# FOOTDROP IN THE FARMERS: CLINICAL AND ELECTROMYOGRAPHICAL STUDY

**M. Ghaffarpour, A. Dolatabadi and M.H. Harirchian**

Department of Neurology, Imam Khomeini Hospital, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran

***Abstract-*** ***Footdrop is a relatively common deficit among the neurological disorders, which has different causes with various levels of involvement in neuromuscular system, including central nervous system (brain cortex, spinal cord), fifth lumbar root, peripheral nerves and muscles. Peroneal nerve injury at the fibular head has been reported to be the most common cause of footdrop, which can be due to infarct, tumor or leprosy but the vast majority of lesions are traumatic .In this article, we report seventeen patients with footdrop in farmers . All of the patients except one, were male with age ranges between 15 to 25 years. They had been doing certain farming activities (harvesting or weeding ) for 1–5 days before developing footdrop. Electrophysiological studies have been done in only seven of them due to patient's unwillingness. Nerve conduction velocity and amplitudes distal to the fibular head were normal, but stimulation above the fibular head showed reduced nerve conduction velocity and amplitudes (mean 22.4 m/s in the abnormal side versus 51.5 m/s in the normal side, mean peak to peak amplitude 3.6 mv in the symptomatic side versus 10.4 in the contralateral side respectively). Forty-three percent of patients had also conduction block. F wave latency increased on the affected side in comparison to the normal side (mean 4.7 m/s). The new and perhaps interesting findings in our cases are unilateral involvement and occurrence of peroneal palsy on the side of dominent hand, indicating that type of the hand activity is probably more important in inducing footdrop than the position of seating during harvesting or weeding. We suggest further investigation in this setting in order to find the mechanisms of nerve injury and prophylactic measures.***

***Acta Medica Iranica, 40(4);214-218: 2002***

***Key Words:*** ***Footdrop, peroneal nerve palsy, farmer, electromyography, nerve conduction velocity***

## INTRODUCTION

Footdrop (weakness of the foot extensor muscles) can be caused by several pathologies at various levels of the central and peripheral nervous systems, among which lesions of the spinal cord or roots, sciatic nerve, common and deep peroneal nerves are more important. Common peroneal nerve descends first along the lateral side of the popliteal fossa, and finally winds around the back and outer side of fibular neck, where it divides into the superficial and deep peroneal nerves, which innervate foot dorsiflexsors and evertor muscles (1). In 20 to 28 percent of individuals, the lateral part of the extensor digitorum brevis is supplied by an accessory deep peroneal nerve arising from superficial peroneal nerve (2-4). Although the peroneal nerve may be damaged by infarct, tumor or leprosy, but the vast majority of lesions are traumatic (5-7). Postoperative palsies occur specially after operations performed in the lateral decubitus position or when the outer aspect of the upper leg rests against the leg strap or metal brace. The nerve may also be stretched by flexion of the hip and knee while the patient is in the lithotomy position and is specially liable to injury after total knee arthroplasty (8). Compressive lesions are usually unilateral, but bilateral (often asymmetric) ones may develop in patients who have just lost considerable weight, are emaciated or bedridden, or have sustained nerve infarct secondary to vasculitis (9). Chronic squatting and protracted sitting in the cross –legged position or during Yoga are other causes of compression (10,11). During natural childbirth, bilateral peroneal palsy may occur due to prolonged pressure exerted by the patient's palm drawing over the upper lateral aspect of the shins while strongly drawing the knee toward her (pushing palsy) (12). Footdrop, caused by peroneal neuropathy may also occur during weight reduction (Slimmer's palsy) (13). With lesions at the fibular head, specially in farmers the deep branch of the peroneal nerve is affected more commonly than the whole nerve (14). When both deep and superficial branches are involved there is paralysis of the foot eversion and dorsiflexion of the toes and foot. A variable sensory loss affects the entire dorsum of the foot, toes and the lateral distal portion of the lower leg. Lesion of the deep peroneal nerve (anterior tibial or deep peroneal syndrome) results in paralysis of toes and foot dorsiflextion and sensory loss between the first and second toes (15). Superficial peroneal syndrome may be seen in isolation, causing paresis and atrophy of the peronei muscles and sensory disturbance affecting the skin of the lateral distal portion of the lower leg and dorsum of the foot, sparing between the first and second toes. Purely sensory syndrome accompanied occasionally by tenderness at the point of fascial perforation (16,17)

**Correspondence:**
M. Ghaffarpour, Department of Neurology, Imam Khomeini Hospital, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran
Tel: +98 21 9192634, 6940033
Fax: +98 21 6940033
E-mail: ghaffarpour@yahoo.com

and sural nerve injury owing to fractures, Achilles tendon reconstructive surgery and stretching by sprains has also been reported (18-21).

