Original Research Article

# The effect of external pressure on nerve conduction in median nerve of healthy individuals and clinically diagnosed patients of carpal tunnel syndrome

Ribu Sam Stephen[1], A G Joshi[2], Basheer MP[3*]

[1]Assistant Professor, Department of Physiology, MES Medical College, Perinthalmanna, Malappuram, Kerala, INDIA.
[2]Professor, Department of Physiology, Krishna Institute of Medical Sciences, Karad, Satara, Maharashtra, INDIA.
[3]Associate Professor, Department of Physiology, Al Azhar Medical College and Super Specialty Hospital, Thodupuzha, Kerala, INDIA.
**Email:** drbasheermp@gmail.com

## Abstract

This study examines the evidence for the ill effects of external pressure on nerve conduction in median nerve and it's after effects in Carpal Tunnel patients comparing to normal subject. Present work is undertaken to study the electrophysiological changes happening to the already compressed nerve when it is subjected to external compression using pneumatic tourniquet. With the help of it we are evaluating the extent of damage and deterioration occurring in median nerve of CTS affected patients compared to healthy individuals and there by trying to find out whether the median nerve of these patients are more susceptible to external compression as in the surgical use of tourniquet. This study may help for the better management of tourniquet, to determine the safe duration and pressure for CTS patients especially in surgeries for compression neuropathies and to reduce surgical failures due to it. **Methodology:** Electro-diagnostic studies were conducted on sixty healthy individuals and sixty Carpal Tunnel Syndrome patients which includes Motor and Sensory conduction of Median nerve on the right hand of the subjects and the patients. It is studied before application of pressure, after 15 minutes of 150 mm of Hg pressure application and after 15 min of rest on releasing the pressure. **Results:** In patients of CTS, significant difference was observed between before application of pressure and after 15 min of rest on releasing pressure in Motor conduction parameters such as a) Motor conduction velocity b) Amplitude of CMAP and c) Distant motor latency. There was very significant difference in all sensory parameters such as a) Sensory Conduction Velocity [SCV], b) Amplitude of SNAP and c) Distal Sensory Latency [DSL] between the initial reading and after 15 min of rest on release of pressure in CTS patients, where in controls this difference was not seen. Which suggests that, the fall in Sensory parameters after pressure was restored to normal level after 15 min of rest in controls, this change was not seen in patients since the changes occurred due to application of pressure was not reverting back. **Conclusion:** All these results indicates that the vascular changes due to external pressure causes a sustained ill effect on the median nerve of CTS patients comparing to the controls. In CTS Patients because of the ischemia and venous congestion the deterioration of nerve takes a more grievous course which is also rapid.

**Key Word:** Carpal tunnel syndrome, Median Nerve, Nerve conduction

**Address for Correspondence**
Dr. Basheer MP, Associate Professor, Department of Physiology, Azhar Medical College and Super Specialty Hospital, Thodupuzha, Kerala.
**Email:** drbasheermp@gmail.com
Received Date: 16/09/2018 Revised Date: 22/10/2018 Accepted Date: 07/11/2018
DOI: https://doi.org/10.26611/103822

Access this article online

Quick Response Code:

Website: www.medpulse.in

Accessed Date: 09 November 2018

Carpal Tunnel Syndrome is the commonest form of entrapment neuropathy which is the combination of symptoms and signs resulting from compression of the median nerve when it passes through the bony carpal tunnel, from forearm to palm. Common symptoms of CTS patients include dull aching pain in hand especially after prolonged work and numbness in the median innervated parts of the hand. The mean annual crude incidence of carpal tunnel syndrome was found to be 329 cases per 100,000 person-years, and the standardized

