ABSTRACT
A diagnostic percolation and approach to the correct methods of approach in Peripheral Neuropathy, has currently advanced. The newer concepts in diagnosis, especially with the advent of Neurophysiological studies and imaging studies, skin biopsy has totally revolutionized the diagnosis of peripheral neuropathy to a remarkable level. It is also observed that various newer theories of pathophysiology, etiopathogenesis in the development of peripheral neuropathies, with special reference to the diabetic neuropathy, has made the disease process to understand clearly. As the result, the approach to the management of the neuropathies, especially of diabetic origin, has reached a level of success. Understanding other types of neuropathy, especially GBS, Entrapment neuropathies, are mostly clearly understood by imaging studies, and neurophysiological studies. The following presentations have been designed to give a clear view and pitfalls in the diagnosis, treatment which are updated.

KEYWORDS
GBS – Guillen Baree Syndrome; PN - PERIPHERAL Neuropathy; PNS – Peripheral nervous system; PKC – Protein Kinase C Tens – Transcutaneous electrical nerve stimulation; HSMN Hereditary Sensory Motor Neuropathy; DM – diabetes Mellitus; LPN – Lateral Popleteal Nerve; SSR – Sympathetic Skin Response; ROS – Reactive Oxygen Species; IPN – Indicator plaster Neuropad; NADH – Nicotinamide Adenine Dinucleotide Phosphate Hydrogenase; NAD1 – Nicotinamide adenine Dinucleotide (Oxidized form); HSN – Hereditary Sensory Neuropathy; HSMN – Hereditary Sensory Motor Neuropathy; QST – Qunatitive Sensory Testing

Peripheral Neuropathy (PN) is a clinical disorder, where in the peripheral portion of the nervous system get involved, in many ways, depending on their etiology. They have a common clinical presentations, with subtle variations in their symptomatology and signs, depending on their etiology. Once this enigma is cleared, PN becomes a clear issue for diagnosis and treatment. To get know the secrets of treatment and diagnosis, one should be “once again” thorough with the Micro anatomy, physiology, and Pathology. There may be lots of overlapping in the symptoms and etiology, but once this is cleared, the treatment is a walkthrough.

INCIDENCE
No clear cut well defined incidence has been observed, owing to the lots of overlap syndromes in PN. Recent studies by Predeepa et al, A cross Sectional Population Based study reports Among urban South Indian type 2 diabetic subjects, the prevalence of neuropathy is 26.1% showing a correlation to age, glycated haemoglobin and diabetic age. But in tropical countries the incidence due to infection of Hansen’s disease is much more than non tropical country. Of course, the major issues with PN stands with diabetic, metabolic neuropathy. Of late HIV neuropathy is a major rising issue. Still in 13-22% of cases, etiology cannot be defined in spite of many modern investigations

BASIC PROPERTIES OF PNS
• Involves the cranial nerves except the optic nerve which is an extension of brain parenchyma.
• The other parts of PNS are spinal nerve roots, dorsal ganglia, Peripheral nerve trunks, up to terminal cutaneous and visceral branches.
• Axon, are part of PNS. They are the primary conducting substance of neural impulses. Axons can differ as sensory and motor, mostly on physiological properties. Their conduction rate is very poor. Sensory axons are responsible for conduction of vibration, touch, proprioceptive sensations. These axons are covered with myelin like the plastic insulation of a copper wire, and form large myelinated fibers. When they are affected it is called large fiber PN. Small myelinated axons are usually autonomic fibers, designated as small fibers, sub serve the conduction of touch, pain, temperature. There are unmyelinated small fibers which are mostly sensory, conduct pain and temperature only. PN involving small myelinated axons, unmyelinated axons are designated as small fiber PN.

Neuropathies can be viewed on their pattern of involvement also. Table I
**ISSUES IN DIAGNOSIS OF PN**

1. **Is it focal** (involving a particular nerve only) or **multifocal** (involving many nerves)
   - In a single or multiple places, or **symmetric** (equal on either portion of the body) \(^{3,4,5,6,7}\)

2. **If symmetric,**
   - a. **Proximal** – predominantly motor (GBS) except lead neuropathy, which is distal
   - b. **Distal** – motor or sensory or mixed (toxic, diabetic, metabolic)

**ETIOPATHOLOGY**

1. Microvascular ischemia is the main sheet anchor of starting the dysfunction of PNS of diabetic and ischemic etiology. Following that, cascade of subsequent metabolic alterations taking place due to anoxia of the axons.