## MATERIALS AND METHODS

Seventeen patients have been evaluted for footdrop that was apparently related temporally to certain farming activities. All of the patients except one were males. Causes other than peroneal injury at the fibular head ruled out by clinical and electrophysiological exclusion criteria (complete weakness of foot inversion, weakness of knee flexion, weakness of glutei, reduced ankle tendon reflex, sensory loss in sole or lateral knee and posterior thigh, hip or thigh and back pain -abnormal finding in tibialis posterior muscle, abnormal finding in flexor digitorum longus, abnormal finding in biceps femoris, abnormal finding in gluteus and tensor fascia latae, abnormal finding in paraspinal muscles, low tibial compound muscle action potential, abnormal H reflex, abnormal sural sensory nerve action potential respectively). The patients were between 15 to 25 years (mean 20.6). They had been harvesting (15 cases) and weeding (2 cases) 1 to 5 days before suffering footdrop, and came to our office for gait difficulty .

**Electrophysiological study:**

A. Motor nerve conducion velocity (Fig. 1).

1- Stimulation was done in three regions:

(a) Ankle: Dorsal aspect of the distal lower leg between the tendones of the tibialis anterior (medially) and the extensor hallucis (laterally) 9 cm to the active recording electrode

(b) Below fibular head: 3 to 4 cm distal to the proximal tip of the fibular head

(c) Popliteal fossa: medial to the biceps femoris tendon,10 cm below the fibular head

2- Recording from G1 (over the extensor digitorum brevis: EDB) and G2 (over the fifth toe)

3- Ground electrode (GND) placed on external malleolus

Existence of accessory deep proneal nerve (27), that arises from the superficial peroneal nerve at the knee, was recognized by having a higher amplitude response on proximal stimulation. This nerve may be stimulated behind the lateral malleolus.

B. Sensory nerve conduction velocity (Fig. 2).

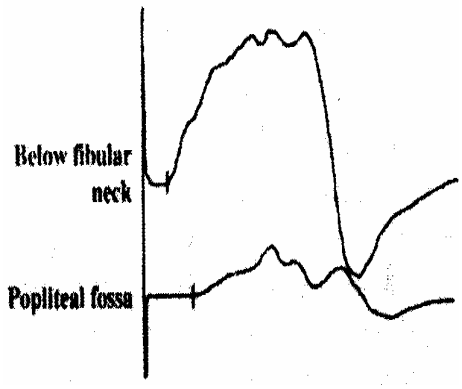
Sensory nerve conduction velocity was evaluated in superficial peroneal nerve, which receives its sensory fibers mainly from the L5 root.

1- Stimulation site is in the lateral calf, 12 cm proximal to G1.

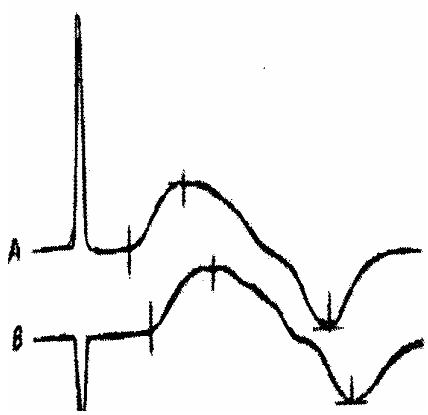
2- Recording electrodes: G1 placed between the tibialis anterior tendon and lateral malleolus, and G2 placed 3-4 cm distally. In addition to the peroneal motor and sensory, we had studied also the tibial motor, F wave and sural sensory potentials to rule out other lesions causing footdrop including sciatic nerve, lumbosacral plexopathy and root lesions.

## RESULTS

Clinical and electrophysiological findings are illustrated in table 3. The patients were between 15-25 years (mean 20.6), 15 cases harvesting and 2 weeding.

