NERVE INJURIES: DIAGNOSIS, EVALUATION AND MANAGEMENT
APPROACH

- Fundamentally clinical:

Through History

Neurological Ex.  Investigations
• **Questions to be answered** –

- Mechanism of injury?

- Severity?

- Focal Vs diffuse injury?

- Nerve element lacerated or in continuity?

- Complete or incomplete injury?

- Static, improving or deteriorating?

  Ø Worsening may be d/t – **Hematoma, Compartment syndrome or pseudoaneurysm**
HISTORY

• High index of suspicion
  - In 5% of pts with poly trauma

• Rule of thumb – To exclude injury, most distal part of nerve should be in function

1- Pain: may be d/t –
  - Disuse phenomenon
  - Compression
  - Autonomic dysfunction
  - Neuroma Formation
2- Sensory Loss

- Anesthesia
- Hypoesthesia
- Hyperesthesia
- Dysesthesia
- Allodynia

3- Motor Deficit

- Location
- Severity
- Change in motor function loss – Motor march

4- Autonomic dysfunction

- Sweating – loss or increased
- Coolness
- Cyanosis
NEUROLOGICAL EXAMINATION

No alternative to a proper and through neurological ex.

: General Principal :

1- Proper exposure

2- Comparison

3- Systematic and orderly approach – proximal to distal

4- Assessment and grade of individual muscles

5- Awareness of adaptive movements
1- Abnormal posture:

- Waiter tip
- Wrist and finger drop
- Claw hand
- Ape hand
- Foot drop

2- Motor examination:

- Muscle bulk
- Tone
- Power of individual muscles
Motor function assessment (LSUMC system):

- MO: Complete paralysis
- M1: Palpable muscle contraction
- M2: Active joint motion with elimination of gravity
- M3: Contraction or full joint motion against gravity
- M4: Contraction or full joint motion against gravity and resistance
- M5: Full range of motion - Normal contraction

3- Reflexes:

- Extremely sensitive indicator
- Do not return even recovery of sensation and muscle function
4- Sensory Evaluation:

- **Principle** - *Ex. of autonomic zones of innervation in which least likelihood of sensory overlap*

  (A) – All modalities to be tested

  (B) – Distribution of sensory loss

  (C) – Paresthesia -
  - *Distribution*
  - *Quality*
  - *Severity*
  - *Continuous or intermittent*
  - *Precipitating, exacerbating and ameliorating factors*
• Following scale (LSUMC system) can be used:
  • SO: Absence of sensation in an autonomous area
  • S1: Presence of deep cutaneous pain and sensation
  • S2: Presence of some degree of superficial cutaneous pain, tactile sensation, and two-point discrimination
  • S3: Presence of appreciable sensation, but no localization
  • S4: Presence of sensation with diminished acuity
  • S5: Normal sensory function

5- Sympathetic Functions :

- Sympathetic fiber : Most resistant
- *Usually confined to hand or feet*
- Sweating (*Starch test*)
- *Color and temperature of affected part*
- *Trophic changes*
5- Local examination:

- Neuroma

- Tinel’s sign:
  - Elicited from below upward
  - Advancing along the anatomical distribution of the nerve, particularly at the expected rate of nerve regeneration – e/o ongoing regeneration

- Associated bony or vascular injury

- Any contracture or deformity

- Presence of an aneurysm and its relationship to a neighboring nerve
NERVE INJURY: Clinical grades

<table>
<thead>
<tr>
<th>CLINICAL GRADE</th>
<th>CRITERIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1- Mild</td>
<td>1- Intermittent symptoms (e.g. Dysthesias, Pain, Numbness)</td>
</tr>
<tr>
<td>2- Moderate</td>
<td>2- Constant symptoms without e/o axonal loss</td>
</tr>
<tr>
<td>3- Severe</td>
<td>3- E/o axonal loss – muscle atrophy, abnormal two point discrimination or both</td>
</tr>
</tbody>
</table>
### NERVE INJURY CLASSIFICATION:

#### SEDDON CLASSIFICATION

<table>
<thead>
<tr>
<th>NEUROPRAXIA</th>
<th>AXONOTMESIS</th>
<th>NEUROTOMESIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Physiological conduction block</td>
<td>• Axonal interruption</td>
<td>• Division of nerve trunk</td>
</tr>
<tr>
<td>• Segmental demyelination</td>
<td>• Nerve in continuity</td>
<td>• Endoneural tube destroyed to variable length</td>
</tr>
<tr>
<td>• Disproportionate motor over sensory loss with sparing of autonomic function</td>
<td>• Axon disintegrate – phagocytosis – Wallerian degeneration</td>
<td>• Regenerating fibres + schwann cells + fibroblasts = Neuroma</td>
</tr>
<tr>
<td>• Recovery – Hrs to days</td>
<td>• Outcome – No return to v. good return of function</td>
<td>• Almost always require repair</td>
</tr>
<tr>
<td>• Crutch palsy</td>
<td>• Regeneration at the rate of 1 mm / day</td>
<td></td>
</tr>
</tbody>
</table>
### Sunderland Classification

<table>
<thead>
<tr>
<th>Sunderland</th>
<th>Seddon</th>
<th>Epineurium</th>
<th>Perineurium</th>
<th>Endoneurium</th>
<th>Axon</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Neuropraxia</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Block</td>
<td>Good</td>
</tr>
<tr>
<td>2</td>
<td>Axonotmesis</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>_</td>
<td>G / fair</td>
</tr>
<tr>
<td>3</td>
<td>Axonotmesis</td>
<td>+</td>
<td>+</td>
<td>_</td>
<td>_</td>
<td>F / poor</td>
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<tr>
<td>4</td>
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<td>+</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>Poor</td>
</tr>
<tr>
<td>5</td>
<td>Neurotmesis</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>Poor</td>
</tr>
</tbody>
</table>
Peripheral Nerve Injuries of the Upper Limb

: Axillary nerve (C5,6) injury :

• **Causes:**
  - Dislocation or reduction of shoulder
  - # of surgical neck of humerus
  - IM injection high in the post. aspect of the shoulder
  - After sleeping in a prone position with the arm raised above the head
- Loss the sensation over the “regimental badge” area
- Flattening of the shoulder
- Deltoid muscle weakness
Musculocutaneous nerve (C5,6,7) injury:

• **Causes:**
  - Usually along with BPI
  - Proximal humerus #
  - Shoulder dislocations
  - Carpet carrier's palsy

• **S/S:**
  - Weakness of elbow flexion
  - Weakness of arm flexion
  - Weakness of supination
  - Loss of sensation at lat. border of forearm
Radial nerve (C5,6,7,8,T1) Injury:

- **M.C. site** – Spiral Groove

- **Cause**:
  - IM injection in Triceps
  - Saturday night palsy
  - Crutch paralysis
  - # Shaft of humerus
• **S/S:**
  
  - Paresis or paralysis of extension of the elbow
  - Paresis of supination of forearm
  - Wrist drop
  - Sensory loss over a narrow strip on back of forearm and on the lateral side of dorsum of hand
  - Hypo or areflexia of triceps and brachioradialis jerk
Ulnar Nerve (C7,8,T1) injury:

- M.C. site - Medial epicondyle, or a little more distally where the nerve enters the forearm b/w the two heads of flexor carpi ulnaris

- Causes:
  - # of medial condyle
  - During anesthesia

- S/S:
  - Radial deviation of wrist on flexion
  - Claw-hand deformity
  - Paresthesia and sensory loss on hand
A- Egawa’s Test – Dorsal Interossei

B- Card Test – Palmer interossei

C- Book Test - Adductor pollicis (Froment,s sign)
Median nerve (C5,6,7,8,T1) injury:

- **CAUSES -**
  - Crutch compression
  - Sleep paralysis
  - Penetrating trauma
  - Shoulder dislocation

- **S/S:**
  - Atrophy of the thenar eminence
  - Simian or ape hand (d/t opponens pollicis)
  - Benediction hand
- Paresis of forearm pronation

