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### Introduction

Clavicle fractures are common, yet associated supraclavicular nerve (SCN) injuries are often overlooked. Given its superficial course, the SCN is highly vulnerable to direct impact or fracture displacement. Localized SCN injuries are often misattributed to the fracture, postoperative somatic pain, or broader brachial plexopathy, causing diagnostic delays. Despite persistent neuropathic pain, SCN injuries are rarely referred for specialized electrodiagnostic evaluation. We present a case of chronic, intractable SCN injury in a 48 years old male, emphasizing that while SCN lesions present distinct clinical patterns, they often remain unrecognized in conventional settings.

### Case report

A 48 years old male sustained a right clavicle fracture after a 1.3 m fall and underwent open reduction and internal fixation. Following successful bone union, the fixation plate was removed. However, the patient reported persistent right chest and shoulder pain. He received conservative treatment at a local clinic for two years without electrodiagnostic evaluation or clinical improvement, prompting referral to our department. Physical examination revealed tenderness in the right anterior chest and lateral deltoid regions. Upper extremity motor strength was preserved, and deep tendon reflexes were normoactive. MRI of the right brachial plexus showed no abnormalities. Electrodiagnostic studies were performed to investigate the pain. Sensory nerve conduction studies (NCS) of the supraclavicular nerve were performed in accordance with the standardized protocol described by Lee and DeLisa (Fig. 1). The

