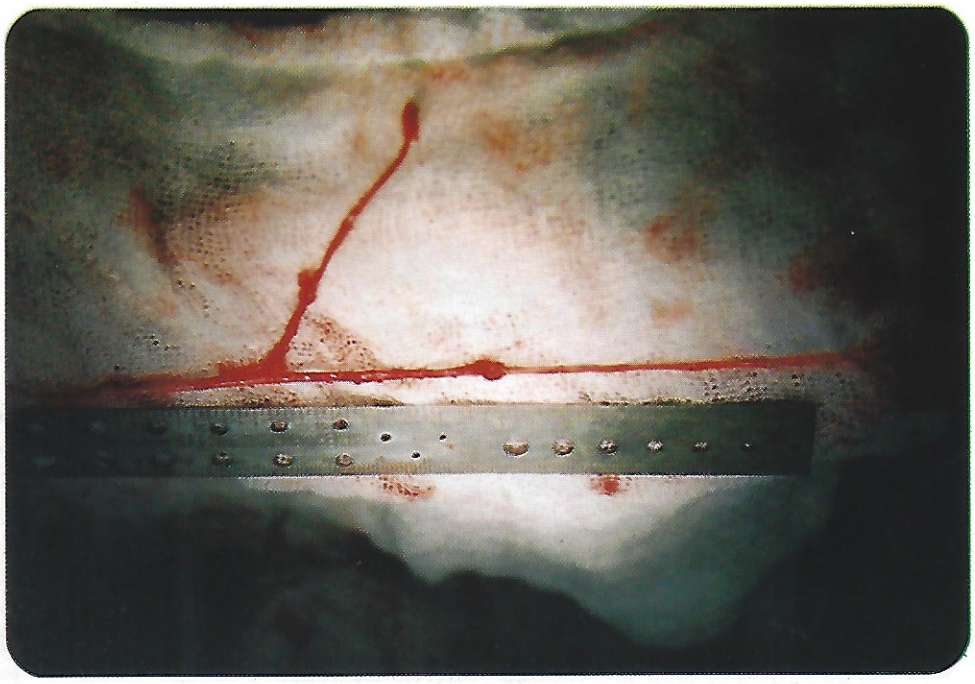

Fig. 12: Harvested sural nerve.

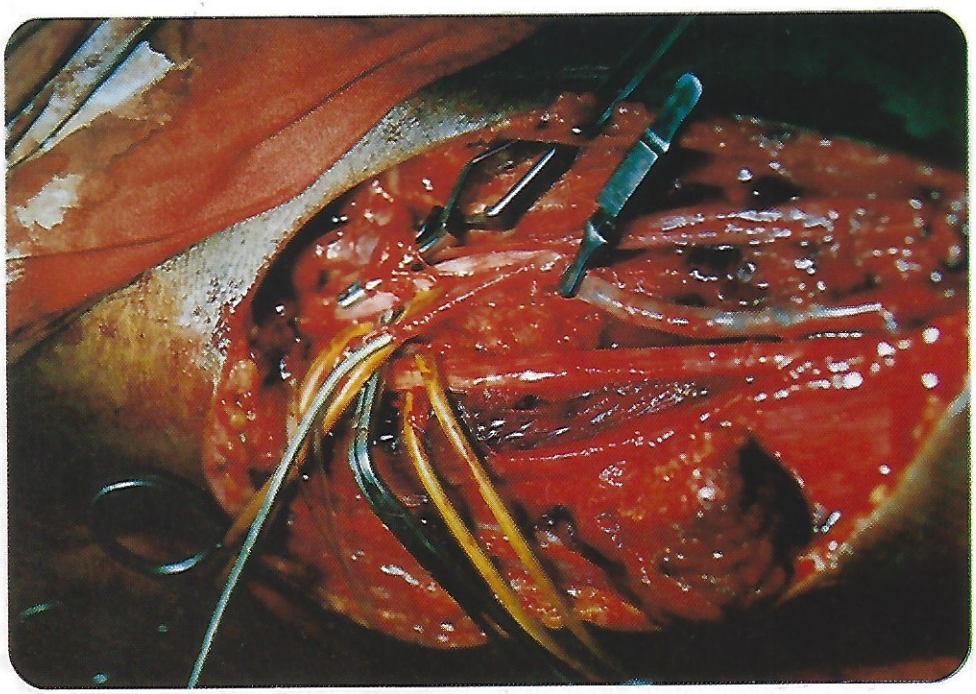

Fig. 13: Saphenoaxillary bypass grafting and neurolysis.