- Paresis of distal flexion of the thumb

- Paresis of radial wrist flexion

- Impaired opposition of the thumb

- Paresis of flexion of the second and to a lesser extent, the third fingers

**Pen Test** – For Abductor pollicis brevis
Peripheral Nerve Injuries of the Lower Limb:

*Sciatic nerve (L4,5,S1,2,3) injury:*

*Complete lesion - rare*

**Causes:**
- Fracture dislocation of the hip
- Apophyseal avulsion fracture
- Hip joint surgery
- IM injection
- Gunshot wounds
- Femur fracture

**S/S:**
- Flail foot
- Wasting of the hamstrings and all muscles below the knee
- Paresis or paralysis of

  Knee flexion (hamstrings)
  Foot eversion (peronei)
  Foot inversion (tibialis anterior)
  Foot dorsiflexion
    (tibialis anterior and ant. leg muscles)
  Foot plantar flexion
    (gastrocnemius and soleus)
  Toe dorsiflexion (ext. of the toes)
  Toe plantar flexion

- Decrease or absence of the Achilles reflex

- High step gait

- Peroneal division > tibial division (75% of cases)
Common peroneal nerve (L4,5,S1,2) injury:

- **M. C. Site** - At the level of the fibular head

- **Causes:**
  - Trauma
  - During anaesthesia or coma
  - Over tight or ill-fitting plaster casts applied for leg fractures
- **S/S:**
  - Foot drop
  - Variable sensory disturbance affects the entire dorsum of the foot and toes and the lateral distal portion of the lower leg
Tibial nerve (L4,5,S1,2,3) injury:

- **Causes:**
  - Trauma - Dislocation of the knee

- **S/S:**
  - Variable paralysis of plantar flexion of the foot and toes
  - Numbness of the heel and part of the sole
  - Lateral side of the foot and posterior aspect of the leg

- Nerve may also be compressed in the fascial tunnel behind the medial malleolus and this can lead to weakness of the intrinsic muscles of the foot
Femoral nerve (L2,3,4) injury:

- **Causes:**
  - Iatrogenic
  - Penetrating gunshot and stab wounds
  - Pelvic fractures
\textit{S/S:}

- Weakness / Atrophy of the muscles of the ant. part of the thigh causing weakness of hip flexion (iliacus, psoas, and rectus femoris)
- Inability to extend the leg (quadriceps femoris).
- Impaired lateral thigh rotation (Sartorius)
- Sensory loss, paresthesias on the anteromedial thigh and inner leg as far as the ankle.
- Patellar reflex depressed or absent
Some Special Nerve Injuries:

A – Brachial Plexus injury in adults:

- Incidence: 10% of PNS injuries
- Ages 15-30 yr,
- Males
- Usually closed

- Stretch / traction – most common (90%)
- Pre ganglionic or Post ganglionic
Law of Seven Seventies (Narakas)

- 70% traumatic lesions due to RTA
- 70% traffic accidents involve a cycle or motorcycle
- 70% of these pts have multiple injuries
- 70% supraclavicular injuries
- 70% of pts w/ supraclavicular lesions will have one or several roots avulsed from the spinal cord
- 70% of pts w/ root avulsions will have lower roots C7, C8, or T1 avulsed
- 70% of pts w/ lower root avulsion will have persistent pain
Pre ganglionic Vs Post ganglionic

- Neuropathic pain
- Horners syndrome
- Winging of scapula (C6)
- Inability to move scapula medially (C5)
- Involvement of phrenic nerve
- Loss of sensation above clavicle
- Fractures of C7 transverse process, first rib
Supraclavicular: 70-75%

- Complete 5 - 50%
- Upper Trunk – 35%
- C6-C8 Avulsion – 8%
- C8 – T1 Isolated – 3%

Infraclavicular: 25 – 33%

- Pan BPI 45%
- Single/Combined 30%
- Isolated Nerve 25%

Proximal rupture, Distal Avulsion 60%
Five level Avulsion 30%
C4–T1 Avulsion
B - Obstetric Brachial Plexus injury:

- More common in:

1- Macrosomic infants -

2- When the arms are extended over the head in a breech presentation

3- When excessive traction is placed on the shoulders

- May be associated with I/L Phrenic nerve injury
• **Incidence** – 0.42 to 2.9 per 1000 births

• **TYPES** –

**A- Erb's palsy:**
- Common
- D/t injury to C5 and C6 roots.
- Moro reflex absent
- UL drops beside the trunk, internally rotated with flexed wrist (policeman’s or waiter’s tip hand)

• *Presence of the hand grasp - favorable prognostic sign*
B- Klumpke’s palsy:

- Less common

- D/t injury to C7, C8 and T1 roots.

- Paralysis of the muscles of the hand and weakness of the wrist and fingers' flexors

- If the sympathetic fibers of the 1st thoracic root are also injured - paralyzed hand and ipsilateral ptosis and miosis.
C – Iatrogenic nerve injuries:

1- Injection injury:
   - *Sciatic and Radial*
   - More often in infants and younger children
   - *Direct needle injury*
   - *Secondary constriction by scar*
   - *Direct damage by neurotoxic chemicals*
     Most toxic agents - penicillin, diazepam, chlorpromazine, tetanus toxoid, procaine and hydrocortisone

2- During surgery
   Common sites: - Carpal tunnel and distal wrist
   - Post. Triangle of neck, Popliteal fossa

3 - Ill fitting splint or plaster cast
D- Positioning & Nerve Injuries

• **M. C. nerves affected:**
  - Ulnar nerve – Most common
  - Brachial Plexus
  - Radial nerve
  - Common peronial nerve

• Compression or stretching leading to nerve ischemia

• Predisposing Factors:
  - Diabetes mellitus
  - Cancer
  - Alcoholism
  - Vitamin deficiency
  - Cigarette smoking
1- ELECTRO DIAGNOSTIC EVALUATION

- EMG/NCV: TWO PARTS
- Adjunct to thorough history, physical exam. and imaging study

“mild shocks and thin needles”

Ideal Time:
- After 7-10 days, neuropraxia can be distinguished from axonotmesis
- 3 to 4 weeks after the injury (Wallerian degeneration) with repeat study after 6 weeks
1 - Can localize the lesion to
   - Ant. horn cell, Nerve root
   - DRG, Plexus
   - Nerve, N M junction
   - Muscle

2 - Lesion can also be localized to
   - Cell body
   - Axon
   - Myelin

3 - Can determine –
   - Duration
   - Severity
   - Prognosis

4 - Can provide objective measure of –
   - Improvement
   - Worsening
A- Nerve Conduction Studies (NCS)

- Sensory conduction assessment — SNAP
- Motor conduction assessment — CMAP

**SNAP:**
- More sensitive than motor conduction studies
- Can localize a lesion as -
  - Pre or postganglionic

- Amplitude is “key” parameter
Remains Normal (on distal stimulation) in -

- No axonal loss
  (conduction block, demyelination)
- Preganglionic BPI

- Normal SNAP with numbness s/o –
  - Lesion proximal to DRG
  - Decrease in amplitude s/o postganglionic

- Decrease conduction velocity in –
  - Severe axonal loss
  - Demyelination
**CMAP (M wave):**

- 3 most imp. aspect of CMAP:
  - Amplitude
  - Conduction Velocity
  - Distal motor latency

- Normal conduction velocity –
  - Arm: 50-70 m/sec
  - Legs: 35-45 m/sec

- Quantification of the area and amplitude provide an estimate of the functioning nerve and muscle.

