Original Article

Protective effect of glove on median nerve compression in the carpal tunnel

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Objectives: To determine the protective effect of gel padded glove on median nerve compression in the carpal tunnel.

Methods: Median nerve conduction parameters, skin temperature, laser Doppler flowmetry and pain modifications were measured during and after a 30-min carpal tunnel external compression protocol performed with and without glove in a random order on six healthy volunteers.

Results: Compression induced a rapidly reversible increase in sensory and motor distal latencies, a decrease in sensory amplitude, finger laser Doppler flowmetry and hand skin temperature supporting the hypothesis of a reversible conduction block of ischemic origin. There was no statistical difference between the tests (with or without glove) except for pain that was significantly reduced by glove protection.

Conclusion: Gel padded glove does not seem to have a protective effect on the carpal tunnel syndrome induced by compression but provides significant comfort.

Spinal Cord (2001) **39**, 215–222

Keywords: electromyography; median nerve; protective devices; spinal cord injury; laser doppler flowmetry

Introduction

Carpal tunnel syndrome (CTS) is the most commonly reported nerve entrapment syndrome especially in SCI patients. The diagnosis of CTS is based on clinical history, physical examination and electromyography. Excessive pressure applied onto the carpal tunnel is considered as the main provocative factor of CTS. Therefore, people who use manual wheelchairs for mobility assistance are particularly at risk. To prevent CTS in such subjects, protective gel padded gloves are usually proposed as a prophylactic or conservative treatment modality.¹ However, little is known about their real efficiency.

This study is designed to evaluate the modifications of median nerve conduction, hand vascularisation and pain consecutive to an external compression applied on the carpal tunnel with and without a protective gel padded glove.

Materials and methods

Subjects

Six healthy adults (four men, two women) volunteered for this study. Subject's age arranged from 22 to 25 years (24 ± 1 years, mean $\pm$ SD). No subjects had symptoms of peripheral nerve impairment or carpal tunnel syndrome. In all subjects antidromic sensory and motor distal latencies of the median nerve were normal. The hospital ethics committee approved the study, and a written informed consent was obtained from all subjects.

Experimental design (Figure 1)

The two tests (with and without glove) were performed in a random order in all subjects with at least a 1-week interval. The subjects were sitting in an air-conditioned room with a mean temperature of 26°C. Localized pressure was applied to the right carpal tunnel 2 cm below the wrist at the exact place where median nerve conduction block and demyelination are found in CTS. We used a piece of 2 cm² moulded rubber specially

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Figure 1 Experimental design: hand with gel padded glove, moulded rubber piece fixed on the dynamometer, SNAP and CMAP stimulation and detection electrodes and laser Doppler flowmetry on the finger and forearm

designed to stimulate the pressure applied on the hand during manual wheelchair propulsion. The external compression of 10 kg was continuously monitored by a digital dynamometer (Sensors and Synergy Model 2712 Belgium). The value of 10 kg was chosen because it is close to that obtained during push-up and transfer. We used a gel padded glove commercially available for disabled people (Smith & Nephew Rolyan). During the test with glove, the pressure was applied on the gel.

Before starting the experiment, during the 30 min of external compression and during the 45 min recovery phase, median nerve conduction studies, skin temperature, laser Doppler flowmetry and pain evaluated by the subjects on the visual analogic scale (VAS) were measured every 2 min and 30 s.

