Peripheral Neurovascular Injuries

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SUMMARY:

This prospective 5-year research study describes 46 male patients with peripheral neurovascular injuries and is the largest reported series from this region at the present time. High-powered rifle injuries accounted for 91% of the lesions. Clavio-axillary region sustained 76% of the injuries. 3 patients presented directly with primary neurovascular injuries whilst the remainder (93%) presented at an average delay of 19 weeks when the neurological deficit was deteriorating (84.7%) or had failed to improve (15.3%) spontaneously. The predominant vascular lesion was a false aneurysm of various sizes in 84.7% of the patients, whose progressive enlargement was thought to be aggravating the original neurological injury. The predominant neurological lesion was neurotmesis in 61% of the patients in one or more regional nerves and axonotmesis in 39% in whom the lesion was either static (39%) or was deteriorating (61%). In the emergency presentation, clinical and doppler evaluations were followed by one-stage neurovascular repair. Where the presentation was delayed, orderly investigations included electrophysiological, doppler and arteriographic studies in all patients. In the upper limbs, aneurysmectomy was performed for continued neurological decompression followed by revascularisation by long saphenous vein grafts. In the lower limbs, simple aneurysmtomy was followed by long saphenous vein grafts. External and internal neurolyses were performed for the involved segments of the nerves. Nerve grafts were performed using the harvested sural nerve. An operating microscope was used for group fascicular and epineural repairs. In cases where neurological reconstruction must be deferred, appropriate nerve tagging etc. must be performed to facilitate secondary nerve repair. In our series the early results were good, for both the vascular as well as neurological lesions but poor re-attendance on subsequent follow-ups has seriously affected the interpretation of the final outcome on a long term basis. It is hoped that this can be rectified through patient-education and better colleague co-operation in the future.

CLINICAL MATERIAL AND METHODS

Patient Population

Fortysix patients were included in this study which began in 1986. Of these 3 presented directly to our Centre as emergencies within 6 hours of trauma and underwent a combined one-stage neurovascular reconstruction immediately after initial resuscitation. Pre-operative Neurophysiological tests or angiography was not performed in these patients.

The remainder (43) presented with varying delays exceeding two months and came to us after initial vascular haemostasis. These patients underwent a more comprehensive assessment.

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Investigations

A doppler flow study was performed in all patients using a combination of clinical and doppler evaluation. Intra-arterial DSA was performed in 11 patients.

A base-line EMG was obtained for initial documentation. Nerve conduction study was undertaken in all these patients and reassessed a month later to see any evidence of spontaneous improvement. For lesions of brachial plexus (25 patients), associated with delayed vascular lesions, such as aneurysm, both surface electrode (utilising a higher voltage) and needle electrodes (utilising a lower voltage) were used to assess neurological deficits. The surface electrode was placed at the Erb's point for stimulation and the needle electrodes were positioned to the proximal segment of the injured

nerve percutaneously using random targets in an attempt to obtain an action potential.

Surgical Procedures

In the upper limb, aneurysmal capsule was totally excised for decompression and end to end anastomoses were carried out using reversed long saphenous vein (LSV).

In the lower limb aneurysms, a simple aneurysmotomy was followed by appropriate LSV graft anastomoses.

Nerve injury was treated either by neurolysis (external and internal), sural nerve interposition graft or a combination of both. External neurolysis consisted of external decompression and release from adjacent tissue, usually the aneurysmal capsule. Internal neurolysis consisted of saline injection using an ultrafine needle throughout the course of the involved nerve wherever perineurial adhesion was seen under magnification. In some cases where the injured nerve was grafted, the contused functioning nerve was internally neurolysed.

Before coaptation, orientation of the nerves was made on visual magnified inspection of arterial arcade and bundle alignment.

At least two interrupted group fascicular sutures per graft were used to maintain orientation followed by circumferential interrupted sutures of the epineurium.

Enough sural nerve length was maintained for elongation during postural stretch, to prevent anastomotic tension.

Vascular conduit was routed well away from the bed of sural graft and the area of neurolysis.

Antibiotics (Zinacef and amoxycillin) were continued until removal of sutures and prophylactic heparin (5000 units subcutaneously 12 hourly) was used until mobilisation became possible.

FINDINGS AND RESULTS

Fortysix patients entered this study between 1986 to 1991. All of these were males. Ages ranged between 17 to 60 (mean=38.5). The three patients who presented as emergencies underwent an immediate one-stage neurovascular repair with clinical and doppler evaluation whilst the remainder presented with an average delay of 19 weeks (range=6-32 weeks) and underwent formal investigations and subsequent surgery.