- Stimulation at proximal site causes:
  - Increase latency
  - Decrease amplitude and area
**F-Wave:**

- Late response of a motor unit
  (20-50 m sec)

- Assess the proximal motor root

- Prolonged asymmetric F waves suggest a proximal root lesion

- Clinical application best for plexopathy

- Quite prolonged in demyelination, mild prolongation in axonal injury
B- EMG :

1- INSERTIONAL ACTIVITY

- Due to injury to muscle fibre by electrode insertion

- Lasts for two seconds

- Indicate viable muscles

- Prolonged in :
  : Denervated muscle
  : Myotonic disorder
  : Inflammatory muscle diseases
2- EMG ACTIVITIES AT REST:

Normal muscle: No spontaneous resting activity

- Abnormal spontaneous potentials
  Positive sharp waves, fibrillations
  (Denervation Changes)
    - 7-10 days (paraspinal)
    - 2-4 weeks (distal muscles)

- May persist from reinnervation (4 month) to complete atrophy
3- EMG FINDINGS DURING ACTIVITIES:
   A- Single motor unit assessment (MUAPs)
      - Sum of APs of muscle fibres of a single motor unit
      - In reinnervation: Increase in size and duration of MUAP

   B- Recruitment to full contraction
      - Usually 20-40 MUAPs within the recording range of the EMG needle firing at about 40 Hz with maximal contraction
      - In neuropraxia: Only recruitment changes
**LOCALIZATION**:

By two methods –
1. Detecting focal swelling or conduction block on NCSs
2. Assessing the pattern of denervation on EMG

**LIMITATIONS**:

- Small diameter autonomic and sensory axons cannot be tested (for pain and temp.)
- Cannot distinguish b/w complete axonotmesis and neurotmesis early
- Cause discomfort and are often painful
3- CT- myelography:
- Useful in predicting spinal nerve root avulsion in BPI

2- MRI and MR Neurography:
- Uses fast spin echo (FSE) pulse sequences on a standard 1.5 T MRI
- Increased signal intensity in denervated muscles (using STIR or T2 weighted pulse sequence)
- Signal change as early as 4\textsuperscript{th} day, in contrast to the 2-3 weeks in EMG

- Signal changes normalize with muscle reinnervation

- Useful in early differentiation from neuropraxia to high grade injury

- Chronic denervation with muscle atrophy- best seen in T1WI
## Correlation of MRI findings with EMG/NCV

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<tr>
<th>DIAGNOSTIC MODALITY</th>
<th>NEUROPRAXIA</th>
<th>AXONOTMETIS</th>
<th>NEUROTMETIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>NERVE CONDUCTION STUDY</td>
<td>Focal nerve conduction block</td>
<td>Initial absence of nerve conduction followed by recovery through regeneration</td>
<td>Persistent absence of nerve conduction</td>
</tr>
<tr>
<td>EMG</td>
<td>No denervation</td>
<td>Denervation after 2-3 wk</td>
<td>Persistent denervation</td>
</tr>
<tr>
<td>MR neurography</td>
<td>Focal signal increase</td>
<td>Transient signal increase distal to injury followed by normalization with axonal regeneration</td>
<td>Signal increase distal to injury followed by delayed normalization</td>
</tr>
<tr>
<td>MRI of muscle</td>
<td>Normal</td>
<td>Transient signal increase followed by normalization with muscle reinnervation</td>
<td>Transient signal increase, then reduction with atrophy and fatty infiltration</td>
</tr>
</tbody>
</table>
MANAGEMENT

CONTROVERSIES

To operate / manage conservatively?

When to operate?

Type of Surgery?
Conservative Treatment

- For Neuropraxia and mild cases of axonotmesis
  - Most of the mild lesions recover within one month

- **AIM**: Preservation of the mobility of affected limb till recovery

- **Components**:
  1. Splintage of the paralytic limb
     - with position to most effectively relax the affected muscles

     Axillary N. – Shoulder abduction splint
     Radial N. – Cock-up splint
     Ulnar N. – Knuckle – bender splint
     Sciatic N. – Food drop splint
2- Care of skin and nails

3- Physiotherapy:
   - Full range of movement at least once every day
   - Massage of paralysed muscles
   - Building up of the recovering muscles
   - Developing the unaffected or partially affected muscles

4- Drugs:
   - Analgesics for pain
   - Steroids ??
   - Methylcobalamine ??
Surgical Treatment

Timing of surgical nerve reconstruction

- **A- Immediate:**
  - Preferred in clean cut laceration

- **B- Early (1 month):**
  - Blunt trauma or avulsion injury causing complete nerve destruction
  - Nerve ends are usually contracted and/or scars need to be resected

- **C- Delayed (3-6 months):**
  - when the degree of injury has not yet been ascertained with expectation as natural recovery better than surgical repair and no signs of recovery
D- Late (1-2 years or more) :
- Generally only carried out for pain control, such as neuroma resection.

