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Short Communication

The type of neuropathy in non-freezing cold injury

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ABSTRACT

The purpose of this study is to classify the neuropathy observed in cold injuries. The electrodiagnostic studies in 6 patients with chronic neuropathic sequelae of non-freezing cold injuries are presented. The neuropathy is a mixed motor sensory, demyelinating-axonal neuropathy. The findings are concordant with descriptions of clinical cases and of experimental studies in the limbs of rabbits, showing failure of conduction and distal degeneration of the affected fibers. Nerve conduction studies may provide an objective indicator of nerve damage in intact organisms affected with cold injury, and it may be of value in monitoring clinical, therapeutic and experimental studies of cold injury. The best therapeutic measures include prevention of prolonged exposure to cold temperatures, the use of protective equipment, rapid warming, and tissue care.

KEYWORDS: immersion foot, axonal neuropathy, demyelinating neuropathy, non-freezing cold injury, nerve conduction studies, electrodiagnostic studies.

INTRODUCTION

Prolonged exposure of a limb to cold produces a spectrum of changes; and the lower temperatures induce the most severe damage. In general, cooling of skin with ice reduces tactile sensibility and the perception of pain. The cold induces anesthesia by reducing the sensibility of cutaneous receptors in the terminal arborization of pain fibers. Then there is pain, tingling, paresthesias and burning sensation. Most severe exposures to cold temperatures for prolonged periods largely stem from the demands and inclemency of war, with significant morbidity; and the spectrum goes from trench foot and immersion foot/hand (at temperatures insufficient to cause tissue freezing) to frostbite (at temperatures when the tissue freezes below minus 2.5 degrees C).

BACKGROUND

Nerve conduction studies may be of value in monitoring clinical, therapeutic and experimental studies on cold injuries. In conventional electrodiagnostic testing there is prolongation of distal motor and sensory latencies when skin temperature is below 35 degrees. Cooling increases amplitude and duration of compound muscle action potentials (CMAPs), and sensory nerve action potentials, and increases dispersion of CMAPs. Cooling also slows sodium channel opening and closing with consequent reduction in phase cancellation [1-3]. Nerve conduction velocity is reduced by cooling, perhaps due to failure of the action potential advancing, due to reduction of kicks of inward membrane current at nodes of Ranvier below the critical value that is required to excite the next nodule. Conduction is more affected in large myelinated fibers than in small myelinated fibers [4]. No consistent changes in conduction are observed in unmyelinated fibers [5]. Previous reports of electrodiagnostic studies of cold injuries in humans are scanty and insufficient to characterize the type of neuropathy observed in these patients [5, 6]. This report is an attempt to characterize the type of peripheral neuropathy produced in humans by non-freezing cold injury, utilizing the electrodiagnostic information obtained from 6 patients who had peripheral neuropathy as

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a sequela of non-freezing cold injury. These patients were seen in a period that spans from 1970 to 2010.

METHODS

All these patients had been in active duty in the United States Army at the time of the cold injury and had no other disease that can account for their clinical peripheral neuropathy. Their cold exposures were considered immersion foot at temperatures insufficient to cause tissue freezing; and they have continued complaining of constant pain interfering with ambulation, and disabling paresthesias. Patient 1 was seen at age 75, and had been in active duty during World War II at age 19 and assigned to Europe. Physical findings include trophic skin changes, stocking level of hypesthesia and absent ankle jerks. Patients 2, 3, and 4 were veterans of the Korean War in 1951 and their age at the time of cold exposure ranged from 20 to 22 years. They had no significant motor deficit but ankle jerks were absent. Most of them had scaling, exfoliative, itching hyper pigmented rash in legs and feet and absent ankle jerks. Sensory deficit included hypesthesia in both feet. Patients 5 and 6 were seen by Hanifin and Cuetter in 1974 [6]. They were medically evacuated to Japan from Vietnam because of persistent foot pain interfering with ambulation. These men were part of a group of 11 who had spent ten days on a mission in Vietnam's central highlands five weeks earlier. They had been exposed to constant rain with temperatures ranging from 35 to 40 degrees Fahrenheit. On return to base camp all noted pain and swelling of the feet to a degree that the boots had to be cut off. Edema subsided after one week and the skin desquamated, but pain persisted to a disabling degree.

Nerve conduction studies and needle electromyography were performed with conventional methods with consideration of all biologic and technical variables including skin temperature, use of supramaximal stimulation, accurate distance measurement, and accurate stimulation [3]. Monopolar needle electrodes were used for needle electromyography.