Injuries were caused by shotgun in 2 patients, low velocity hand pistol in 2 patients and high powered rifle in 42 patients. Firing range averaged 30 feet.

TABLE 1

SITE OF INJURY AND LESIONS

SITE	NO.	VASCULAR LESIONS	NEUROLOGICAL LESIONS	
			Axonotmesis	Neurotmesis
Neck	1	Caroticojugular AVF	1 (X)	-
Clavio-axillary	13	S/C II aneurysm	6 (BP)	7 (BP)
	1	S/C II laceration	–	1 (BP)
	1	S/C Vein+ Innominate Vein lac	–	1 (BP)
	6	S/C III aneurysm	3 (BP)	3 (BP)
	5	Prox Axillary anneurysm	-	5 (BP Nerves)
	9	Dist Axillary aneurysm	4	5
Lower arm	2	Brachial aneurysm	2	-
Groin	1	SFA laceration	-	1 (Femoral N)
	1	SFA aneurysm	-	1 (Sciatic N)
Upper thigh	1	SFA aneurysm	-	1 (Sciatic N)
	1	SFA aneurysm	-	1 (Tibial N)
Knee	1	Popliteal aneurysm	-	1 (Lat Pop N)
	1	Popliteal aneurysm	-	1 Lat+ Med Pop
	1	Popliteal AVF	1 Lat Pop N	-
	1	Popliteal art laceration	1 Lat+ Pop	-

X = Vagus Nerve
S/C II = Subclavian artery (second part)
AVF = Arteriovenous fistula
SFA = Superficial femoral artery
BP = Brachial plexus

76% of the injuries were sustained in the Clavioaxillary region (See Table 1) and this had resulted in a high incidence of Brachial Plexus element neurotmesis. In this group, S/C II was involved in 40% of the cases. The incidence of Upper limb involvement was higher than the lower limbs by a factor of 4.6 and this was a reflection of the nature of trauma. Criminal firearm injuries are normally targeted principally to the head and neck and chest regions, whereas in road traffic accidents (RTA), a fair number of injuries involve the neurovascular structures of the lower limb. This was not found to be the case in our study. There were numerous other patients treated by the author with major vascular and neurological trauma in various parts of the body. Since this particular study deals with combined neurovascular trauma, I have excluded those other cases.

The subclavian aneurysms in this series were fairly large. In the lower limbs, only the SFA aneurysms were large whereas the remainder were small.

The BP injuries were rather complex. Table 2 gives a general view of the overall injuries but does not describe this complex nature. For instance, in the axonotmetic group, the large sized and densely adherent aneurysms had produced diffuse neuropathy of a fairly large segment of the various elements. Thus although there were only 13 patients in this group, several patients had more than one element of his plexus involved and hence the disparity of figures.

Similar pattern was present in the Neurotmetic group. Another extreme example of this complex injury was the single patient with total disruption of his cords, in whom during arterial reconstruction, the C5 root was inadvertently divided.

Majority of the aneurysms were located in clavioaxillary region (82.5%). Most of the aneurysms were easily detectable (See Table 3) by inspection of palpation (87 %) but some of the deeper and small sized aneurysms of the 2nd and the 3rd parts of S/C artery, existed behind the clavicle and were not visibly or palpably identified (12.5%) and of these, 2 were suspected on the basis of a transmitted distal bruit whereas the remainder (3) were subtotally thrombosed and were detected only on arteriography.

TABLE 2

Type of Vascular Lesion	Type of Neurological Lesion	Type of Brachial Plexus Lesions				
		Roots → Trunk	Trunk → Div	Division → Cord	Cord → Nerve	Nerve
Aneurysm	Axonotmesis (13)	6	2	6	1	3
Aneurysm	Neurotmesis (20)	5	2	4	2	10
Laceration	Neurotmesis (1)	1	1	1	-	-
Laceration	Neurotmesis (1)	1(C5)	-	-	1(all)	-

TABLE 3

CLINICAL FEATURES OF ANEURYSMS

Blood Vessel Involved	No.	Lump Visible Or Palpable	Lump Neither Visible Nor Palpable	Local Bruit	Distal Emboli/ Ischaemia	Aneurysmal Thrombosis
S/C II	(13)	9	4	10	5	6
S/C III	(6)	5	1	6	1	4
Prox Axillary	(5)	5	-	5	1	1
Dist Axillary	(9)	9	-	9	4	4
Brachial	(2)	2	-	2	2	2
Sup Femoral	(3)	3	-	3	2	2
Popliteal	(2)	2	-	2	-	-

S/C II= Subclavian artery (second part)

S/C II (38.46%) and Distal Axillary (44.4%) aneurysms yielded the highest incidence of peripheral ischaemia and embolisation.