2. Normal euglycemic persons have glucose metabolic Kreb cycle pathway and the end product is CO2 and water, which is harmless. In spite, a small portion of 3% may go in for alternate polylol pathway, embracing the anoxic cycle, whose end product is sorbital and fructose, which are neurotoxic. This is because of Michaelis constant of aldose reductase (the enzyme responsible for anoxic cycle) is much higher than that for glucokinase or hexokinase. But in both Type I and II DM, due to metabolic imbalance, there is hyperglycemia, more than 30% of glucose enters the alternate pathway. This heralds, microvascular system, which is the beginning of various complications of target organs like brain, myocardium, retina, and renal vasculature \(^{3,4,5,6,7}\).

3. Hyperglycemia is the main cause of complications in DM. Maintaining euglycemia in a diabetic individual is comparatively difficult. The factors which herald the anoxic cycle in a hyperglycemic individual are \(^{3,4,5,6,7}\).
   - a. Enhanced activity of aldose reductase
   - b. Activation of Protein Kinase C isoforms (PKCs contain the isoforms \(\beta, \beta'\), and \(\gamma\))
   - c. Accumulation of Advanced toxic glycation products
     - d. Reactive oxygen species

**NEURONS IN CNS REACT**

Hence under hyperglycemia, hexokinase is saturated with glucose, resulting in more influx of glucose through polylol pathway, which is highly harmful, as well reduction in the normal signaling of NADPH, NAD \(^1\). The amplification of polylol pathway of glucose reduction, causes gross metabolic imbalances in the cells which are insulin independent uptake of glucose. Reduction in NADPH, NAD \(^1\), causes reduction in function of dependent enzymes, resulting in reduction in NO, ultimately damaging the endothelial cell metabolism, causing apoptotic reactions in endothelial cells. The ultimate culprit of all these cascades is enhanced activity of Aldose Reductase, whose inhibition may prevent many cascade of complications of microvasculature, especially in neuronal, axonal system, myocardial tissues especially in diabetes.

**PATHOPHYSIOLOGY**

Even though the etiologies are multiple the type of injury to the PN is more or less same.

1. Axonal damage, called axonopathy, happening as in Wallerian degeneration, (distal degeneration of PN consequent to proximal injury)

2. In distal toxic and metabolic neuropathy the most distal part of the PNS axon degenerates with concomitant length dependent degeneration of myelin which is called “ Dying Back Neuropathy”

3. Degeneration of myelin or axons close to spinal cord or dorsal ganglia results in damage to the central spinal system. Myelin degeneration can be of varied etiology, such as ischemic, degenerative, infective, immune mediated, heredo familial. Acquired demyelinating neuropathies are mostly patchy, and hence recovery is complete wherein the axons are relatively spared. HFN are usually diffuse, involves axons also, hence they are progressive and incompletely recover also.

**DIAGNOSTIC CASCADE.**

**PITFALLS**

1. PN can be mimicked by myelopathy, synringomyelia, or ta-
Peripheral Neuropathy - Some Newer Concepts and Diagnostic Ways

Table 2: Distal Symmetric Sensorimotor Polyneuropathies

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endocrine diseases</td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td></td>
<td>Hypothyroid</td>
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<tr>
<td>Nutritional diseases</td>
<td>Alcoholism, Vitamin B12, Folate deficiency</td>
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<td></td>
<td>Whipple’s disease</td>
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<td></td>
<td>Post gastrectomy syndrome</td>
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<td></td>
<td>Rheumatoid Arthritis, Polyarteritis nodosa</td>
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<td></td>
<td>Systemic lupus erythematosus</td>
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</tbody>
</table>

Table 3: Proximal Symmetric Motor Polyneuropathies

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal Symmetric Motor Polyneuropathies</td>
<td>Guillian – Barre Syndrome, Chronic inflammatory demyelinating polyradiculoneuropathy</td>
</tr>
<tr>
<td></td>
<td>Diabetes mellitus, Porphyria</td>
</tr>
<tr>
<td></td>
<td>Macroglobulinaemia, Monoclonal gammopathy</td>
</tr>
<tr>
<td></td>
<td>Monoclonal gammopathy</td>
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</tbody>
</table>