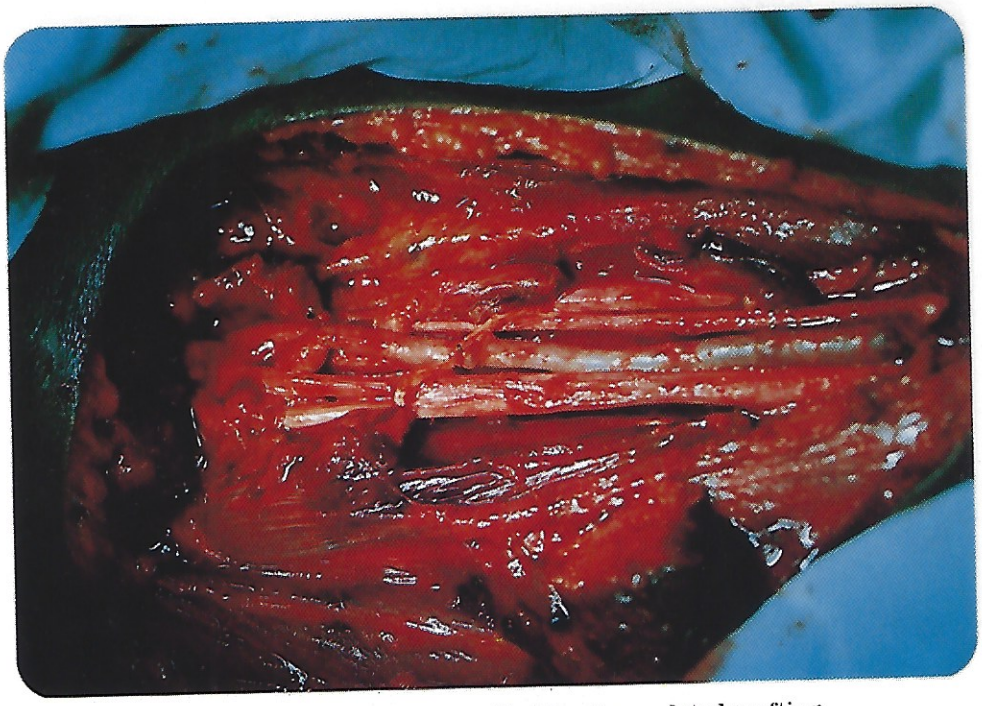

Fig. 14: LSV-Axillary and Median-Sural completed grafting.

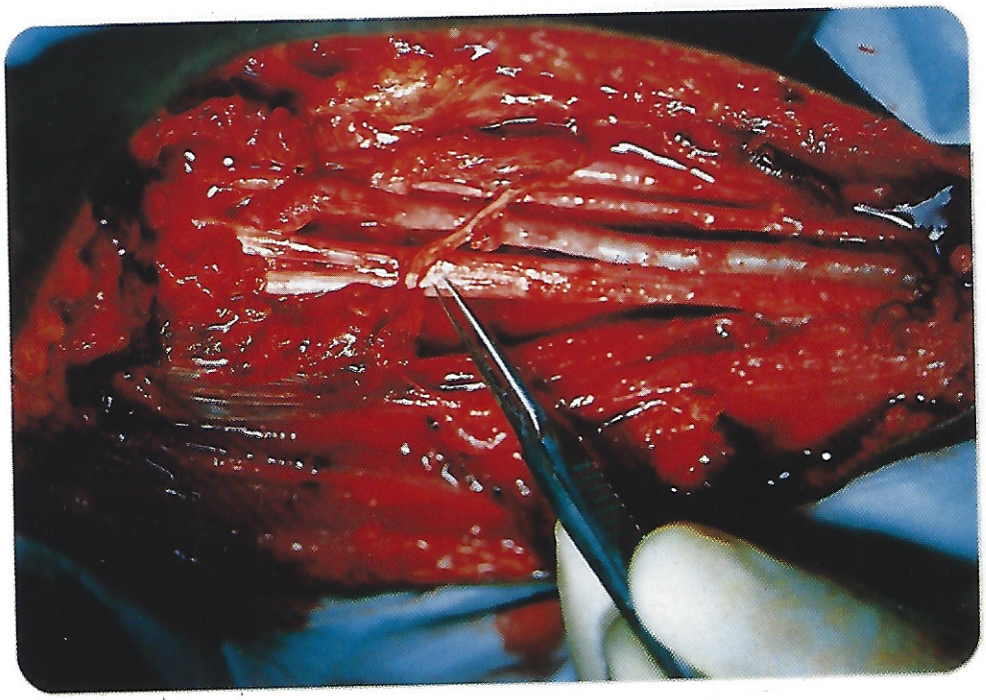

Fig. 15: Ulnar-Sural "Y" graft.

roma, it was quite clear that the bulk of the nerve had been damaged. The Median and the Ulnar nerves were dissected distally down to the upper third of the arm.