There was a positive correlation of intra-aneurysmal thromboses (47.5%) with distal ischaemia or embolisation (37.5%). This was considered significant.

Because of the gross aneurysmal adhesions with the involved vessel, aneurysmectomy was performed for sustained decompression in most patients with

SURGICAL TREATMENT OF VASCULAR LESIONS

TABLE 4

Lesion	Aneurys-mectomy	Aneurys-motomy	Vein Graft	E-E Anast	Both Ends Ligated
Aneurysm S/C II (13)	13	-	13	-	-
Laceration S/C II (1)	-	-	1	-	-
S/C + Innomm Vein Laceration	-	-	1	1	-
Aneurysm S/C III (3)	3*	-	3	-	-
Prox Axillary Aneurysm (5)	5	-	5	-	-
Dist Axillary Aneurysm (9)	7	2	7	2	-
Brachial Aneurysm (2)	2	-	-	2	-
Caroticojugular AVF (1)	-	-	-	-	-
Laceration SFA (1)	-	-	1**	-	-
Aneurysm SFA (3)	-	3	1	2	-
Popliteal Aneurysm (2)	-	2	1**	1	-
Popliteal AVF (1)	-	-	1**	1	-
Popliteal Laceration (1)	-	-	-	1	-

S/C II = Subclavian artery (second part)
AVF = Arteriovenous fistula
SFA = Superficial femoral artery
* = Partial aneurysmectomy
** = Vein patch

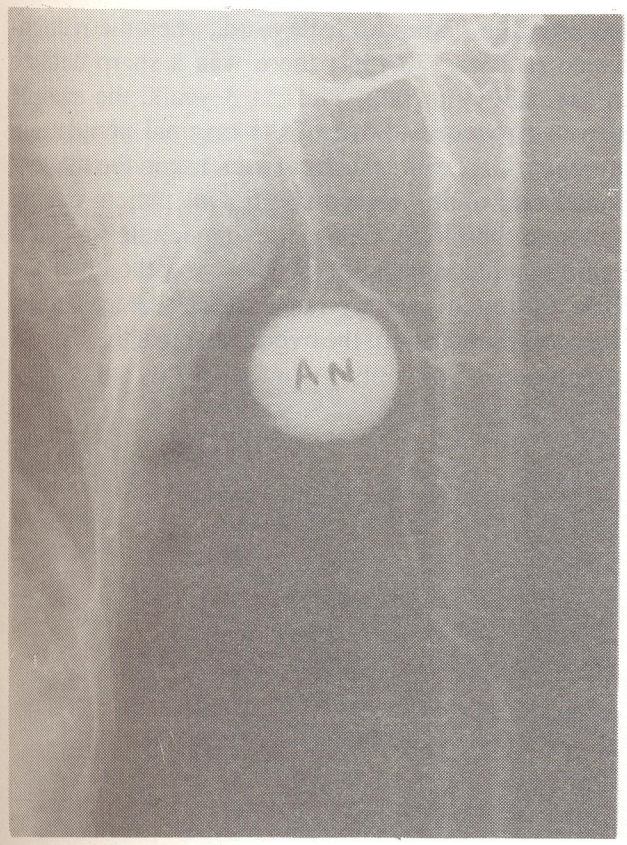

Fig. 1. Axillary Aneurysm with BP Nerve Lesions.

upper limb trauma (See Table 4). Total aneurysmectomy was performed in 27/32 patients (84.3%) with upper limb aneurysms. Since total aneurysmectomy was not possible technically in 3/32 such patients (9.37%) with S/C III aneurysms, a partial aneurysmectomy was performed with adequate decompression. In 2 patients with Distal Axillary aneurysm, the capsule fully collapsed on aneurysmotomy and therefore it was not considered necessary to proceed to formal aneurysmectomy.

The single neurorrhaphy was performed for an iatrogenic injury to Musculocutaneous Nerve which was mobilised and repaired end-to-end.