Table 4: Comparative Patterns of Neuropathies and Neuronopathies by Fiber Type (Important ones)

<table>
<thead>
<tr>
<th>Pure Sensory neuropathies and neuronopathies</th>
<th>Small – Fiber neuropathies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paraneoplastic Medications, Crohn’s disease</td>
<td>Leprosy</td>
</tr>
<tr>
<td>Friedreich’s ataxia</td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Carcinomatous sensory neuronopathy</td>
<td>Alcoholic neuropathy</td>
</tr>
<tr>
<td>Lymphomatous sensory neuronopathy</td>
<td>Amyloidosis, B1 Deficienciy</td>
</tr>
<tr>
<td>Siogren’s Syndrome, Paraproteinemias</td>
<td>HIV/AIDS</td>
</tr>
<tr>
<td>Chronic gluten enteropathy</td>
<td>Hereditary</td>
</tr>
<tr>
<td>Nonsystemic vasculitic neuropathy</td>
<td>Neurpathies with autonomic involvement</td>
</tr>
<tr>
<td>Idiopathic sensory neuropathy</td>
<td>Diabetic neuropathy</td>
</tr>
<tr>
<td>Vitamin E deficiency, Hereditary sensory neuropathy types I and IV</td>
<td>Amyloidosis, Porphyria, Lymphoma, Drugs toxicity, arsenic, mercury toxicity</td>
</tr>
<tr>
<td>Primary biliary cirrhosis</td>
<td>Guillian – Barre syndrome</td>
</tr>
<tr>
<td></td>
<td>Paraneoplastic neuropathy</td>
</tr>
<tr>
<td></td>
<td>Alcoholic neuropathy</td>
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Table 5: Neuropathies with Abrupt / Rapid Onset

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic neuropathies</td>
<td>Rheumatoid arthritis, Polyarteritis nodosa, Diabetes Mellitus</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>Cranial Neuropathies, Diabetic amyotrophy.</td>
</tr>
<tr>
<td>Nerve compression</td>
<td>Hemorrhage and swelling within a restricted anatomical compartment</td>
</tr>
<tr>
<td></td>
<td>Penetrating wounds, thermal injury, iatrogenic (Injection injury).</td>
</tr>
</tbody>
</table>

Table 6: Differential Diagnosis of Neuropathies by Clinical Course

<table>
<thead>
<tr>
<th>Acute onset (within days)</th>
<th>Subacute onset (weeks to months)</th>
<th>Chronic Course/Insidious onset</th>
<th>Relapsing/remitting course</th>
</tr>
</thead>
<tbody>
<tr>
<td>Guillian – Barre syndrome</td>
<td>Maintained exposure to toxic agents/medications</td>
<td>Hereditary motor sensory neuropathy</td>
<td>Guillian Barre syndrome</td>
</tr>
<tr>
<td>Acute intermittent porphyria</td>
<td>Persisting nutritional deficiency</td>
<td>Dominantly inherited sensory neuropathy</td>
<td>CIDP</td>
</tr>
<tr>
<td>Critical illness</td>
<td>Abnormal Metabolic state</td>
<td>CIDP</td>
<td>HIV / AIDS</td>
</tr>
<tr>
<td>Polyneuropathy</td>
<td>Paraneoplastic syndrome</td>
<td>TOXIC</td>
<td></td>
</tr>
<tr>
<td>Thalium toxicity</td>
<td>CIDP</td>
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</table>

**APPROACH TO NEUROPATHY**

Various ways of approaching the PN has been postulated by different authors. The following tables gives the various details.4,5,6

**Table 1, 2, 3, 4, 5 & 6 give different ideas of approach**10,11,12

**SOME GLIMPSES OF PN**

- Cranial nerves and autonomic nerves are involved in specific PN, like 7th Nerve involvement in GBS
- Hansen’s disease selectively pick up more in cooler area nerves involving lower half of the face rather than upper half, and involves as branch paralysis than trunk involvement. The taste fibers are never involved. Fig 1
- Small fiber neuropathies are mostly seen in diabetic, metabolic, and in alcoholism.
- Toxic neuropathies like Arsenic, involves predominantly sensory, rarely autonomic and motor system.
- Nutritional Neuropathies are mostly sensory

**HIV NEUROPATHY**

- Distal symmetric, axonal sensory motor neuropathy
- Small fiber painful neuropathy
- Autonomic neuropathy is rare in AIDS neuropathy, but common in DN and alcoholism

bes dorsalis, and tumors of spinal cord.