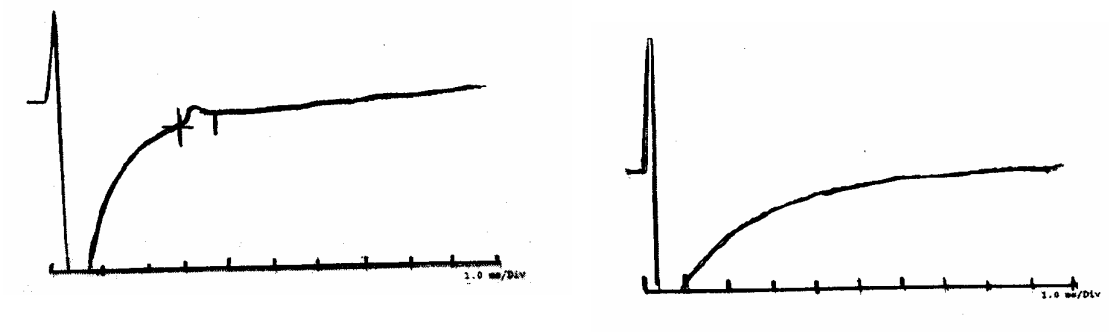



Recording the tibialis anterior in peroneal neuropathy. A: Stimulation before fibular neck. B: Stimulation at popliteal fossa




Normal peroneal motor study. A: Distal stimulation site below the fibular head, recording the tibialis anterior .B: Proximal stimulation site in the lateral popliteal fossa

**Fig 1.** Peroneal motor conduction



A: Normal sensory peroneal nerve study B: Abnormal sensory peroneal nerve study

**Fig. 2.** Peroneal sensory study

**Table 1.** electrophysiological findings

| **Patient number** | **1** | **2** | **3** | **4** | **5** | **6** | **7** |
|---|---|---|---|---|---|---|---|
| Age (years) | 5 | 17 | 19 | 21 | 22 | 24 | 24 |
| Sex | M | M | M | M | M | M | M |
| Handedness | R | R | R | R | L | R | R |
| Type of work, harvesting (h) or weeding (w) | h | h | h | w | h | w | h |
| NVC of the peroneal nerve in the fibular head (m/s), number in parenthesis shows velocity in contralateral side | 20.1 (53.8) | 24.9 (52.9) | 25.9 (49.7) | 17.8 (50.8) | 23.8 (52.8) | 22.7 (49.2) | 21.6 (51.3) |
| Peak to peak motor amplitude (mv), number in parenthesis shows the findings in the other side | 2.1 (10.9) | 3.8 (12) | 5.2 (8.6) | 4.5 (9.7) | 1.9 (12.8) | 3.9 (9.6) | 4.2 (9.5) |
| Conduction block at the fibular head | + | - | - | + | - | - | + |
| Increases in F-wave latency in comparison to the normal side (m/s) | 5 | 6 | 3 | 5 | 7 | 3 | 4 |
| Peroneal sensory study: conduction velocity | 35 (50) | 27.5 (47.2) | 22.8 (51) | 15.6 (46.2) | 31.7 (49.1) | 25.8 (53.2) | 24.9 (42) |
| amplitude (micro volt) | 3.2 (7.8) | 3.1 (9) | 2.8 (7) | 1.9 (11.1) | 4.2 (8.9) | 3 (10.3) | 2.1 (7.5) |
| Time to recovery (months) | 3 | 6 | Not returned | 7 | 7 | 5 | 6 |

Neurological examination revealed unilateral footdrop (16 right-handed had right and 1 left handed had left footdrop) with variable sensory deficit confined to territory of the deep and superficial peroneal nerves, lateral sural cutaneous nerve of the knee or combination of them. Ten patients (mean age 20.9) refused electrophysiological studies. They have been given vitamins and physical therapy. In other 7 patients (mean age 20.2, all but one right-handed males), electrophysiological studies have been performed. In nerve conduction studies, the patients had reduced motor nerve conduction velocity (NCV) of peroneal nerve at fibular head in the affected side (mean 22.4 m/s versus 51.5 m/s in the normal side).Amplitudes had also been reduced at the affected side (mean peak to peak amplitude 3.6 mv in the symptomatic side, 10.4 mv in the contralateral side). Nerve conduction velocity and amplitude distal to the lesion were normal. F-wave latency increased in the affected side in comparison to the normal side (mean 4.7 m/s) (Table 3). Electromyography revealed only reduced interference pattern in the muscles innervated by peroneal nerve below the knee which is a nonspecific finding. Thirteen of them (6 from the neurophysiologically studied cases) recovered completely in the later visits (between 3 and 8 months, mean 6.5 months). The remaining 4 patients were lost to follow-up.