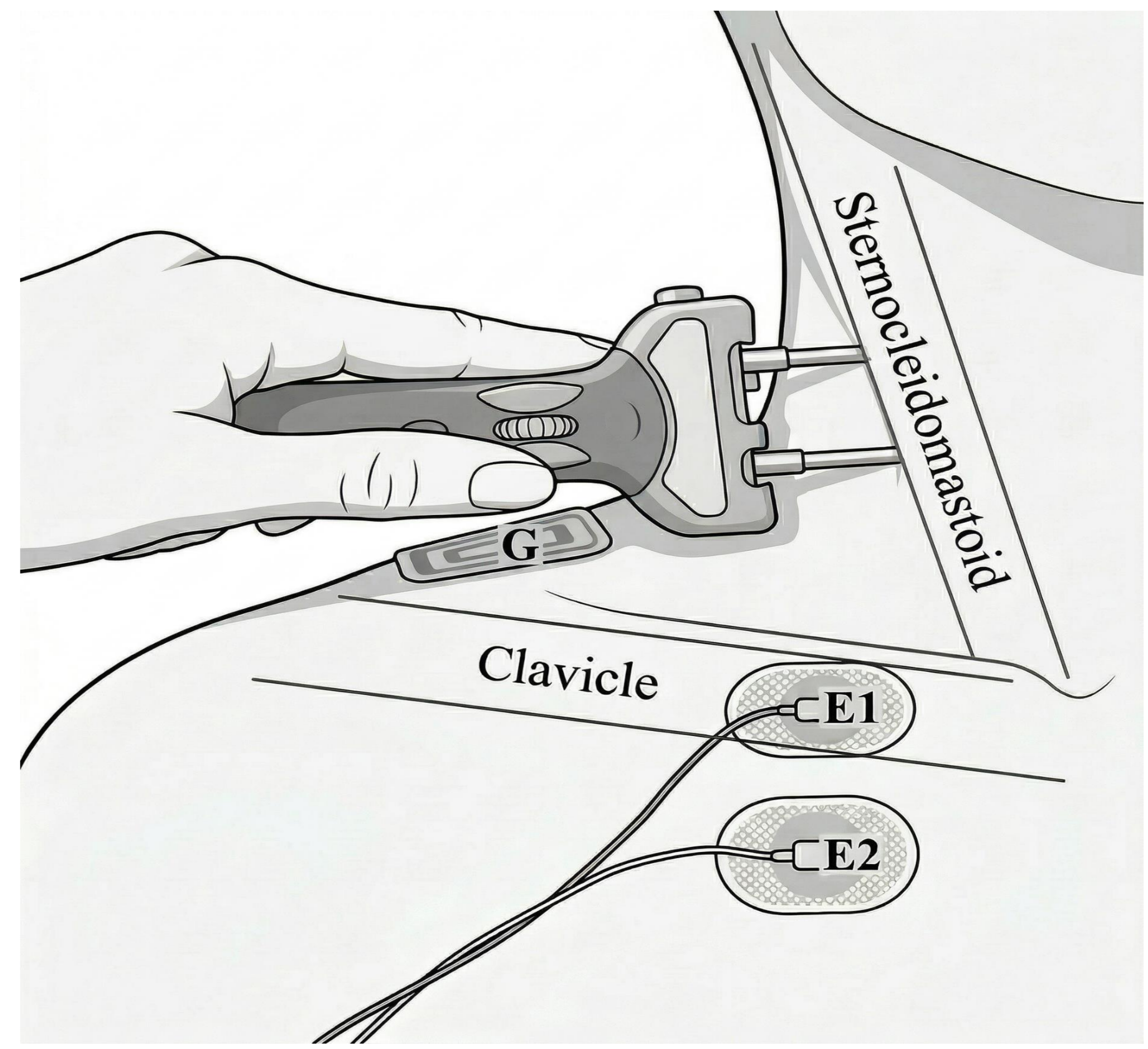


Figure 1. Supraclavicular sensory nerve conduction study

The active recording electrode was placed over the outer surface of the clavicular shaft and the reference electrode was placed approximately 3 cm distal to the active electrode. Stimulation was delivered using a cathode positioned at the posterior border of the sternocleidomastoid muscle at the level of the lower margin of the thyroid cartilage. The NCS revealed a significantly reduced sensory nerve action potential amplitude of the right SCN (9.4  $\mu$ V) compared to the unaffected left side (43.8  $\mu$ V), suggesting an axonal injury (Table 1). To differentiate this from a cervical lesion or other radiculopathies, needle electromyography (EMG) was performed on the right upper extremity and paraspinal muscles. The EMG findings were entirely normal in all tested muscles, including the C4/5 paraspinals, with no evidence of abnormal spontaneous activity or motor unit action potential changes. These comprehensive findings confirmed an isolated right SCN injury, effectively distinguishing it from cervical radiculopathy or brachial plexopathy.

Table 1. Nerve conduction study in both upper extremities

Nerve	Latency (ms)		Amplitude ( $\mu$ V)	
	Right	Left	Right	Left
Lateral antebrachial cutaneous	2.4	2.2	19.9	35.8
Medial antebrachial cutaneous	2.3	2.1	14.5	14.9
Median	3.5	3.5	34.5	41.1
Superficial radial	2.6	2.5	33.6	34.6
Ulnar	3.2	3.3	17.1	31.5
Supraclavicular	1.5	1.2	9.4	43.8

Nerve	Latency (ms)		Amplitude ( $\mu$ V)	
	Right	Left	Right	Left
Axillary	4.5	4.0	8.8	8.2
Median	4.1	4.0	7.1	8.0
Musculocutaneous	4.1	3.9	8.7	7.9
Radial	2.7	2.3	5.8	6.4
Ulnar	2.9	2.7	10.7	8.7

### Conclusion

This case highlights the SCN as a potential, yet frequently ignored, source of refractory peri-clavicular pain. SCN injuries are often misdiagnosed or overlooked because the nerve is small and excluded from standard electrodiagnostic protocols. However, this case demonstrates that with a history of clavicle trauma and a typical neuropathic pain distribution stretching from the anterior thorax to the scapular spine a targeted SCN-NCS provides a definitive diagnosis. Raising clinical awareness of SCN injury patterns and utilizing specialized NCS is essential to ensure timely diagnosis and appropriate neuropathic pain management.

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