How to cite this article: Ribu Sam Stephen, A G Joshi, Basheer MP. The effect of external pressure on nerve conduction in median nerve of healthy individuals and clinically diagnosed patients of carpal tunnel syndrome. *MedPulse International Journal of Physiology.* November 2018; 8(2): 19-24. https://www.medpulse.in/Physiology/

incidence was 276 with prevalence in the general adult population ranging from 2.7 to 5.8 percent[1]. The worldwide incidence of CTS among computer users (both vocational and recreational) is estimated to be about 15-25%. CTS is more common in females than in males, its occurrence is generally bilaterally with a peak age range of 30 to 60 years[2]; although it occurs in all age groups. The Incidence of CTS is alarmingly increasing all over the world due to the increased use of computers and increased prevalence of obesity and diabetes mellitus[3,4]. Many of the CTS cases finally ends up in Carpal tunnel releasing surgery, which is one of the most frequently done hand surgery. During the procedure, to achieve bloodless fields, pneumatic tourniquets are applied. Recommended safe limits of an upper arm tourniquet is 50-75mm of Hg above systolic pressure for 60 minutes[5-7]. In CTS patients the median nerves are already compressed because of which they are more vulnerable to the ischemic effects of tourniquet compression. Modern pneumatic tourniquets are designed to minimize the incidence of potential complications, but their use is still associated with potentially serious morbidity and even mortality, Nerve palsy being one of the most frequently encountered complications of this procedure [8,9]. The potential for injury assumes startling proportions. Injuries resulting from pneumatic tourniquet uses are commonly pressure related, and can also be caused by excessive tourniquet time [10]. This study may help for the better management of tourniquet, to determine the safe duration and pressure for CTS patients especially in surgeries for compression neuropathies and to reduce surgical failures due to it.

## MATERIALS AND METHODS

The study was conducted in the electrophysiology lab of the department of physiology, Krishna Institute of Medical Sciences, Karad, Satara, Maharashtra. The study was conducted on sixty clinically diagnosed patients of Carpal Tunnel Syndrome who were referred to the department for nerve conduction and on sixty age and sex matched healthy individuals who were selected from students and staff of this institution. This study was approved by the Ethics committee of Krishna Institute of Medical Sciences, Karad and the patients and subjects were informed the detailed procedure and written consent was taken.

**Inclusion Criteria:** Sixty Patients clinically diagnosed based on the symptoms and signs suggestive of Carpal Tunnel Syndrome are selected for the study. Sixty apparently healthy age and sex matched Students/staff of Krishna Institute of Medical Sciences, Karad, without any other illness and neurological abnormality are selected as subjects.

**Exclusion Criteria:** Patients with hypertension, h/o fractures, skin graft, skin diseases, cardio-vascular diseases, sickle cell anaemia, pregnancy, peripheral artery diseases, diabetes, CTS patients with symptoms of polyneuropathy or radiculopathy, any other marked systemic or local abnormality were excluded from the study.

**Study procedure:** Electro-diagnostic studies was conducted on these sixty healthy individuals and sixty Carpal Tunnel Syndrome patients which includes Motor and Sensory conduction of Median nerve on the Right hand of the subjects and the patients. After a normal conduction study, 150mm of Hg pressure is applied on the Right arm of the subjects and the patients, using a sphygmomanometer cuff, for a period of 15 minutes. The pressure was released after 15 minutes and Nerve Conduction Study is repeated immediately. Nerve Conduction Study was repeated once again to know the rate of recovery After 15 minutes of pressure release. The following parameters are studied with electro-diagnostic test, which include Motor conduction (Distal motor latencies of Median nerve, Motor conduction velocity, Amplitude of compound muscle action potential, Difference between distal motor latencies of median nerve) and Sensory conduction **(**Distal sensory latencies of Median nerve, Sensory conduction velocity, Amplitude of Sensory nerve action potential and Difference between distal Sensory latencies of median nerve). For recording sensory and motor nerve conduction, surface metal electrodes were used. For recording motor conduction of Median nerve, recording electrode was placed close to the motor point of Abductor Pollicis Brevis and reference electrode 3cm distal to it at first metacarpophalangeal joint. A supramaximal stimulus was given at wrist and at elbow near volar crease of brachial pulse. For orthodromic sensory conduction of median nerve, surface recording electrode was placed 3cm proximal to distal wrist crease and surface reference electrode at 3cm proximal to recording electrode. For stimulation ring electrodes were fixed on second digit. During nerve conduction study, laboratory temperature was maintained between 21$^{o}$C to 23$^{0}$C.For nerve conduction studies, Recorder and Medicare System (RMS) machine from Chandigarh (India) was used.