2. Not but not the least, but most confusing pictures will be given in hysterical conversion reactions.

3. Early lesions mimic pure sensory neuropathy but, careful examination will reveal subtle motor neuropathy, which is a common feature in diabetic PN. Unless it is observed it is often missed. Symptoms of distal motor weakness are more reliable than our examinations.
COmPLICATIONS UNNOTICED IN PN

Long standing neuropathies cause tropic changes, painless ulcerations, pes cavus, kyphoscoliosis, alopecia, bone density loss, thinning of phalanges, neuropathic arthropathy (Charcot joints) Pathological fractures, nerve thickening as in leprosy, HSMN, DM and amyloid neuropathy.

EVALUATION OF NEUROPATHY.

Neurophysiological studies performed in best hands, gives enormous facts in the diagnosis or elimination of PN existence. EMG, NCS are the sheet anchor of diagnosis.13,14,15

NEUROPHYSIOLOGICAL STUDIES GIVES THE FOLLOWING IDEA

1. Confirmation of presence of neuropathy
2. Small or large fibre is involved
3. Whether, Motor or sensory or mixed
4. Axonal loss Vs demyelination or mixed

Axonal loss is identified by loss of action potential amplitude volume and denervation potentials in EMG.

FINDINGS IN DEMYELINATION

1. Velocity of conduction is reduced
2. Prolonged distal latency
3. Conduction block
4. Temporal dispersion
5. Prolonged or minimum volume of F wave latencies.

LIMITATION OF NERVE CONDUCTION STUDIES

1. No way to find out proximal sensory conduction
2. Age related changes in latencies in advanced age group which confuses the interpretation.

To consolidate, Neurophysiological studies are highly complementary, with the clinical data, and always correlated with the clinical appearances.

QUANTITATIVE SENSORY TESTING (QST)

Quantitative sensory testing is a method used to assess the damage to the small nerve endings which detect changes in temperature and the large nerve endings which detect vibration. Therapeutic improvement also can be assessed with QST. QST uses a computer testing system to measure how the nerves involved respond to vibration and changes in temperature. The test quantified and compared to "normal" patients as well as to the patient's unaffected side, if neuropathy is isolated. The procedure is non invasive and no needles are used.

AUTONOMIC FUNCTION STUDIES

1. Simple ECG is a good evidence of autonomic function. Findings of sinus arrhythmia, R-R' intervals variations, and Heart rate variations are good index of good autonomic PNS functions.
2. Heart rate determination during lying, standing positions and in good isometric handgrip are good index of PNS
3. Sympathetic Skin response give an index of autonomic insufficiency and small fiber status, which is not of great reliability. The sensitivity and specificity of SSR is poor.

IMAGING STUDIES IN PN 17,18,19

Of late imaging studies plays a major role in the diagnosis of entrapment neuropathies. An entity called Peripheral MR Neurogram is being more popular in MR studies. The sensitivity and specificity is high in imaging studies, in the diagnosis of PN, especially entrapment neuropathy. The following conditions shows high degree of diagnostic yield.