- After 6 months the overall results of nerve repair steadily deteriorate

- Nerve reconstruction is not recommended after 18 months
Preoperative planning priorities

1. Review clinical examinations

2. Scrutinize electro diagnostic studies

3. Review CT myelography/imaging

4. Assemble operative team, plan for intraoperative electro diagnostic studies

5. Plan a preoperative conference, including priorities and contingency plans

6. Prepare patient’s expectations
Principles Of surgical Repair

- Exposure Of Healthy nerve proximal and distal to injury
- Intra operative Electrophysiological evaluation
- Intraoperative microscopic examination by quick section
- Fascicular /Group fascicular /Epineural microsuture technique
- **Avoid damage to the blood supply to the nerve**
- Avoid Tension
- Using minimal number of sutures
- Fibrin glue can further strengthen the anastomosis

- Maximizing the number of motor fibers available for repair

- Matching the graft to recipient nerve fascicles

- Per-operative nerve stimulation and EMG - To establish the continuity of the nerves

- If found to be in continuity – only external / internal Neurolysis is sufficient

- If No NAP is recorded and microscopically the nerve appears damaged – Resection of the lesion in continuity and repair
Surgical treatment options

1- Neurolysis
2- Nerve repair
3- Nerve grafting
4- Neurotization
5- Nerve root replantation
6- Free Muscle and tendon transfers
Neurolysis

- Used to decompress nerves from internal fibrosis or from surrounding tissue

- Effective only if scar tissue seen around nerve or inside epineurium, preventing recovery or causing pain

- External Vs Internal
Management of Neuroma in continuity

- Resected until normal looking nerve tissue is present
- If it extends to the root level – some sort of Neurotization required
- According to published results, some recommend resection and nerve grafting with int. neurolysis, whereas others maintain that neurolysis is beneficial, esp. if intraoperative electrophysiological testing reveals significant conduction across the neuroma
Nerve Repair

Magnification, micro-sutures and micro instruments considerably improved the results in nerve repairs.

Primary Repair

- Sooner the better
- Tissue dissection away from nerve
- In secondary repair – dissection from normal to injured area
- Ragged ends – pared.
- Use microscope and 10\0 suture.
• Suture epineurium in background of glove piece

• Epineural vs perineural repair (slightly superior)
  - Orgel and Terzies, 1977

• **Sutureless anastomosis:**
  - Laser welding
  - Tubular/ cuff union
  - Tissue adhesives

  (May reduce scar tissue
  No definitive improved functional results)

• Splinting for 3-4 week
Nerve Grafting

- Indicated for well defined nerve ends with gap

- Intraoperatively a good fascicular pattern should be seen after the neuroma is excised

- Donor nerves
  - Sural nerve - M. C, (single cable)
  - Cutaneous nerves of arm and forearm
  - Dorsal sensory branch of radial nerve
  - Distal portion of ant. interosseous nerve.