**Statistical Analysis:** Statistical analysis was performed using the software origin-8 (Origin LAB, Origin Lab Corp., MA, USA).

## RESULTS

**Motor Conduction**: It was noted that motor conduction parameters such as; Motor conduction velocity and Amplitude of compound muscle action potential were greatly reduced and Distal motor latency greatly

increased after application of pressure. All these parameters came back to normal values after 15 min of rest on releasing the pressure in normal subjects but it was observed that it was not coming back to normal in CTS patients. Thus there was significant variation between before application of pressure (A) and after 15 minutes of rest on release of pressure (C) in patients and that of control was not significant (Table 1)

**Sensory Conduction:** It was noticed that, in CTS patients as well as in normal subjects, Sensory conduction velocity and Amplitude of SNAP were greatly reduced after application of pressure and Distal sensory latency was increased. Later on after 15min of rest on releasing the pressure it was found that the SCV, Amplitude of SNAP and DSL became normal back to the pre pressure application level in normal subject but it was not reverting back to normal in CTS patients. Very significant difference was observed between initial values (A) and after 15min of rest on releasing pressure (C) (Table 2).

**Table 1:** Motor Nerve Conduction parameters in Controls Versus Carpal Tunnel Syndrome patients

| | | A Vs B | | P value | A Vs C | | P value | B Vs C | | P value |
|---|---|---|---|---|---|---|---|---|---|---|
| Velocity | Patients | 50.57± 9.7 | 42.15±10.6 | <0.001 *** | 50.57±9.7 | 44.6± 9.9 | <0.05 * | 42.15± 10.6 | 44.6± 9.9 | >0.05 ns |
| [m/sec] | Controls | 60.2± 5.3 | 52.1± 5.7 | <0.001 *** | 60.2± 5.3 | 58.2± 5.2 | >0.05 ns | 52.1± 5.7 | 58.2± 5.2 | <0.001 *** |
| CMAP | Patients | 12.8 ± 4.7 | 10.3 ± 4.4 | <0.01 ** | 12.8 ± 4.7 | 10.8 ± 4.9 | <0.05 * | 10.3 ± 4.4 | 10.8 ± 4.9 | >0.05 ns |
| [mV] | Controls | 17.2 ± 4.5 | 15.2 ± 4.3 | <0.01 ** | 17.2 ± 4.5 | 16.4 ± 5.1 | >0.05 ns | 15.2 ± 4.3 | 16.4 ± 5.1 | <0.05 * |
| DML | Patients | 3.94± 1.07 | 4.66± 1.05 | <0.01 ** | 3.94±1.07 | 4.53± 1.1 | <0.05 * | 4.66± 1.05 | 4.53± 1.1 | >0.05 ns |
| [ms] | Controls | 2.72± 0.42 | 3.15± 0.45 | <0.001 *** | 2.72±0.42 | 2.8± 0.39 | >0.05 ns | 3.15± 0.45 | 2.8± 0.39 | >0.001 *** |

Before application of pressure [A], immediately after 15 minutes of 150mm of Hg pressure [B] and after 15 minutes of rest on release of pressure [C].*** Highly significant,** Very significant,* Significant, Ns: Non significant

**Table 2:** Sensory Nerve Conduction parameters in Controls Versus Carpal Tunnel Syndrome patients

| | | A Vs B | | P value | A Vs C | | P value | B Vs C | | P value |
|---|---|---|---|---|---|---|---|---|---|---|
| Velocity | Patients | 40.34± 7.5 | 33.98± 8.3 | <0.001 *** | 40.34± 7.5 | 36.22± 6.9 | <0.01** | 33.98± 8.3 | 36.22± 6.9 | >0.05 ns |
| [m/sec] | Controls | 57.99± 4.8 | 50.8± 9.98 | <0.001 *** | 57.99± 4.8 | 56.1± 4.4 | >0.05 ns | 50.8± 9.98 | 56.1± 4.4 | <0.01 ** |
| SNAP | Patients | 16.9± 10.2 | 10.37± 7.2 | <0.001 *** | 16.9± 10.2 | 13.08±9.02 | <0.01 ** | 10.37± 7.2 | 13.08±9.02 | >0.05 ns |
| [µV] | Controls | 33.4± 11.4 | 26.06±10.5 | <0.001 *** | 33.4± 11.4 | 31.1± 11.7 | >0.05 ns | 26.06±10.5 | 31.1± 11.7 | <0.001*** |
| DSL | Patients | 3.07± .97 | 3.83± 1.1 | <0.001 *** | 3.07± .97 | 3.72± 1.2 | <0.01 ** | 3.83± 1.1 | 3.72± 1.2 | >0.05 ns |
| [ms] | Controls | 2.3± 0.14 | 2.52± 0.26 | <0.001 *** | 2.3± 0.14 | 2.37± 0.2 | >0.05 ns | 2.52± 0.26 | 2.37± 0.2 | >0.001*** |