# DISCUSSION

Footdrop has many causes. Various pathologic lesions at different anatomic levels in nervous system can lead to it, but for this present discussion we confine ourselves to the peripheral nerve ethiologies. The sciatic nerve (L4,5-S1,2,3), the largest human's

nerve in diameter is the main branch of sacral plexus (22). It passes out of the pelvis through the greater sciatic foramen, below the piriformis, descends between the greater trochanter of the femur and the tuberosity of the ischium and along the back of the thigh to about its lower one-third, where it divides into two large branches, named the tibial and common peroneal nerves. Although these two branches separate in the distal of the thigh, but from the beginning of the sciatic nerve, they are anatomically distinct. In most lesions of the sciatic nerve, the peroneal division is more severely involved. In Sunderland's opinion this fact is due to presence of larger but fewer funiculi in peroneal devision. In addition the peroneal nerve is fixed at the fibular head, which leads to more mechanical stretching (23). In the posterior aspect of the thigh, the tibial component of the sciatic trunk gives off a series of short branches to the hamstring (long head of the biceps femoris, semitendinosus, and semimembra-nosus) muscles. The peroneal component supplies the short head of the biceps femoris. The tibial nerve (L4,5-S1,2,3), the largest branch of the sciatic nerve, supplies all of the calf muscles and small muscles of the foot. Immediately after its origin, the common peroneal nerve (L4,5-S1,2) becomes superficial as it winds laterally around the head of the fibula. After entering the leg it gives off a small recurrent nerve that supplies sensation of the patella and then bifurcates into the superficial and deep peroneal nerves. The superficial peroneal nerve supplies the peroneus longus and the peroneus brevis, which allow eversion of the foot. The nerve descends between the peroneal muscles, then divides into medial and intermediate dorsal cutaneous nerves. These sensory branches pass anterior to the extensor retinaculum to supply the anterolateral aspect of the lower half of the leg and dorsum of the foot and toes. The deep peroneal nerve innervates the muscles that allow dorsiflexion of the foot (24). Foot dorsiflexion is mainly due to tibialis anterior weakness which can lead to footdrop (25). This muscle is also a foot invertor. Roots of L4,5-S1, but predominantly L5 innervates it. Peripheral nerve lesions that lead to footdrop consist of L5 radiculopathy (the inversion of the foot is also impaired because of the tibialis posterior involvement), sciatic and deep peroneal nerves lesions. In the sciatic nerve lesions, there are evidence of damages to the tibial innervated muscles, both clinically (such as weak plantar flexion of the foot) and electrophysiologically. Denervation in the short head of biceps can also be detected by electromyography. Causes include trauma such as hip, pelvic and femoral shaft fractures, injections in the gluteal region and other uncommon ethiologies such as vaginal operations, difficult delivery, traumatic hematoma in the posterior thigh, tumors, use of bicycle and compression from an arterial aneurysm (23). Sitting for a long period with legs flexed and abducted (lotus position) under the influence of narcotics or barbiturates or lying flat on a hard surface, ischemic necrosis of the sciatic nerve in diabetes melitus or polyarteritis nodosa and Idiopathic forms were also described (26). Peroneal nerve may be involved by pressure during an operation, sleep or from tight plaster casts, obstetric stirrups, habitual and prolonged crossing of the legs while seated (especially in patients who lost a great deal of weight), tight knee boots,diabetic neuropathy, fractures of the upper end of the fibula or tibia, Baker's cyst, muscle swelling or small hematomas behind the knee in the asthenic athletes (26), repeated squatting, minor athletic injury, dislocation of the knee, thrombosis or embolism of the femoral or popliteal arteries, pressure from the ganglion cysts or hematoma in hemophiliac and lipomatosis in the popliteal fossa, leprosy and fabella (sesamoid bones in the gastrocnemius) (23). Patients described in this article are examples of peroneal nerve injury at the fibular head. As the site of injury correlates with the dominant hand, it can be deduced that the particular action of the patient (harvesting and weeding) was responsible for injury and not the sitting or squatting. This may be due to a repeated trauma by the hand of the patient to the peroneal nerve at the fibular head or it may be due to particular sitting position that the patient assumes. As a matter of fact, during harvesting or weeding a farmer squates, bends the dominant knee completely, but the other knee partially, and then doing repeated to and for movements of the dominant hand to cut the plants or weeds. Whether the complete flexion of the knee or the repeated trauma to the head of fibula by the forearm, injures the peroneal nerve is not fully understood. The relative young age of the patients is noteworthy. Young workers may not be as experienced as the older farmers in their tasks or perhaps they work more vigorously. Also the relatively good prognosis of footdrop in these patients is important. However as one can not do a second electrophysiologic study the possibility of residual subclinical sequels remains open.