The single patient with Vagus Nerve lesion (See Table 5) refused surgery for Carotico-jugular AVF and persists with the neurological lesion and with

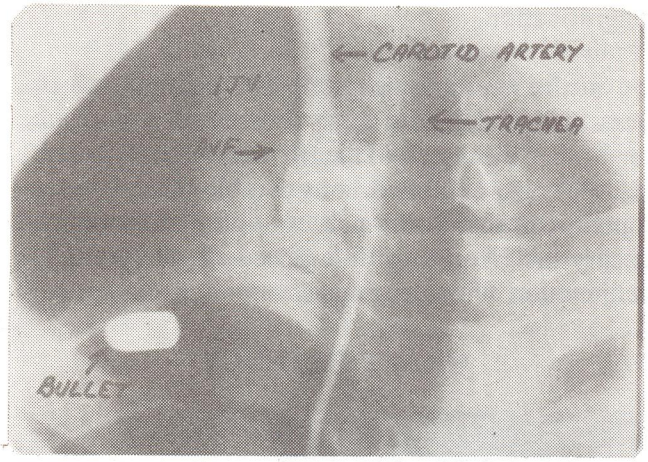

Fig. 2. Carotico Jugular Arteriovenous Fistula with X Nerve Lesion.

SURGICAL TREATMENT OF NEUROLOGICAL LESIONS

TABLE 5

Type of Lesion	Site	Expectant Observation no Surgery	External Neurolysis	Internal Neurolysis	Sural Graft	Neuro-rrhaphy
Axonotmesis	Upper Limb (15)	-	15	15	-	-
	Neck (1)	1*	-	-	-	-
	Lower Limb (2)	-	2	2	-	-
Neurotmesis	Upper Limb (22)	-	8	8	22	1
	Lower Limb (6)	-	2**	-	6	-

* = Vagus Nerve partial injury ** = Sciatic nerve

progressive cardiac failure and remains under our follow up.

In the arm there was considerable aneurysmal adhesion present contributing to neuropathy and in all (10) patients with Axillary artery aneurysms, in addition to sural grafts for neurotmesis, the distal nerves were additionally neurolysed after ascertaining their anatomical intactness.

For axonotmetic lesions (17 patients), often involving more than one element in the given BP, a combination of external decompressive neurolysis (using sharp dissection) and internal neurolysis (by saline injections with microneedles) were done under magnification.

In the neurotmetic group (28 patients) all patients had sustained a total division of at least one (or more in the region of the BP) nerve but 10 patients (35.7%) also exhibited considerable adhesion-neuropathy of the distal nerves with the associated aneurysms requiring interposed sural grafts in conjunction with neurolysis.

GRAFT PATENCY

TABLE 6

F/U Time	Patients attended Total=45	Patent Graft	Thrombosed Graft
6 Weeks	44	42	2
3 Months	40	38	2
1 Year	31	28	3
2 Years	16	15	1
3 Years	7	6	1
4 Years	6	6	-
5 Years	10	9	1

F/U = Follow Up

Of the 46 patients vascular reconstruction was performed in 45. At 6 weeks, 2 patients (distal axillary artery end-to-end anastomosis and proximal axillary artery vein graft) revealed thrombosis (See Table 6). In both these patients there was wound sepsis present (patency rate=95.45%).

This trend continued throughout the follow-up but after the 12-month follow-up, when the reattendance rate was 70.45%, there was a sharp decline in the follow-up rate. Thus at 3 years, the sample size had reduced to 1/6 of the original population. At 5 years, there was a slight increase in the reattendance rate owing to more efficient patient-tracing but even then a 22.72% reattendance rate was poor from an objective point of view and therefore the 90% exhibited patency rate cannot be regarded as truly representative of the overall result of the original population of patients included in this study.

OVERALL RESULTS OF NEUROLOGICAL RECONSTRUCTION AT 1 YEAR

TABLE 7

Lesion	Recovered to Grade III or Better	
	Neurolysis	Sural Graft
BP Elements with Aneurysm (33)	9/13=69%	13/20=65%
BP Elements with Vascular Lacerations (2)	-	2/2=100%
Distal Arm Nerves with Aneurysm (2)	2/2=100%	-
Lower Limbs (8)	2/2=100%	3/6=50%

BP = Brachial Plexus

The results of neurological reconstruction were assessed at 1 year (See Table 7). The criteria of recovery were sensory or motor improvement to grade III or better. Nerve conductions were found very unreliable for BP lesions (Even with improvement from motor Grade "O" to Grade "IV"), in some of the patients, Conduction defects perisisted, Nerve Conduction tests were more reliable with distal reinnervation.

The results of neurolysis for BP elemental injury was 69.2% at 1 year with a reattendance rate of

70.45%. At the same time, the result of sural graft was 65%.

Statistical observations were made on a very small minority of patients with too small a sample size to yield an accurate information, Since this is an ongoing study, it is hoped that these patients can be traced in time to assess results in a realistic and statistically reliable way.