The same electromyographer performed median nerve conduction studies with a Viking I (Nicolet Biomedical, Inc., Madison, WI, USA). Antidromic sensory nerve action potential (SNAP) was obtained at the index and compound motor action potential (CMAP) was obtained at the abductor pollicis brevis muscle at the thumb after median nerve stimulation at the wrist respectively 13 and 5 cm proximal to the pickup electrodes. Inter electrode distance was 2 cm. SNAP distal latency at the onset (ms), negative peak amplitude (μ V), CMAP distal latency at the onset (ms), negative peak amplitude (mV) and negative peak

area (mVms) were measured. For distal latencies a temperature correction factor of 0.2 ms under 33°C at the hand was applied.² Skin surface temperature was measured by using the Multifeedback thermometer (J Tronic, Brussels) on the palm of the hand and on the forearm at 15 cm above the wrist. The thermometer had an accuracy of 0.1°C. Skin blood flow was recorded by a two-channel laser doppler flowmetry (LDF) Periflow 4001 device (Perimed, Stockholm) at the middle finger and at the forearm. The light source was a 635-nm diode laser. The maximum output power was 1 mW. The core of the probe (120 μ m) was composed of three parallel optical fibres. The light penetration depth was limited to 1 mm and the area of measurement was about 1 mm². The probe was placed in a plastic holder and fixed to the skin by a double-side adhesive ring. Blood flow is expressed in perfusion unit (PU). This relative value is equal to a calibrated output of 10 mV.

Data analysis

Data are expressed as mean values $\pm$ SD. The evolution of the parameters during compression was studied by regression analysis of repeated measures, using generalized estimating equations with the value of the parameters at the beginning of the experiment, the

time, the presence or absence of glove and the order of tests as covariate.³ This analysis was performed by use of the RMGEE program.⁴ Two-way analysis of variance for repeated measures was used to compare parameters in both groups at the beginning and the end of the experiment. All statistical tests are two-tailed.

Results

Figures 2 to 7 show the modifications of the SNAP distal latency and amplitude, CMAP distal latency, hand skin temperature, finger LDF and pain along the two tests.

Table 1 lists the means and the standard deviations of the SNAP distal latency at peak, amplitude, CMAP distal latency, negative peak amplitude and area, skin temperature at hand and forearm, LDF at the finger and forearm and pain score (VAS) at the beginning, after 25' of compression and after 45' of recovery in both tests. In four cases, the SNAP has disappeared 25 to 30 min after the beginning of the compression. Nerve conduction modifications can not be attributed to low skin temperature as a temperature correction factor has been applied. Clinical paresthesiae occurred with large variations between 1 to 28 min without differences between the tests. In only two cases, paresthesiae occurred after SNAP modifications. Surprisingly, it occurred in the two subjects whose

SNAP did not disappear. Compression induced a significant increase in SNAP ($P<0.001$) and CMAP ($P=0.002$) distal latency and VAS score ($P<0.001$) and a decrease in SNAP amplitude ($P<0.001$), hand skin temperature ($P<0.001$) and finger LDF ($P=0.029$) but no modification in CMAP amplitude and area, forearm skin temperature and forearm LDF. There was no difference between the tests (with and without glove) except for pain, which was evaluated at 35 ± 12 ($P<0.0001$) with glove and at 62 ± 10 without glove ($P<0.001$). The second test, in order of appearance, was less painful ($P<0.001$) probably due to habituation effect. All parameters are characterized by a slow progressive modification with a rapid recovery after release of compression within the first 2 min. Only pain took 15 min to disappear completely. After 45 min of recovery, all parameters were restored without statistical difference between the tests. No subjects experienced neurobiological deficit or pain subsequent to the completion of the protocols.

Discussion

Excessive pressure applied onto the carpal tunnel is considered as the main provocative factor of CTS. Repetitive hand motion, awkward posture, vibrations and mechanical compression at the base of the palm in association with personal factors and blood volume movement increased the pressure in the carpal tunnel.