Before application of pressure [A], immediately after 15 minutes of 150mm of Hg pressure [B] and after 15 minutes of rest on release of pressure [C]. *** Highly significant,** Very significant, * Significant, Ns: Non significant

## DISCUSSION

This study was done in point of view that the incidence of CTS is increasing in India and all over the world due to the wide use of computers and increase in other risk factors. Most of the cases finally end up in an Open release surgery in which we very commonly use pneumatic tourniquets to get bloodless fields, knowing its potential complications but believing in those guidelines for safer usage. Earlier clinical studies say that the already compressed median nerves of CTS patients are more susceptible to ischemia and this makes those nerves more vulnerable to external pressures. This could be a reason for delay in recovery or permanent damages after open release surgeries using pneumatic tourniquet. The mean INTRACARPAL PRESSURE: in the normal subjects with the wrist in neutral position is 2.5 millimeters of mercury (standard deviation, 0.61; range). With palmar flexion the pressure becomes thirty-one millimeters of mercury (standard deviation, 3.0; range), and with dorsiflexion it rose to thirty millimeters of mercury (standard deviation, 4.3), whereas in CTS patients the mean elevation of pressure with the wrist in neutral position was found to be thirty-two millimeters of mercury[11]. Marked elevation of pressure was noted in the patients when the wrist was in flexion (mean, ninety-four millimeters of mercury) and in extension (mean, 110 millimeters of mercury). Compression of a nerve affects intraneural blood flow. Pressures as low as 20-30 mm Hg can retard venular blood flow in a nerve[11]. Axonal transport is impaired at 30 mm Hg. Neurophysiologic changes manifested as sensory and motor dysfunctions are present at 40 mm Hg. Further increases in pressure produce increasing sensory and motor block. At 60-80 mm Hg, complete cessation of intraneural blood flow is observed. As we have mentioned before the carpal canal pressures in patients with CTS averaged 32 mm Hg which is significantly elevated during flexion and extension (90-110mm of Hg), compared with only about 2 mm Hg in control subjects. And earlier studies prove that the signs and symptoms of CTS are mainly due to ischemia to the nerve. Thus the increase in carpal tunnel pressure resulting in ischemia is the reason for all the clinical and physical changes occurring distal to the pressure[11]. The