- Graft orientation should be reversed to minimize axonal branch loss
For success of nerve grafts –
- small, thin grafts used
  (vascularized faster)

- Length of graft –
  Best result - upto 4 cm
  Not to be used > 8 cm

- For gaps (> 20 cm) with associated soft tissue loss over the repaired area, current recommendation –
  - free vascularized nerve grafts

- Good Results in 70 % cases
Types of graft

- Cable nerve graft
- Group interfascicular nerve graft
- Free neurovascular nerve graft
- Pedicle nerve graft
- Tubal graft
Nerve Transfer

- Indicated for root avulsions of brachial plexus.
- Spinal accessory to suprascapular nerve.
- Intercostal nerves to musculocutaneous nerve
- Medial pectoral to musculocutaneous nerve
- Descending cervical plexus to upper and middle trunk
Tendon Transfer

- Motor end plate must have degenerated (i.e. 18 – 24 months after injury)

- Assess
  Muscles – lost
  Muscles – available

- Donor Muscle
  Expendable
  Adequate power
  Synergistic

- Transferred tendon
  Routed subcutaneously
  Straight pull
Principle in surgical management of BPI

- **AIMS OF SURGERY**
  ‘To a man with nothing, a little can be a lot’
  Depends on what is lost, what is needed and what is possible

- In pan BPI –
  - first goal of the surgery is to obtain **shoulder stabilization and abduction** and an active **elbow flexion**, while extension is achieved from gravity

- In a Meta-analysis of literature involving nerve transfers shoulder abduction is best restored by XI Nerve – SS anastomosis, while elbow flexion is best regained using IC Nerves without grafts (R. Midha. Neurosurgery Focus. Vol 16; May 2004)
Options for Neurotization

- Suprascapular: Phrenic
  Spinal accessory
  C7 fascicle

- Axillary: Phrenic
  Spinal Accessory
  Medial pectoral

- Musculocutaneous: Ulnar
  Intercostal
  Medial pectoral

- Oberlin Technique - Biceps reinnervation
  using some ulnar nerve fascicles
  - flexor Carpi ulnaris
Post operative Care

- 3-6 weeks immobilization
- Physiotherapy
- Electrical stimulation
- Re-education of muscles
- Follow up Electro diagnostic studies
- Occupational therapy
- Limb reconstruction
- Psychotherapy
PROGNOSIS

- DEPENDS ON:
  - TYPE OF LESION
  - LEVEL OF LESION
  - TYPE OF NERVE
  - SIZE OF GAP
  - AGE
  - TIME OF SURGERY
  - ASSOCIATED LESION
  - SURGICAL SKILL
Recent Advances:

1- Endoscopic technique for harvesting the sural nerve
   (Eich BS et al, J Reconstr Microsurg 2000; 16: 329-31)

Adv: Avoids long unaesthetic scar
     wound infection
     wound pain

Disadv: Cost of the equipment
        Long learning curve
2- Nerve Allografts:

(Mackinnon SE et al, *Plast Reconstr Surg* 2001)

Acellular nerve graft

- Removal of components that would cause rejection
  (tissue sources: cadaver, animal)
- Nerve naturally re-grows on its own “skeleton”
- Natural tissue chemically and physically aids nerve regeneration

Adv:

- Avoids use of nerve graft from patient
- Minimizes time of procedure
- Option for previous “non-operable” cases
- Familiar to surgeons and easy to handle
- Easily modified
3- Nerve conduits

(Ashley WW et al, J Neurosurg 2006; 106: 452-6)

- Direct axonal sprouts from the proximal to the distal nerve stump

- Channel for diffusion of neurotropic and neurotrophic factors

- Constructed from:
  - Polylactide-co-caprolactone
  - Polyglycolic acid
  - Silicone
  - Laminin, Collagen

  (Biological, degradable)
4- Fibrin glue in nerve repair  
(Narakas A. Orthop Clin North Am, 1988)

- Less inflammatory response
- Better axonal regeneration
- Better fiber alignment
- No definitive e/o improved functional results

5- Nerve Growth Factors (Midha R et al, Neurosurg 2003)

- Neurotrophins
- Glial-cell-line derived neurotrophic factor ligands (GLFs)
- Neuropoietic cytokines

- Enhance regeneration and increase the nerve gap that can be bridged (to 15 mm)
Management of nerve injury requires thorough anatomical knowledge and function with complete pre-operative work-up.

No alternative to a proper and thorough neurological examination.

Controversies exist regarding optimum management thus every case to be individualized.

Magnification, micro-sutures and micro-instrument considerably improved the results.
Thank You!