Figure 2 Mean $\pm$ SEM SNAP distal latency modifications during and after carpal tunnel compression with and without glove

Figure 3 Mean $\pm$ SEM SNAP negative peak amplitude modifications during and after carpal tunnel compression with and without glove

Figure 4 Mean $\pm$ SEM CMAP distal latency modifications during and after carpal tunnel compression with and without glove

Figure 5 Mean $\pm$ SEM hand skin temperature modifications during and after carpal tunnel compression with and without glove

Figure 6 Mean $\pm$ SEM finger laser Doppler flowmetry modifications during and after carpal tunnel compression with and without glove

Figure 7 Mean $\pm$ SEM pain score modifications during and after carpal tunnel compression with and without glove

Table 1 Means and standard deviations of median SNAP distal latency and amplitude, CMAP distal latency, amplitude and area, skin temperature at hand and forearm, laser Doppler flowmetry (LDF) at finger and forearm and pain (VAS) at the beginning (T0), after 25' of compression (T25) and after 45' of recovery (T70)

	T0		T25		T70	
	Without glove	With glove	Without glove	With glove	Without glove	With glove
SNAP distal latency (ms)	2.9 $\pm$ 0.2	2.9 $\pm$ 0.2	4.0 $\pm$ 0.8	4.0 $\pm$ 0.3	2.9 $\pm$ 0.2	3.0 $\pm$ 0.4
SNAP amplitude (μ V)	57.6 $\pm$ 12.5	55.1 $\pm$ 10.1	27.9 $\pm$ 18.4	27.4 $\pm$ 13.9	55.6 $\pm$ 19.1	59.1 $\pm$ 10.2
CMAP distal latency (ms)	3.1 $\pm$ 0.4	3.1 $\pm$ 0.2	3.2 $\pm$ 0.4	3.3 $\pm$ 0.3	3.1 $\pm$ 0.2	3.0 $\pm$ 0.2
CMAP amplitude (mV)	20.5 $\pm$ 1.9	22.3 $\pm$ 5.8	22.4 $\pm$ 1.9	21.7 $\pm$ 5.2	19.4 $\pm$ 4.0	20.8 $\pm$ 5.0
CMAP area (mVms)	56.8 $\pm$ 14.4	63.8 $\pm$ 25.4	60.0 $\pm$ 17.9	85.3 $\pm$ 44.5	49.2 $\pm$ 14.3	73.5 $\pm$ 35.4
TEMP hand ($^{\circ}$ C)	33.5 $\pm$ 0.8	33.7 $\pm$ 1.7	30.6 $\pm$ 1.5	31.7 $\pm$ 1.8	33.4 $\pm$ 0.7	32.8 $\pm$ 3.0
TEMP forearm ($^{\circ}$ C)	32.8 $\pm$ 0.7	32.4 $\pm$ 0.8	32.5 $\pm$ 0.8	31.9 $\pm$ 1.0	32.4 $\pm$ 1.0	31.5 $\pm$ 1.1
LDF finger (PU)	108 $\pm$ 74	95 $\pm$ 123	12 $\pm$ 7	5 $\pm$ 3	112 $\pm$ 79	103 $\pm$ 114
LDF forearm (PU)	10 $\pm$ 8	6 $\pm$ 2	13 $\pm$ 11	7 $\pm$ 4	11 $\pm$ 10	11 $\pm$ 10
Pain (VAS)	0 $\pm$ 0	0 $\pm$ 0	61 $\pm$ 10	35 $\pm$ 12	2 $\pm$ 3	0 $\pm$ 1

Gelberman measured an intracarpal tunnel tissue pressure of 32 mmHg in non-paraplegic CTS patients compared to 2.5 mmHg in control subjects.⁵ In SCI patients, forced dorsal flexion of the wrist during transfer and repetitive trauma on the pushrim during wheelchair propulsion are added specific factors leading to a higher incidence estimated at 40% to 55%.⁶⁻¹⁰ Surprisingly, wheelchair athletes do not seem to be more affected by CTS than sedentary paraplegics suggesting that transfer and wheelchair propulsion technique may be more important than the duration of the propulsion.^{10,11} As pointed out by Boninger, this explanation is attractive because it implies that paying

more attention to the fit of the wheelchair and to the technique of transfer and propulsion may be useful to prevent CTS.¹⁰ For instance, transfer with the fist, when possible, is probably at lower risk of CTS than transfer with forced dorsal flexion.