pathophysiology of carpal tunnel syndrome (CTS) is typically demyelination[12,13]. In more severe cases, secondary axonal loss may be present. The most consistent findings in biopsy specimens of tenosynovium from patients undergoing surgery for idiopathic CTS have been vascular sclerosis and edema. Localized amyloid deposition in the tenosynovium also has been reported in persons with idiopathic CTS. Signs of damage appeared early in the compressed segment which became oedematous and infiltrated with lymphocytes and macrophages. The compressed segment was greatly narrowed with oedematous tissue on either side of the constriction. The affected nerve fibres showed swelling, notching, and vacuolation of the axons, and a change in their staining qualities. The myelin showed granulation, fissuration, and vacuolation, particularly in the vicinity of the nodes and Schwann cells[14]. These changes finally resulted in parts of the axon becoming bared or being left with only a thin coat of myelin. There was no, or very little, Schwann cell activity. The nerve fibres appeared normal below the site of compression, though beading of the axons and myelin was observed in some specimens. Conduction through the affected segment was blocked for long periods[14]. Some scientists strongly suggested the possibility of structural damage due to mechanical compression and few others observed the coexistence of both mechanical compression and ischemia[15,16,17]. Pressure obstruction to the venous return from the nerve originating in this way would lead to hyperaemia, venous congestion, and circulatory slowing in the epineurial and intrafunicular tissues. With increasing pressure in the tunnel these circulatory disturbances worsen and ultimately lead, both directly and indirectly, to pathological changes in the nerve, the most damaging of which take place inside, and not external to, the funiculi[14]. When the pressure increases the capillary circulation slows to a point where the resulting anoxia damages the capillary endothelium, which leads to the leakage of protein into the tissues, which become oedematous. The capillaries of the epineurium are affected earlier than those inside the funiculi. On the other hand, the consequences are far more serious in the case of the funiculi. Here, protein steadily accumulates in the endoneurial spaces because it cannot escape across the perineurium and the endoneurial tissue becomes increasingly oedematous. As it progress intrafunicular edema is resulted[14,15]. Therefore, intrafunicular oedema, swollen endoneurial spaces, and distended funiculi, together with epineurial oedema, which are collectively responsible for the enlargement of the nerve observed at the margins of the retinaculum. Damming of the axoplasmal flow, extrusion of axoplasm, and nodal displacement and telescoping of the axon and myelin would not, alone, be sufficient to account for the large swellings that are occasionally seen above the retinaculum. These changes are aggravated during the application of external pressure but could be resolved when the pressure is released. The recovery from any motor or sensory loss present before the operation is delayed depending on whether individual nerve fibres have sustained first or second degree damage[14]. If long-standing pressure in the tunnel is allowed to continue, the lesion takes on a more permanent state to stage 3. Fibroblasts start to proliferate in the protein exudate. This commencing intrafunicular fibrosis is irreversible and is associated with the destruction of increasing numbers of nerve fibres. The final stage is reached when nutrient vessels are obliterated and the affected segment of the nerve becomes converted into a fibrous cord in which only a few fine nerve fibres survive inside fibrosed funiculi which encased in a now dense relatively avascular epineurium[14,15]. Attempts at regeneration through this tissue are rarely successful because of its length and density. Most regenerating axons terminate at the proximal margin of the retinaculum to contribute to the swelling of the nerve at that site. With the degeneration of nerve fibres the perineurium contracts about its contents and the funiculus shrinks. The effects of this thinning on the general appearance of the nerve trunk are determined by the relative amounts of funicular and epineurial connective tissue originally constituting the nerve in the tunnel (Sunderland and Bradley, 1950a, b). Because this is a variable feature, the cross-sectional dimensions of the nerve after funicular atrophy will vary from individual to individual[14,15,16]. With a progressive lesion which continues to reduce the cross-sectional area of the carpal tunnel, a stage will be reached when the deforming forces generated in the tunnel[1] interfere with not only the venous return from the nerve but also the blood supply to it, and[2] contribute directly to the lesion by mechanically deforming the entire nerve trunk[14]. One possible explanation for the slowing of conduction would be that repeated attacks of ischemia destroy the larger fibres in the nerve trunk leaving only the smaller fibres capable of conducting impulses[13] The Amplitude depends upon the number of nerve fibres. As more and more nerve fibres get affected the amplitude also drops proportionately. Reduction in the number of active motor units due to median nerve damage will also result in a fall in amplitude[13,14,18]. The primary factor accounting for observed functional abnormalities in human nerves compressed by the pneumatic tourniquet is ischaemia. The reduction in nerve conduction velocity observed in the present study during 15 min of arterial occlusion may be predominately due to other factors besides oxygen lack, for the latter condition was reached early in the