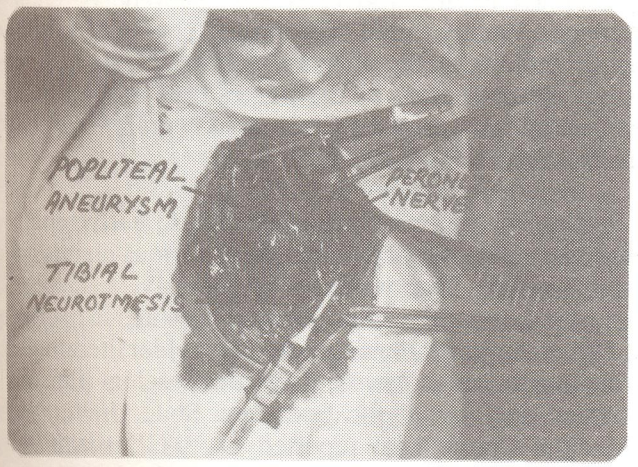

Fig. 3. Popliteal Aneurysm with Tibial + Peroneal Nerve Lesions.

The lower limb results were 50% for sural grafts and 100% (2/2) for neurolysis. The sample size is as can be seen, too small to reliably generalise the findings.

Discussion

Bullet wounds cause primary track formation which results in disintegration of soft tissue in the tunnel of the track. This is also associated with considerable regional disruption of the neurovascular structures caused by the secondary shock wave effect. The development of an aneurysm is a slow process caused by leakage of blood in the form of a haematoma after the arterial wall has ruptured from penetration or from the remote shock wave effect. The communication of the haematoma with the parent artery via an ostium results in dynamic intermittent pulsatile stretch of the surrounding tissue. This results in condensation of the surrounding tissue into a rigid wall (pseudoaneurysmal capsule or false capsule). With the acquisition of a capsule, the aneurysm stretches intermittently, producing local compressive damage to the adjacent nerves such as those of the BP. The size of the aneurysm is related to local manifestations of complications[1]. European and North American literature does not report long term effects of untreated giant aneurysms because these tend to be treated much earlier[2]. Most large sized aneurysms and turbulent small sized aneurysms tend to develop multiloculations due to adhesive organisation of intrinsic thrombi and their attachment to the false capsule. This phenomenon produces septa which compartmentalises the cavity and some of these loculi become fully thrombosed and some remain in continuity with the parent vessel dynamically. Many of these seemingly stable aneurysm discharge microthrombi into the distal circulation[3,4,5,6].

After division of a peripheral nerve, Wallerian degeneration occurs throughout the distal segment and over a few mm proximal to the injury. The axons disintegrate, myelin sheaths fragment and the debris is removed over a period of several weeks. Sprouting and distal migration of axons in the proximal stump begin after a day or so. If the ends of the nerve are in opposition, axons cross the site of injury and have

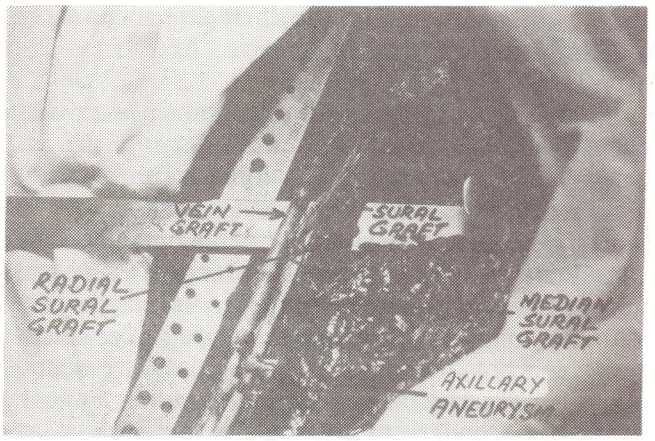

Fig. 4. Axillary Aneurysm with BP Lesion.

been identified distal to the repair after only seven days[7]. Experimental evidence suggests that the rate of regeneration is between 2 and 4 mm per day, though 1-2 mm per day corresponds better with clinical observation in man[7]. The ends of the cut nerve unite by forming a scar which the regenerating axons have to cross.

If the nerve ends are not in contact, the regenerating axons mingle with proliferating Schwann cells and fibroblasts to form a neuroma on the proximal stump. A similar swelling without axons is formed at the distal stump. The axons within the neuroma are sensitive to mechanical deformation, producing painful paraesthesia if touched.

Sedon[8] classified the degree of injury as neuropraxia, axonotmesis and neurotmesis.

In neuropraxia, there is a block of the neurological conduction present. The axons remain intact and no reaction of degeneration occurs. Recovery therefore follows after some weeks and is excellent.