As the vascular grafting commenced, a long segment of the sural nerve was concurrently obtained from the elevated leg (Figures 11 and 12). The prepared reversed and Heparin-flushed measured segment LSV was anastomosed with 5/0 prolene to the proximal segment of the axillary artery stump using the parachute technique. Proximal vascular clamp was removed and applied to the distal end of the vein graft. A minute later, with pulsatile elongation of the graft under arterial pressure and after confirming haemostasis, the distal end was further trimmed to size, (Figure 11 and 12) allowing for some stretch during maximum abduction and grafted to the distal stump of the axillary artery (Figure 13). Clamps were released and distal arterial supply was seen to be restored adequately. Radial pulse had return palpably.

The Posterior Cord was now followed down from its divided end to the radial and axillary nerves. Both these branches were found intact. The neurotoma was excised and the ends freshened with a resultant gap of three cm. Two strands of the thickest part of the sural nerve were grafted using an operative microscope and 7/0 prolene (one interfasicular and four superficial sheath interrupted sutures) to both end of the posterior cord. The damaged end of the Ulnar nerve was resected with freshening of either end. The gap now stood at three cm. and this was bridged with two strands of sural graft using the side branch of the sural nerve in a" Y" fashion (Figure 15). The remaining piece of graft (3 cm.) was coapted with freshened end of the Median nerve without any tension under operative microscopic orientation (Figure 14).

Peroperatively commenced gentamicin and metronidazole were continued for five days and the patient was rapidly mobilized. Chest and limb therapy was commenced early. The upper limb was splinted at night (cock-up wrist splint and forearm back-slab splint in 90 degrees mid prone) and was passively mobilised during the day under the guidance of our physiotherapist.

A month postoperatively, he was discharged home whilst continuing with outpatient physiotherapy. Transcutaneous Erb's point stimulation and stimulation to the distal muscles were regularly carried out. No movements or sensation had returned but the periphery was warm, pink and Doppler confirmed pulsatile flow in main digital branches of the palmer arch.

Two months postoperatively, NCVs revealed no CMAPs from Erb's point stimulation but the patient described patchy return of sensations in the volar and the extensor aspects of the forearm.

A month later, (three months post operatively) visible finger flexion (grade 2/5) was present with some wrist flexion (grade 2/5) and elbow flexion (grade 2/5). Wrist extension and pronation were absent.

Five months post- operatively, finger flexions had improved to grade 3/5, elbow flexion to between (2/5-3/5). Arm abduction was possible to 30 degrees (grade 2/5). With gravity eliminated, wrist extension had commenced (early grade 2/5) with a comparable weak pronation (grade 2 /5).

Six months postoperatively, all of the movements had improved to grade 3/5 but the wrist extension had not progressed beyond grade 2/5. The patient was able to grip pens etc. in his hand. Patchy return of sensations to pin prick stimuli was present in the upper limb.

The patient remain continues to remain under our follow up programme and further improvement is likely.

In view of the good recovery of neurovascular functions for this stage of repair, this first case report of its kind in Pakistan, has been presented here.

DISCUSSION

Some of the effects of the combined neurovascular lesions in the region of the thoracic outlet and axilla are obviously immediate. Distal ischaemia and expanding haematoma warrant immediate vascular decompression and revascularisation procedures. In cases where haematoma becomes consolidated in the presence of recognised neural injury the extent of the nerve injury is exaggerated with compression of an expanding false aneurysm[1]. In patients with severe neurovascular injury presenting with ischaemia and neurogenic defict, it is not always possible to be sure if the neural injury is primarily caused by the offending weapons or if it is secondary to a vascular event such as expanding haematoma[2]. Exploratory surgery has therefore become an accepted mode of therapy for neurovascular salvage even when there is no frank distal ischaemia present, simply because prompt decompression of the haematoma very often

benefits the brachial plexus[2,3,4].