ischemic state. Moreover, it is difficult to conceive of the ATP store in large myelinated nerves, of the type studied in this investigation, being materially depleted during so short a period of hypoxia. A possible mechanism could have been an alteration in extracellular-intracellular electrolyte concentrations, such as a build-up of extracellular potassium, originating from the nerves themselves and from the surrounding muscles and other tissues. Such a situation would have led to a relative inactivation of sodium conductance, followed by a progressive reduction in nerve conduction velocity. Resulting changes in the concentration of metabolites in extracellular fluid (an increase in lactic acid and carbon dioxide, a decrease in pH, etc.) could also have contributed to the observed alterations [17,19,20,21,22,23,24]. The changes occurred in patients after application of pressure were highly significant [p= <0.0001] compared to the changes observed in Controls. This could be due to: The external compression aggravates the condition by increasing the venous congestion at the periphery, increasing anoxia which damages the capillary endothelium that leads to leakage of protein into the tissues and resulting in edema[12]. This still further embarrasses conduction in the nerve fibres, and may even threaten their survival by:[1] interfering with their nutrition and metabolism;[2] compression secondary to the increase in the intrafunicular pressure which is maintained by the tensile strength of the perineurium; and,[3] promoting the proliferation and increased activity of fibroblasts with the formation of constrictive endoneurial connective tissue[14]. Intrafunicular and epineurial edema accelerate the structural changes by increasing the extrusion of axoplasm, nodal displacement and telescoping of the axon and myelin. With these advanced structural changes the sensory and motor deficit now deepens as more and more fibres suffer a conduction block. MCV, SCV, Amplitude of CMAP, SNAP, DML and DSL were found to be significantly different before application of pressure and after 15min of rest (p=<0.05) on releasing the pressure in CTS patients. There was no difference seen in Controls (P=>0.05). These findings shows that the changes due to application of pressure are reverting back to initial normal level in Controls after 15 minutes of rest whereas it was not recovering in CTS patients. In Controls: all the changes that happened due to application external compression were transient. Those changes were reverting back very fast when the pressure was released. When the circulation is re-established, there was no more anoxia, the nutrition is returned and all the metabolic and electrolytic changes became normal. This shows that 15min of 150mm of Hg is not enough to cause any grievous effects on the nerves in a normal adult individual. In CTS Patients: there are two processes concerned in the carpal tunnel syndrome, one being ischemia occurring in attacks which cause pain, paraesthesiae, and transient weakness with reversible failure of nerve conduction, and the other direct pressure producing a slow change in the nerve fibres with reduction of fibre diameter and increase in latency. This concept would explain the dissociation frequently observed between motor latency and susceptibility to ischemia. While this dissociation may be seen at any time during the course of the disease it is particularly obvious following surgical division of the retinaculum. Attacks of pain are immediately relieved by operation, whereas the recovery of conduction velocity is a slow and progressive change, sometimes taking as long as 18 months after operation in severe cases[13]. We have already discussed the pathophysiological changes occurring in CTS patients due to ischemia and structural changes due to direct compression and the vascular changes. We have also discussed how external pressure worsens the condition. After 15 minutes of rest on releasing pneumatic tourniquet pressure the electrolyte and metabolic imbalance are corrected to some extent but because of the still persisting raised carpal tunnel pressure, the capillary circulation is not fully re-established and the venous congestion and edema may not be subsided. More importantly the structural deterioration due to the vascular changes on application of pneumatic tourniquet takes lot more time to get corrected. External pressure application not only worsens the structural damage it also affects more and more fibres[13]. This why the electrophysiological parameters are not coming back to normal in CTS patients even after 15 min of rest.

## CONCLUSION

Results of this study indicates that the vascular changes due to external pressure causes a sustained ill effect on the median nerve of CTS patients comparing to the controls. In CTS Patients because of the ischemia and venous congestion the deterioration of nerve takes a more grievous course which is also rapid. Our study shows that the median nerves of CTS patients are more susceptible to the hazardous and injurious effects of pneumatic tourniquet compression. So the upper limit of for safe duration and pressure in pneumatic tourniquet application should be less than that of normal individuals.

## REFERENCES

1. Atroshi I, Gummesson C, JohnssonR, Ornstein E, Ranstam J, Rosén I. Prevalence of carpal tunnel syndrome in a general population. I. JAMA. 1999 Jul 14; 282(2):153-8.
2. Ibrahim I, Khan WS, Goddard N, Smitham P. Carpal tunnel syndrome: a review of the recent literature. Open Orthop J. 2012; 6:69-76.