In axonotmesis there is axonal degeneration present and the supporting structures such as the perineurium and the major part of the epineurium are intact. The recovery after axonotmesis is usually good and follows spontaneously after three weeks plus.

In neurotmesis, the entire nerve is usually disrupted and there is considerable distal degeneration present. A small degree of proximal reaction of degeneration is also present. No spontaneous recovery occurs. A few weeks after this injury, the nerve is enveloped in a scar. Attempts to dissect out and repair the divided fascicles at this stage carry a definite risk of damage to intact fibres.

Some of the effects of the combined neurovascular lesions are obvious immediately. Distal ischaemia and expanding haematoma warrant immediate vascular decompression and revascularisation procedures. In cases where the haematoma becomes consolidated in the presence of a recognised neural injury, the extent of the neural injury is exaggerated with the compression of an expanding false aneurysm[9]. It is not always possible to assess if the neural injury is primarily caused by the offending weapon or if it is secondary to a vascular event such as an expanding haematoma[10]. 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 involved nerves[10,11,12].

The surgical approaches for emergency vascular surgery varies according to side and to the site of the injury. If bleeding is aggressive, proximal and distal control is essential prior to the exploration of the involved segment. If life-threatening emergency does not exist then arteriography should always be performed to properly evaluate a vascular injury[13].

When nerve injuries are present, one of the two courses of action can be adopted. The repair can be deferred for a later date and in this event the identified nerves are properly tagged and clipped to an aponeurosis etc. away from the site of a vascular graft. This course of action should be taken if the condition of the patient is unlikely to withstand the prolonged exploration or if the other nerve ends cannot be easily found. The second course of action is to undertake the primary repair (clean nearly surgical transection of the nerves[9,12,14] and for this a preliminary sural or antebrachial graft must be harvested from one or both legs.

One of the principal objections to early repair has been an inference drawn from various claims of spontaneous improvement. Obviously a simple neuropraxic injury is likely to improve in due course of time. Amongst the major relevant older literatures relating to gunshot wounds of the brachial plexus, critical analysis of the Louisiana State University Medical Centre trial reported in 1983[15] seems to suggest that the conservatively treated patients improved because they had sustained a partial lesion (neuropraxia or axonotmesis). Even amongst the group of patients who did not improve spontaneously and were subjected to exploration, only 21% of the patients were found to be suffering from complete or near complete lesions whilst the remainder exhibited only partial lesions. This was not an exceptional finding and many similar reports suggested the same features. With improved peroperative assessment, use of operating microscope, stimulations for Nerve action potential to exclude lesions in continuity etc. the trend has shifted towards an immediate or early repair of the nerve lesions.

Another important change has been to recognise "lesions-in-continuity". These seemingly uninterrupted injuries are produced by blunt trauma or stretch but may occur with gunshot wounds as well. When peroperatively tested, these lesions show electrophysiological deficits and indicate the necessity to resect and graft or resect and repair the lesion[16,40]. If action potential is recorded across the injured segment then no further intervention beyond a neurolysis may be necessary and the axonotmetic lesion will improve in due course of time. If action potential is not evident then in a fresh lesion intrasheath disruption has probably occurred and in

a 8-12-week injury either no axonal growth is taking place or misguided growth is present. Neither will result in function-recovery and surgical repair is necessary[16]. Where possible for lesions in continuity local resection and end to end anastomosis[16,40] is preferable (successful functional recovery occurs in about 60% of cases) to nerve grafts (success rate is just less than 50%).

Yet another major policy shift has been towards the individual assessment and treatment of the "components" rather than of the overall plexus. An intraoperative NAP study may show a small component to be electrophysiologically injured. Many studies[17] involving resection of intraoperatively detected damaged components have confirmed either histological neurotmesis or Sunderland Grade IV lesions. Such lesions, if left untreated on macroscopic appearance of "continuity", will not recover spontaneously or even with neurolysis. Thus the surgeon cannot positively rely on simple preoperative clinical judgment or even on finding the nerves macroscopically intact and in continuity. However, it must be emphasised that no single assessment provides criteria for predicting functional recovery in a given patient[16].

In gunshot wounds, the disrupted artery can only rarely be primarily end-to-end repaired[18] and a graft (usually a long saphenous vein autograft) is necessary. If a major peripheral vessel is injured, it should be veinpatch repaired if possible. For concomitant arteriovenous injuries, under clamps, the vein should be repaired or grafted first, followed by the arterial reconstruction[19,20,21].