1. Carpel tunnel syndrome
2. Guyan's canal syndrome (Ulnar nerve deep branch involvement in hand)
3. Cervical rib syndrome, and plexopathies
4. Cervical plexopathy of various causes, Viral etiology like Pancoast tumor etc.
5. Root avulsions syndromes, entrapment in spinal disc and injuries
6. Fracture and mal healing entrapment neuropathies.
NEWER METHODOLOGIES

INDICATOR PLASTER NEUROPAD
Response to this test by application of a patch which turns pink if nerve conduction is normal, and turning blue if PN is present, whatever may be the type of etiology or fiber type. Some author's claim this test as a “Gold standard test”

SKIN BIOPSY
There are situations wherein very early lesions of the small fibers sub serving the erector pilorum. End bulbs of Krouse, free nerve endings, could be stained with appropriate staining technique especially with fluorescent tagging in a skin biopsy of the suspected peripheral neuropathy. These results are high yielding and confirmative in early PN, even in the absence of neurophysiological evidence of PN. It is a painless punch biopsies, often very easy as outpatient procedures, but requires advanced staining technique under trained neuro pathologists. It allows general practitioners dialektologists and specialists in orthopedics – to diagnose neuropathy thereby avoiding delayed or incorrect diagnosis, to investigate its etiology, and to focus treatment, in particular for neuropathic pain. PN of Hansen's type is highly recognized with skin biopsy, as the cutaneous involvement is high in Hansen's

PERIPHERAL NERVE BIOPSY
The nerve biopsy is one of the conventional but assertive diagnostic tool for confirmation of the type of neuropathy. But ultimately, it lies in the effective hand for diagnosis, which requires newer staining methods, and highly experienced neuropathologist, who would be dealing mainly in peripheral nerve.

NEWER SIGNS
Phalen's sign is classically described, for the diagnosis of CTS. Many of the patients get confused with the methodology of keeping both hands down. Instead, if both the palms are kept in a position as wishing “Namaste”, the same symptoms appear. Appears to be an easy method of determining clinically the CTS.

CTS is diagnosed by delay in the conduction difference between median and ulnar digital stimulation of the fourth digit in the hand, and recording the sensory potential latency over the nerve in volar surface of the arm. Wherein it is observed there is a significant delay in the median latency, but apparently no delay in the latency of ulnar part of stimulation with ring electrodes, as the ulnar nerve never pass through the carpal tunnel

MANAGEMENT
Management of PN is tricky, and once the diagnosis is achieved regarding the type of fiber involved, it is easy to tackle.

DRUGS USEFUL IN LARGE FIBER NEUROPATHY
1. Dysmyelination and demyelination are the complications in recovering neuropathy. The basic requirement is B12 and folic acid. Methylcobalamin appear to be one of essential drug in the recovery process of any type of neuropathy.
2. Immune mediated injuries like GBS respond well to IVlg, in doses of 400 mg per kg per day, given at 5 day course
3. Steroids once avoided is gaining a place again in immune mediated neuropathy especially when proximal cord parenchyma degenerates in consequence to the root demyelination.

SMALL FIBER PAINFUL NEUROPATHY
As we see such phenomenon in Diabetic Neuropathy, and other related disorder which is very common.
1. Drugs towards reconditioning the nerves. (Associated demyelination)
   The drug methycobal, appears to be highly superior to the conventional hydroxyl cobalamin, both in repairing the axon, and remyelination. Alleviating the symptoms like paresthesia, pain, restless legs are done better with duloxetine, in doses 40-60 mg once daily to twice daily, Gabapentin 300-600 mg per day, to a certain extent.
2. Alpha lipoic acid, along with Berin are good combination in the recovery process.
3. Pregabalin is a molecule of late considered to have greater alleviation of neuropathic pain, of any origin. The mechanism of action appear to be central desensitization of the pain signals, especially reduction in the area of allodynia.
   Observed to be of good use in post herpetic neuralgia, diabetic neuropathy, and other painful neuropathy, including entrapment syndromes. The dosage recommended is around 75 to 150 mg in Indian context, for 30 days, observed to have the relief after a brief period, but sustained relief. (Lyrica 75 mg capsules)
4. Counter irritants, like capsicum extracts, are not indicated as they subserve only symptomatic relieve masking the degeneration to a great extent
5. Of late, therapy towards preventing the complications is focused. Researches view through drugs, which may alleviate the primary process of dysfunction in me tabolic neuropathies, especially in DM. The primary enzyme Aldose Reductase hyperactivity (wide supra), appear to be the culprit in developing the primary endothelial cell damage and dysfunction, by enhancing the polyal pathway causing various toxic products is totally prevented by consumption of these drug molecules. Epalrestat, an aldose reductase inhibitors gives total umbrella coverage, from not only neuronal axonal damage, also prevents micro vascular com-
APPLICATIONS OF ANY ETIOLOGY SUCH AS PROTECTING THE VASCULATURE OF CORONARY, CEREBRAL, MUSCULAR, RENAL AND PARENCHYMAL. COMBINATIONS OF ALDOSOME REDUCTASE WITH OTHER MOLECULES OF ALPHALOICO ACIDS, METHYCOBAMINE WITH B VITAMIN APPEARS TO BE A RATIONAL COMBINATION OF MANAGEMENT OF MANY TYPES OF PERIPHERAL NEUROPATHIES, ESPECIALLY IN PN OF DM, ISCHEMIC NEUROPATHIES. THE MANAGEMENT IS NOT ONLY SYMPTOMATIC BUT CURATIVE ALSO.