With penetrating trauma, such as gunshot wounds (as in this patient) or stab wound, the incidence of vascular laceration and transection is much higher than in pseudo-aneurysm formation[2]. The incidence of the axillary artery injury ranges (in the civilian and the military series) between 2.9-9%[5-7]. If pseudo-aneurysm is formed, despite the extensive scapular-profunda anastomosis, the risk of distal ischaemia from embolisation is substantial and frequently dramatic[8-13]. The collateral circulation here, is so rich that in many cases, the radial pulse is palpably present[14,15], therefore a high index of suspicion is necessary to recognise such injuries and arteriography must be generously undertaken for this site of injury. Concomitant venous injury may also be present. In a very large series[16], 40% of the patients with neurovascular injuries in the region of axilla were found to have significant venous injuries. Isolated venous injury is much less dramatic and may not produce easily detectable signs[2,16].

Following a gun shot wound of the axilla, the resultant false aneurysm is densely adherent to the brachial plexus and adequate vascular control requires a generous exposure of the vascular segment. This, very often means, control of the subclavian artery above the clavicle. Following aneurysmotomy, under vascular control, the bleeding orifices of the involved branches must be ligated from within the aneurysm meticulously. If the aneurysm is simply by-passed, the collateral vasculature will bleed into the isolated aneurysmal sac, recreating the aneurysm and reproducing the vascular progressive compression of the nerves. Therefore it is essential to control all these vessels and to excise the aneurysm as well. The end result is a much wider disruption of the artery than is angiographically

visualised, necessitating a vein graft[2,37] which should be left slightly lax, to allow for abduction - stretch[17,18]. Because the vein graft may adhere to the dissected nerves, it is practical to route the vein graft medially and away from the site of neural grafts. It may also be advantageous to ensheath the nerve anastomosis in a tube of non-absorbable material to protect it from tissue adhesions. Foreign body reaction is an important consideration to bear in mind in this respect. It is important to prevent development of reciprocal compression between the axillary artery and the brachial plexus and therefore the vein graft should be sited away from the brachial plexus as much as possible. In such lesions the adherent vascular graft "compresses" the nerves, producing neurological symptoms and the adherent nerves and nerve graft may compress the artery producing vascular symptoms[19].

When concurrent brachial plexus and thoracic outlet (or axillary) vascular injuries are present, the primary consideration is, immediate vascular salvage, followed by immediate brachial plexus reconstruction (if the situation of the patient allows it) or planned early neural reconstruction[20]. Immediate or early brachial plexus surgery is advantageous for a number of technical and physiological reasons. The traumatised nerve ends are easily identified, reaction of degeneration is absent, end-to-end direct nerve anastomosis may be possible, shorter grafts frequently suffices and if necessary neurotization can be performed using the IX Cranial nerve or intercostal nerves, with relatively greater ease[21]. This is in sharp contrast to the traditional teaching[22] that the outcome of spontaneous improvement is better than the results of surgically corrected lesions. Critical retrospective analysis of the same studies confirmed that only those patients spontaneously improved significantly in whom the neural lesion was surgically seen to be partial such as neuropaxia and axonotmesis (whilst the preoperative assessment had suggested a complete lesion of the nerve). Therefore the prior claims were spurious due to lack of proper preoperative assessment. The greater use of intra operative nerve action potential study to exclude "lesion in continuity", ready availability of the operating microscope and the recently emerging neurovascular expertise, have shifted the trend towards immediate or early combined neurovascular reconstruction for better functional salvage.

The current incidence of brachial plexus injuries associated with axillary arterial trauma is around 25%[3,6,23,24] and therefore both angiographic as well as neurophysiologic assessment must be supplemented with an orderly and exhaustive clinical examination, in stable patients[25].

If concimitant root avulsion of the brachial plexus from spinal cords is suspected then NMR (MRI), Metrizoate enhanced CT scan,

Myelography etc should precede formal BP exploration[26]. Spinal cord lesions are largely untreatable but for the freshly detected root avulsions neurotization can and should be performed for some functional restoration. A thorough neurological mapping should always be done to evaluate the full extent of the brachial plexus lesion. Evidence of

medullary involvement may come from presence of Horner's Syndrome and serratus anterior palsy associated with BP lesions. Paralysis of shoulder and elbow with preservation of scalene muscles suggests a liesion of C5 and C6. C8 and T1 root avulsions produce paralysis of the hand. A base line EMG should be performed and repeated after one month post operatively. Absence of denervation and fibrillation supports the contention that nerve recovery is in progress. This is an important evaluation. After three weeks of injury to the nerves, larger potentials called "sharp positive waves" usually develop in the denervated muscle.