3. Violante ES, Mattioli S, Camagni A, Bottoli E, Farioli A, BonfiglioliR. Assessment of fitness for work in health care workers: biomechanical riskfactors. Med Lav. 2012 May-Jun; 103(3):198-202
4. Nordstrom DL, Vierkant RA, DeStefano F, Layde PM. Risk factors for carpaltunnel syndrome in a general population. Occup Environ Med. 1997Oct; 54(10):73440.
5. Barash, Cullen, Stoelting. Clinical Anaesthesia. 2nd ed. Philadelphia: Lippincott, 1992.
6. Rogers, Tinller, Covino, Louguedler. Principles and Practice of Anaesthesiology. New York: Mosby, 1993.
7. Odinsson A, Finsen V. The position of the tourniquet on the upper limb. J Bone Joint Surg Br. 2002 Mar; 84(2):202-4. Erratum in: J Bone Joint Surg Br. 2003 Jan; 85(1):153.
8. Colin G. MURPHY, Desmond C. WINTER, David J. BOUCHIERHAYES. Tourniquet injuries: pathogenesis and modalities for attenuation. ActaOrthop. Belg., 2005, 71, 635-645
9. Livia S Malanjum and Barrie Fischer. Procedures under tourniquet. Available from www. Tourniquet.com dated 10 November 2006.
10. McEwen JA. Complications of and improvements in pneumatic tourniquets used in surgery. Medical Instrum. 1981; 15(4):253-257.
11. Gelberman, RH, Hergenroede PT, Hargens AR, Lundborg GN and Akeson WH, The carpal tunnel syndrome. A study of carpal canal pressures. J Bone Joint Surg Am. 1981; 63: 380-383.
12. Geethanjali Sharma, SushmaSood, Vivek Sharma. Early diagnosis of Carpal Tunnel Syndrome (CTS) in Indian patients by nerve conduction studies. Internal Journal of Medical Update2010 july;5(2): 15- 20
13. Pamela M. Fullerton. The effect of ischaemia on nerve conduction in the carpal tunnel syndrome. J. Neurol. Neurosurg. Psychiat., 1963, 26, 385
14. Sydney Sunderland, The nerve lesion in the carpal tunnel syndrome, Journal of Neurology, Neurosurgery, and Psychiatry, 1976, 39, 615-626
15. Fullerton, P. M., and Gilliatt, R. W. (1967a). Pressure neuropathy in the hind foot of the guinea pig. Journal of Neurology, Neurosurgery and Psychiatry, 30, 18-25.
16. Fullerton, P. M., and Gilliatt, RW. (1967b). Median and ulnar neuropathy in the guinea-pig. Journal of Neurology, Neurosurgery, and Psychiatry, 30, 393-402
17. Ochoa J,Fowler TJ and R. W. Gilliatt RW Anatomical changes in peripheral nerves compressed by a pneumatic tourniquet. J. Anat. (1972), 113, 3, pp. 433-455.
18. J. Grapperon, A. Trousset, S. Bellard, and L. Force,Effects of high pressure on nervous conduction velocity in man. Eur J ApplPhysiol (1988) 58:141-145
19. Parul Mittal*, ShwetaShenoy and Jaspal S Sandhu. Effect of different cuff widths on the motor nerve conduction of the median nerve: an experimental study. Journal of Orthopaedic Surgery and Research 2008, 3:1 doi: 10. 1186/1749-799X-3-1.
20. Abramson, David I., Barbara L. Rickert, Jennifer T. Alexis, Anna. The Effect of repeated periods of ischemia on motor nerve conduction velocity in forearm. J. Appl. Physiol. 30(5): 636-642. 1971.
21. Bentley, F. H. and Schlapp, W. (1943). The effect of pressure on conduction in peripheral nerve. Journal ofPhysiology (London) 102, 7282.
22. Fowler TJ., Danta G. andGilliatt, R. W. (1972). Recovery of nerve conduction after a pneumatic tourniquet: observations on the hindlimb of the baboon. Journal of Neurology, Neurosurgery and Psychiatry 35, 638-647.
23. Fowler TJ. (1973). Tourniquet Paralysis in the Baboon. D.M. Thesis, University of Oxford.
24. Causey Gand Palmer E The effect of pressure on nerve conductionand nerve-fibre size J. Physiol. (I949) I09, 220-23I 12.0I4.47:6I2.8I6.3.


Source of Support: None Declared
Conflict of Interest: None Declared