RESULTS

The results of the nerve conduction studies are summarized in Table 1. Motor nerve conduction studies showed prolonged F waves and nerve conduction times (latencies), reduced amplitude of CMAPs with and without temporal dispersion, and slow nerve-conduction velocities. Most sensory conductions were absent. All these findings are compatible with a motor-sensory, demyelinating-axonal neuropathy of affected limbs (Note: Prolonged proximal and distal latencies, temporal dispersion of CMAPs and slow nerve-conduction velocities are features of a demyelinating neuropathy; and reduction of CMAP amplitudes without temporal dispersion, and relative preservation of conduction velocities are features of axonal neuropathy). Needle electromyography in all cases showed rare fibrillation activity upon insertion of the electrode. The recruitment of motor unit action potentials was decreased; and remaining potentials showed a rapid firing of about 15-20 Hz with increased number of large polyphasic potentials. Paucity of fibrillation activity with a neurogenic pattern of reinnervation correlates with the nerve conduction findings.

DISCUSSION

Many soldiers during World War II, the Korean War and Vietnam's central highland campaigns have suffered cold injuries including frostbite and non-freezing cold injuries [6, 7]. Documentations of many cases may be lacking because of battlefield conditions. There are well-recognized long-term and delayed sequelae to cold injuries including peripheral neuropathies. The electrodiagnostic studies can distinguish the presence of a neuropathy from other types of neurogenic injury. These electrodiagnostic studies of 6 cases support the view that the neuropathy is a mixed motor sensory, demyelinating and axonal neuropathy. The findings are concordant with descriptions of clinical cases and of experimental studies in the limbs of rabbits showing failure of conduction and distal degeneration of the affected fibers [4, 8-10]. In distal fibers, prolonged latencies of somatosensory evoked potentials obtained by tactile stimulation, and lack of stain on immunohistochemical assessments suggest that cold injury may produce pronounced pathologic changes in the very distal portion at the nerve fiber touch-receptor junction [11, 12].

Pathologically, the lower the temperature the greater the damage [13].There is endoneurium vasogenic

Table 1. Nerve conduction studies.

Nerve	Distal motor latency	Proximal motor latency	Amplitude motor (mV) Dist/Prox	Sensory conduction		Nerve conduc.velocity (M/S)	F wave (ms)
				Distal Latency (ms)	Ampl. (µV)		
Patient 1:							
R. peroneal	7.2	15.5	2/1.6			33	63
R. sup. peroneal				Absent			
L. peroneal	8.1	16	1.8/1.5			31	54
R. tibial	11	22	0.7/1			31	61
L. tibial	8.5	20	1.2/0.6*			34	57
L. sural				Absent			
R. ulnar	3.3	12	7.5/7			56	29
R. ulnar				2.9	34µ		
Patient 2:							
R. peroneal	Absent	Absent					Absent
R. tibial	8.9	17	08/06*			33	Absent
L. peroneal	7.2	17.8	0.9/06			25	Absent
R. sural				Absent			
R. ulnar	3.9	9.5				49	
Patient 3:							
R. peroneal	6.8	15	2/1.5*			35	52
L. peroneal	6.2	16	2.2/1.9*			33	49
R. sural				Absent			
Patient 4:							
R. peroneal	5.5	13.1	2.2/1.8			39	63
L. peroneal	6.2	13.6	2.4/2			39	49
L. tibial	5.5*	14.7	4/3.7			42	57
R. sural				Absent			
L. sural				5.4	7		
R. median				4.8	43		
R. ulnar				4	31		
Patient 5:							
R. peroneal	Absent						
L. peroneal	Absent						
L. sural				Absent			
Patient 6:							
R. peroneal	8.5	20	0.6/0.5		23		
L. peroneal	8.9	1.8	1/0.7		25		
R. sural				Absent			

(*)Temporal dispersion of compound muscle action potential.

edema as the prelude for axonal swelling, axonopathy, and the loss of myelinated fibers. Macrophages in the endoneurium contain myelin debris. There is shrinking of nerve fibers followed by gangrene and necrosis.

CONCLUSION

The electrodiagnostic studies in 6 patients with chronic neuropathic sequelae of non-freezing cold injuries were presented. The neuropathy is a mixed motor sensory, demyelinating, axonal neuropathy. The characterization of the neuropathy by nerve conduction studies may provide an objective indicator of nerve damage in intact organisms affected with cold injury; and it may be of value in monitoring clinical, therapeutic and experimental studies of cold injury. The best therapeutic measures are prevention of cold injuries, the use of protective equipment, rapid warming and tissue care.

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CONFLICT OF INTEREST STATEMENT

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