Following a gunshot wounds of the axilla and the groin, the resultant false aneurysm is densely adherent to the Brachial Plexus and to Femoral or Sciatic Nerves, adequate vascular control requires a generous exposure of the vascular segment. This very often means control of the Subclavian artery above the clavicle and External Iliac artery in the pelvis[39]. Following aneurysmotomy, the bleeding orifices of the involved branches must be ligated from within the aneurysm meticulously. If the aneurysm is simply bypassed, the collateral vasculature will bleed into the isolated aneurysmal sac, recreating the aneurysm and reproducing progressive vascular compression of the nerves. Therefore it is essential to control all these vessels. The end result is a much wider disruption of the artery than is angiographically visualised, necessitating a vein graft[10,22] which should be left slightly lax, to allow for postural-stretch[21,23]. Because the vein graft may adhere to the dissected nerves, it is practical to route the graft away from the site of neural grafts.

Autogenous long saphenous vein should be used in preference to synthetic graft because of better patency results[32,39]. The intact segment of the artery should not be unduly dissected or mobilised for fear of sacrificing valuable collateral branches. Autogenous vein grafts provide the best results in potentially contaminated vascular injuries[11,19,32,33,34,35]. Synthetic graft is often thrombosed in presence of an active infection.

Recognition of secondary nerve compression by an expanding aneurysm is very important[41]. Deep pressure sense is still present in the tips of the fingers and toes with aneurysmal nerve compression and is usually absent in totally divided nerves[24]. In a very large series[24], Dunkerton reported on a large number of patients with secondary Axillary pseudoaneurysms without an obvious nerve injury. He found that in his cases, vascular repair alone, did not result in significant functional recovery. Excision of the aneurysm and vein bypass of the arterial segment when combined with neurolysis of the affected nerves, resulted in significant functional recovery. Others have supported this view[25,41].

If neurorrhaphy of the cleanly transected nerves can be performed without tension (this is rare), then this is preferable to nerve grafting[26]. However, easy coaptation is not usually possible. Any tension in the anastomotic site is catastrophic for the sprouting axons. Perineurial fibrosis of the stump leads to fibril adhesion and therefore whenever there is the slightest doubt about tension, nerve grafting must be performed in preference to direct neurorrhaphy. The worst possible technique is to anastomose nerve grafts under tension[26]. Using an operating microscope the interfascicular anastomosis of the sural nerve graft gives good results when fibril orientation is maintained[26].

An aneurysm of a major artery of the limb such as the Femoral and the Subclavian is potentially emboligenic and is therefore dangerous[40].

Following a gunshot injury the combined nature of the injury may be difficult to distinguish. In the shoulder girdle, because of the extensive scapulohumeral and other collateral vascular anastomoses, in some cases, even the radial pulse may be palpable following total disruption of the subclavian ar-

tery[40,41,42]. Therefore the presence or absence of a distal pulse is not a strict criterion of absence or presence of vascular injury[27, 28].

Angiography must be considered whenever arterial injury is suspected. The indications of angiography should include the following factors:

1. If the mechanism injury suggests a strong probability of an arterial injury then a preoperative or a peroperative angiography must be performed.

2. If acute arterial insufficiency is present.

3. If an aneurysm, fistula, bruit, large haematoma is clinically present.

If the condition of the patient is such as to preclude prolonged anaesthesia, then at the time of vascular salvage, identification-tagging of the divided and damaged nerves should be performed. Post-operatively, the neurological deficit is correlated with current electrophysiological status, to allow for a better planning of the subsequent delayed primary repair of the damaged nerves.

When patients present late (after initial haemostasis), in addition to comprehensive clinical neurovascular assessment, both vascular as well as appropriate neurological investigations must be undertaken for appropriate overall assessment of the injury. This includes doppler and duplex ultrasounds, venography, angiography, nerve conduction velocities, EMGs, evoked potentials and if indicated, CT scans, MRI (NMR), Myelography etc.

Primary or early nerve repair, when feasible, is superior to secondary repair[29,30]. The main advantages of such a primary repair are:

- anatomical structures are easily identifiable and can be preserved more reliably.

- surgical dissection is easy, there being no reaction of degeneration or scar tissue present, the nerve ends can be more easily found.

- because nerve end retraction has not pathologically taken place, there is a possibility of an end-to-end nerve anastomosis (neurorrhaphy) without tension.

- if a graft is needed, a shorter length may suffice.