**Epalrestat**, (S-[(Z,E)-B-Methylcinnamylidene]-4-oxo-2-thioxo-3-thiazolidineacetic acid) is a molecule of aldose reductase inhibitor is a recent introduction in the management of metabolic PN. It is a highly protein bound compound with binding rate around 90%. The clearance is renal as conjugated products by liver with half life of one hour: and not dose dependant. Oxidative stress in RBC has taken up an index of the therapeutic efficacy of this drug in many metabolic neuropathies especially in DM.²⁴ The average duration of the course of the drug would be 3 to 4 weeks, with maintenance further. Effective enhancement in the NCV, and autonomic functions has been observed in various studies after Epalrestat administration.²⁴ Various other parameters of assessing the improvement in autonomic functions like pupillogram, proximal latency tests²⁴ observed to have promising reports. (EPAREL – 50 Micro) Dosage of 50 to 150 mg per day do not cause any significant side effects. Reports regarding the toxic effects like thrombotic events, fulminate toxic hepatitis have been reported in rare cases, depending on the idiocyroch of the drugs. The drug should be discontinued if one observes increased SGPT, at the end of 7 days course of the drugs.⁹,²⁷,²⁸,²⁹ Comparative trials have shown good tolerability of the drug. On the available drugs at present Epalrestat, appears to be an ideal drug with respects to the alleviation of symptoms and cure of the Peripheral Neuropathy, especially of diabetic and ischemic origin. Early administration of the Epalrestat appear to prevent the disease process. As there are no significant side effects, the drug can be effectively used in the clinical practice.

**NEUROTROPHIC FACTORS**

Studies of Neurotrophic factors represent one of the most promising areas of research aimed at finding new, more effective treatments for peripheral neuropathies. These substances, produced naturally by the body, protect neurons from injury and encourage their survival. Neurotrophic factors also help maintain normal function in mature nerve cells, and some stimulate axon regeneration. Mouse Nerve Growth Factor has been tried much in mouse trials, for the regeneration of the axon in Diabetic Neuropathy, Requires further authentication ³⁰

**PHYSICAL MEASURES**

Many physical measures appear to be alleviating the symptomatic relief in painful PN. One such good method is to apply TENS. Application of TENS, significantly reduce the painful situation, especially nocturnal cramps, painful paresthetic peaks. Application of TENS in the lower and upper limbs over a period of 10 mts in each limb, alleviates the initial surge of suffering. Of course, it is not curative but gives symptomatic relief.

Ultrasound is not the method of management in the entrapment neuropathy, as it increases the edema and vascularity of the entrapmed compartment, causing nerve compression sometimes more acute. It is not a procedure of choice.

**LOCAL STEROIDS**

Local steroids application in the entraped nerve syndromes may give some relief which is not permanent. The danger of injuring the median nerve physically and chemically may result in acute damage to the nerve.

**SURGICAL CORRECTIONS**

Entrapment neuropathies are best treated by relieving the entraped nerve, by dissecting the flexor retinaculum under physical visual, microscopical surgical technique, without damaging the micro neuro vasculature. The ideal management is by surgery, and local steroids with relative risk .

**CONCLUSION**

PN is a eluding subject, but when managed correctly with diagnostic tool with neurophysiological studies under trained neurophysiologists, the management is a child’s play. More so, the introduction of modern drugs like Epalrestat proves to be a boon in the management as on date, with added remyelinating, and pain alleviating drugs, like Methycober and Pregabalin allied drugs.

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