Such waves are never present in the normally innervated resting muscle[26]. When spinal denervation is suspected (as in complete avulsion of the nerves from the spinal cord the use of myelogram is helpful but it is not an infallible test[27,28]. Preoperative somatosensory evoked potentials via median or ulnar nerve stimulation at the wrist can record potentials from the scalp overlying the contralateral hemisphere. This denotes an intact spinal path way of the sensory neurone indicating absence of total root avulsion. This test is even more reliable if intraoperative stimulation of the suspected nerve is undertaken[29,30].

Recognition of secondary nerve compression by an expanding aneurysm is very important. Deep pressure sense is still present in the tips of the fingers with aneurysmal nerve compression and is usually absent in the totally divided nerves[31]. In a very large series[31], Dunkerton reported a fair number of patients with secondary axillary pseudoaneurysms without an obvious nerve injury. He also reported that in his cases, vascular repair alone, did not resulted in significant functional recovery. Excision of the aneurysm and vein by-pass of the arterial segment when combined with the neurolysis of the brachial plexus, resulted in significant functional recovery. Others have supported this view[32].

If neurorrhaphy of the cleanly transected nerves can be performed without tension (this is rare), then this is preferably to nerve grafting[33]. However, easy coaptation is not usually possible. Any tension in the anaestamotic site is catastrophic for spouting axons. Intra neural fibrosis of the stump leads to fibril adhesions and therefore whenever there is the slightest doubt about tension, nerve grafting must be performed in preference to direct neurorraphy. The worst possible technique is to anastomose nerve grafts under tension[33]. Using an operative microscope the interfasicular anastomosis of the sural nerve graft gives good results when fibril orientation is maintained[33]

The overall result of brachial plexus repair depends on a number of factors which are very difficult to group together in a given patient for prognostic evaluation. The greater the time before reconstructive surgery, the more likely is the atrophy of the nerve stumps and irreversible damage to neuromuscular structures[34]. Lesions below the clavicle when surgically repaired give much better results than the supra clavicular lesions[35]. When distal lesions are present, the distance of the effectors from nerve repair site determines the outcome. If the repair site is closer to the effector, nerve grafting is followed by satisfactory result in 70-80% of the cases. Such lesions usually involve the suprascapular, musculocutaneous, axillary and radial nerves. If the repair site is far away from the effector, the results are correspondingly poor[35]. This is usually seen in the lateral and medial cords and median and ulnar nerves. Reinnervations of wrist and hand flexors may occur in some 60% of the cases but the recovery of intrinsic muscle is rare. Nevertheless reinnervation provides very useful protective sensibility in the median nerve distribution and this is extremely important[35]. Because of the widely demonstrated beneficial outcome of early neurovascular cervico-brachial reconstruction, there has been an increased awareness for the treatment of the components of the plexus[36].

In other words, it has come to be recognised that to repair the divisions of the plexus is just as important as the repair of the formed definitive nerves such as median and the radial nerves. Based on an intraoperative assessment, these "component" injuries should be as nearly reconstituted as they exist anatomically in the uninjured state.

ACKNOWLEDGMENT

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REFERENCES

1. **Raju S, Carner DV**. Brachial plexus compression. *Arch Surg 1981;* **40**: 116-75.