Kline[31] has reported on a large series of 141 patients with nerve injuries over a period of eighteen years (1968-85). In his series, in the clinically "complete" lesions, the results of neurolysis alone were good in 40 out of 44 patients. With direct primary end-to-end anastomoses, good results were obtained in 13 out of 20 patients and with interposition nerve grafts, good results were obtained in 51 out of 96 patients. In the same series where the nerve lesions clinically were "incomplete", good results were obtained by neurolysis in 44/47 patients, by direct primary end-to-end suture in 5/6 cases and by interposition grafts in 2/2 patients. In the same series six subclavian aneurysms were also simultaneously treated.

Present day assessment of nerve functions includes demonstration of active muscle contraction in the distribution of the element distal to the site of injury, a compound muscle action potential (cmap) evoked through the site of injury or a direct nerve action potential (nap) seen after proximal stimulation. This also allows determination of an intact pathway distal to the area of injury. Sequential testing may lead to identification of a nerve segment which does not transmit such stimulation. This injured site must be bypass-grafted or must recover by axonal growth if functional recovery is to occur at all. Using such a technique, the elusive "lesion-in-continuity" can be easily detected and be surgically treated.

Pre-operative electrophysiological assessment and visual inspection are not totally reliable. In clinical situations, where clinical and preoperative physiological studies suggest a total lesion but intra-operative stimulation conducts the stimulation through the injured site producing a recognisable phenomenon (muscle contraction or cmap), then the nerve should be spared resection and stands a good chance of spontaneous recovery in 3-6 months.

When the nerve injury is clean and almost surgical, primary repair by neurorrhaphy gives the best results provided the anastomosis is tension-free and the nerve orientation is anatomically correct.

A good nerve repair requires time, experience and proper equipment. Unless the surgeon is skilled at neurovascular repairs, is rested and can conduct an unhurried repair, it is best to close the wound after nerve tagging and, then to arrange delayed primary nerve repair. This can be done as early as in 2-5 days when there is evidence present of infection-free wound healing. A rushed repair by a tired surgeon

using inadequate equipment does the patient considerable disservice. Unlike a vascular repair, several months later it finally becomes clear that the nerve repair has failed and re-exploration must be considered. Re-exploration will have lesser chances for functional recovery. In other words, the first repair must be the best one.

Satisfactory nerve repair can only be done when the following criteria are fulfilled[40]:

- proper excision of the damaged segment of the nerve has taken place.
- correct axial alignment of the bundles has been done.
- accurate fascicular apposition is achieved.
- atraumatic technique using atraumatic fine needle and suture (6/0-10/0 prolene) are used.
- No tension is present at the site of anastomosis.
- haematoma is prevented at the site of anastomosis.

There are two basic techniques available for nerve repairs; epineurial repair and group fascicular repair:

Epineurial repair:

Sutures are applied to the epineurial sheath. This is the traditional method of nerve repair and is appropriate for primary nerve sutures and especially for the partially transected nerves. A good orientation and a tension-free vascularised bed are essential.

Group fascicular repair:

Sutures are placed in the perineurial tissue adjacent to a group of fascicles. The epineurial sheath may need to be reflected 1-2mm from the cut ends to expose the fascicular groups but extensive dissection of the individual fascicle bundle is unnecessary and will probably promote fibrosis. One or two sutures (8-10/0 gauge) of polyamide) are sufficient for each group. The epineurial sheath can be separately sutured to support the perineurial repair. This technique is employed for nerve grafting.

Numerous experimental studies have failed to show a consistent advantage of one technique over the other, provided the repairs are performed with the same degree of precision.

Good orientation of fibres is essential for the recovery of functions. Orientation can be obtained on the basis of visual matching of fascicular pattern or vascular arcade under magnification (operating microscope). Histochemical staining of choline acetyltransferase in thin slices to show motor or sensory fascicles[36] is not easily reproduceable or popular because the thin slices are studied after staining and the procedure takes more time than a surgeon can wait (cf frozen sections). Selective electrical stimulation of the bundles using a microelectrode can be used to orientate bundles but this is more useful in direct end-to-end anastomoses. Of these methods, the visual matching method is the most popular one.

Encasing the nerve anastomosis in a silastic tube carries with it no special advantages and in fact prevents secondary neocollateralisation of vessels which is so necessary for continued regeneration of axons. A good vascular bed on a muscle sheath is much more preferable.

The overall results of nerve repair depend on a number of factors:

-the earlier the time of repair from the time of injury, the better are the results[37].

-shorter distance of the effectors from the sites of repair is associated with better functional recovery[38]. Longer distances from the effector give correspondingly poorer results.

Tinel's sign (paraesthesia occurring in the anaesthetic limb on persussing the site of injury) is used to clinically judge the rate of recovery. As recovery proceeds the Tinel's sign also proceeds distally, usually at the rate of 1 mm per day.

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