# REFERENCES

1. Brazis W, Masdeu C, Biller J. Localization in clinical neurology, third ed, Little-Brown company, New York 1999; PP: 35-44.

2. Dessi F, Durand G, Hoffmann J. The accessory deep peroneal nerve: A pitfall for the electromyography. J Neurol psychiatry 1999; 55: 214.

3. Gutmann L. AAEM minimogograph# 2: Important anomalous innervation of the extremities. Muscle Nerve 1993; 16: 339.

4. Stamboulis E. Accessory deep peroneal nerve. Electromyogr Clin Neurophysiol 1987; 27: 289.

5. Jones HR, Felice KJ, Gross PT. Pediatric peroneal mononeuropathy: A clinical and electro-myographic study. Muscle Nerve 1993; 16: 1167.

6. Katirji MB, Wilbourn AJ. Common peroneal monomeuropathy: A Clinical and electrophysiologic study of 116 lesions. Neurology 1988; 38: 1723.

7. Wertsch JJ, Sanger JR, Matloub HJ. Pseudterior interosseous syndrome. Muscle Nerve 1985; 8: 68.

8. Rose HA et al. Peroneal nerve palsy following total knee arthroplasty. A review of the hospital for special surgery experience. J Bone Joint surg 1982; 644: 547.

9. Wlbourn AJ. AAEE case report: 12 common peroneal mononeuropathy at the fibular head. Muscle Nerve 1986; 9: 825.

10. Yoga CJ. Foot drop. JAMA 1971; 217: 828.

11. Marwah V. Compression of the lateral popliteal (common peroneal) nerve. Lancet (Dec 26) 1964; 1367.

12. Andornato BT, Carlini WG. Pushing palsy, a case of the self – induced bilateral peroneal palsy during natural childbirth. Neurology 1992; 42: 936.

13. Smith BE, Litchy WJS. Sural mononeuropathy; A clinical and electrophysiological study. Neurology 1989; 39: 296.

14. Sotaniemi KA. Slimmer's paralysis-Peroneal neuropathy during weight reduction. J Neural Neurosurg Psychiatry 1984; 47: 564.

15. Esselman PC et al. Selective deep peroneal nerve injury associated with arthroscopic knee surgery. Muscle Nerve 1993; 16: 1188.

16. Banerjee T, Konns DD. Superficial peroneal nerve entrapment. J Neurosurg 1981; 55: 991.

17. Sridhara CR, Izzo KL. Terminal sensory branches of the superficial peroneal nerve: An entrapment syndrome. Arch Phys Med Rehab 1995; 66: 789.

18. Gould N, Trevino S. Sural nerve entrapment by avulsion fracture of the base of the fifth metatarsal bone. Foot Ankle 1981; 2: 153.

19. Gross JA, Hamilton WJ, Swift TR. Isolated mechanical lesions of the sural nerve. Muscle Nerve 1980; 3: 248.

20. Pringle RM, Protheroe K, Mukherjee SK, Entrapment neuropathy of the sural nerve. J Bone Joint Surg 1974; 56B: 465.

21. Reisin R et al. Sural neuropathy due to external pressure, Report of three cases. Neurology 1994; 44: 2408.

22. Williams PL, Warwick R. Gray's anatomy, 36th edition, Churchill Livingstone, London, 1980; PP: 1112-1115.

23. Liveson JA. Peripheral neurology case studies in electrodiagnosis. Second edition, Philadelphia, F. A. Davis Company 1991; PP: 48-55.

24. Kimura J. Electrodiagnosis in diseases of nerve and muscle. Second edition, FA. Davis Philadelphia 1989; PP: 21-22.

25. Daniels L, Worthingham C. Muscle testing techniques of manual examination, 5th edition, W.B.Saunders company Philadelphia 1968; P: 76.

26. Victor M, Ropper AH. Adams and Victor's Principles of neurology. 7th edition, Mc Graw- Hill Medical publishing division, New York 2001; P: 1437-1438.

27. Sethi RK, Thompson LL. The electromyographer's handbook. Second edition, Little Brown and Company, New York 1989; PP: 67-74.

28. Preston David C, Shapiro Barbara E. Electromyography and neuromuscular disorders, first edition, Butterworth Heinemann, Boston 1998; PP: 307-314.