2. **McCready RA.** Upper extremity vascular injuries. *Surg Clin North Am 1988;* **684**: 725-40.
3. **Rich NM, Baugh, Hughes CW.** Acute arterial injuries in Vietnam - 1000 cases. *J Trauma 1970;* **10**: 359-68.
4. **Zelenock GR, et al.** Non penetrating subclavian artery injuries. *Arch Surg 1985;* **120**: 685-92.
5. **DeBaky MF, Simon FA.** Battle injuries in the arteries in the World War 2; an analysis of 2471 cases. *Ann Surg 1946;* **123**: 534.
6. **Drapanas T, et al.** Civilian vascular injuries: a critical appraisal of three decades of management. *Ann Surg 1970;*
7. **Batey NR, Makin GS.** Neurovascular traction injuries of the upper limb root. *Br J Surg 1982;* **69**: 35.
8. **McCready RA, et al.** Subclavian axillary vascular trauma. *J Vasc Surg 1986;* **3**: 24.
9. **Babar SMA, Khan AH.** Subclavian arterial and subclavian venous fistula. *Indian Pract 1988;* **42**: 727-32.
10. **Babar SMA, Usmani R.** Compressive vascular disorders of the upper limbs. *Pakistan Heart J 1988;* **21**: 34-8.
11. **Babar SMA.** Thoracic outlet syndrome. *JPMA 1987;* **37**: 308-8.
12. **Babar SMA.** Proximal brachial pseudoaneurysm. *Med Pract (Feb) 1990; 5-10.*
13. **Babar SMA.** Distal brachial pseudoaneurysm with neuropathy and arm claudication. *Pakistan J Med Res 1990;* **29**: 1: 36-50.
14. **Graham JM, et al.** Management of subclavian vascular injuries. *J Trauma 1980;* **20**: 537.
15. **Lim LT, Saletta JD, Flanigan DP.** Subclavian and innominate artery trauma. *Surgery 1979;* **86**: 890.
16. **Graham IM, et al.** Vascular injuries of axilla. *Ann Surg 1982;* **195**: 232..
17. **Alnot JY.** Infraclavicular lesions. *Clin Plast Surg 1984;* **11**: 127.
18. **Watelet J, et al.** Rupture of Subclavian artery. (Eng transl). *J Chir (Paris) 1976;* **112**: 37.
19. **Aruio JD, et al.** Reciprocal compression between the axillary artery and the brachial plexus. *J Cardiovasc Surg 1988;* **29**: 172-6.
20. **Nichols JS, Lillehei KD.** Nerve injury associated with acute vascular trauma. *Surg Clin North Am 1988;* **68**: 837-52.
21. **Magalon G, et al.** Emergency versus delayed repair of severe brachial plexus injuries. *Clin Orthop 1988;* **237**: 32-4.
22. **Kline DG.** Operative management of brachial plexus lesions. *Neurosurgery 1983;* **58**: 631-49.
23. **Kline DG.** Civilian gun shot wounds to the brachial plexus. *J Surg 1989;* **70**: 166-74.
24. **Rich NM, Hughes CW, et al.** Subclavian artery trauma. *J Trauma 1973;* **13**: 485.
25. **Borman KR, et al.** Civilian artery trauma of the upper extremity. *Am J Surg 1984;* **148**: 796-9..
26. **Leffert RA.** Clinical diagnosis, testing and electromyographic study in brachial plexus traction injuries. *Clin Orthop 1988;* **237**: 24-31.
27. **Heon M, Mylogram.** A questionable aid in diagnosis and prognosis in avulsion of brachial plexus component by traction injuries. *Conn Med 1965;* **29**: 260.
28. **Jelasic F, et al.** Functional restitution after cervical avulsion injury with typical myelographic findings. *Eur Neurol (Basel) 1974;* **2**: 158.
29. **Jones S.** Investigations of brachial plexus traction lesions by peripheral and final somatosensory evoked potentials. *J Neurosurg Psychiatry 1979;* **42**: 107.
30. **Jones SJ, Wynn Parry et al.** Diagnosis of brachial plexus traction lesion by sensory nerve action potentials and somatosensory evoked potentials. *Injury 1981;* **12**: 376.
31. **Dunkerton MC, Boome RS.** Stab wound involving the brachial plexus: a review of operated case. *J Bone Joint Surg [Br] 1988;* **70**: 566-70.
32. **Sodel L.** The results of surgical repair of brachial plexus injuries. *J Bone Joint Surg [Br] 1982;* **64**: 54-66.
33. **Millesi H.** Brachial plexus injuries: nerve grafting. *Clin Orthop 1988;* **237**: 36-42.
34. **Hentz VR, Narakas A.** The result of microneurological reconstruction in complete brachial plexus palsy: assessing outcome and predicting results. *Orthop Clin North Am 1969;* **1**: 107-14.
35. **Steven JH.** The classic brachial plexus paralysis. *Clin Orthop 1988;* **237**: 4-8.
36. **Seddon HJ.** *Surgical disorders of the peripheral nerves.* Baltimore : Williams and Wilkins, 1972: 174-98.
37. **DeRosa JP, et al.** Brachial plexus paralysis associated with subclavian artery rupture (Eng. trans.). *Ann Chir 1980;* **34**: 277.

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