To avoid CTS it is also usually proposed to wear gel padded gloves specially designed to protect the palms of the hand from external compression.¹ In order to reproduce the physiopathological mechanism leading to CTS, we have chosen the experimental model of acute pressure previously described by Lundborg.¹² Unfortunately, this model cannot produce vibrations and this is the main methodological bias of our

experiment. Lundborg found that compression applied inside the carpal tunnel at 60 and 90 mmHg induces a complete sensory block within 30 to 50 min preceding a motor block by 10 to 30 min.¹² We found that external compression applied directly onto the CT induced a progressive increase in median nerve SNAP distal latency and a decrease in SNAP amplitude suggestive of a sensory conduction block, which was complete in four subjects after 30 min. The CMAP distal latency was increased in a lesser proportion probably because our experiment was stopped after 30 min. The greater sensibility of sensory fibres to compression is consistent with the clinical observation that sensory qualities are the first affected in the nerve entrapments. Moreover, clinical paresthesiae generally occurred before nerve conduction modifications. This is an argument to inform patients to release compression when paresthesiae arise.

Median nerve conduction modifications were associated with a decrease in skin blood flow measured with hand skin temperature and finger LDF supporting the general opinion that nerve conduction block caused by compression is due to ischemia.^{13–15} Different facts support the hypothesis of an ischemic reversible conduction block. First, pressure applied to nerve fibres induces an increase in endoneurial fluid pressure interfering with intrafascicular capillary flow. Capillary ischemia and impaired oxygenation induce secondary alterations in the function of nerve fibers exposed to compression.¹⁵ Secondly, inflating a pneumatic cuff around the upper arm induces a conduction block (or maintains a conduction block induced by compression) which is similar to that obtained by local compression and which is rapidly reversible after the release of the cuff.^{12,13} Third, the rapid recovery of the nerve conduction after the release of compression suggests a rapidly reversible phenomenon consistent with a microvascular impairment interfering with nerve fibers conduction.¹⁶ This recovery seems to be impaired in CTS as Rosecrance found that the necessary time for SNAP amplitude to return to 95% of the preprovocative amplitude was no longer than 0.62 min in normal subjects and 2.25 in confirmed CTS.¹⁷ Last, we found that skin blood flow decreased during compression and returned to baseline values after compression release in parallel with nerve conduction parameters. This suggests a rapidly reversible phenomenon probably of ischemic origin as nerve conduction and skin blood flow resolve simultaneously.

The most important observation is that, for all nerve conduction and skin blood flow parameters, during or after compression, there was no difference between the tests. Only the pain was significantly reduced with glove protection. This suggests that wearing a protective gel padded glove does not seem to provide significant protection for the CTS induced by compression but significant comfort. Lundborg noted that the sequence of events at 60 and 90 mmHg was not significantly different while a 30-mmHg

compression caused mild nerve conduction changes and paresthesiae.¹² This probably suggests a critical level of pressure localised around 30 mmHg above which conduction block and clinical symptoms appear. We hypothesised that gel padded glove can probably reduce the compression applied on the CT by distributing the pressure on a larger contact zone but not sufficiently to reach this critical value. To our knowledge, only Burnham¹ evaluated protective effect of glove on median motor and sensory nerve conduction parameters. A conduction block was found as a result of wheelchair propulsion but not cycling and glove was found to have no protective effect. However, the delay between compression and nerve conduction studies was about 20 min in the Burnham's experiment so that the values found may have underestimated the true extent of the conduction block.

We conclude that gel padded gloves do not seem to provide significant protection for the CTS induced by compression. However, they provide significant comfort, a better grip on the pushrim and probably reduce vibrations so that we still propose our SCI patients to wear gloves. But one should be aware that their capability to prevent CTS is probably limited and that education to transfer and wheelchair propulsion is an important point of the rehabilitation program with regard to the CTS prevention.

To confirm our experiment, it would be interesting to compare the prevalence of CTS in two groups of SCI patients, one with gloves and one without.

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