The Goosebump Sign

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Abstract

Keywords

- peripheral nerve injury
- nerve palsy
- sympathetic dysfunction
- autonomic dysfunction
- clinical sign

Nerve injury may occur following shoulder trauma. However, joint or bony damage can often dominate the clinical picture such that signs of nerve injury are overlooked. We describe the “goosebump sign” as a hitherto undescribed objective clinical finding of sympathetic dysfunction in peripheral nerve palsy, which can be a useful complementary sign to the standard assessment of muscle power and sensibility.

Introduction

The axillary nerve is vulnerable to injury following shoulder trauma and surgery because of its close relationship with the glenohumeral joint and its course around the neck of humerus. Prospective electrophysiologic studies in patients with anterior dislocations of the shoulder after low-velocity trauma have shown nerve lesions to be present in up to 19 to 55%.¹ Joint or bony injury can often dominate the clinical picture such that the signs of associated nerve injury are overlooked.² Therefore, in addition to the standard assessment of muscle power and sensibility, clinicians need to be vigilant to the presence of subtler clinical signs of nerve injury.

Sympathetic unmyelinated fibers in peripheral nerves are among the most resistant to mechanical trauma. Damage to these fibers suggests a more severe injury, and the disturbance of autonomic function is an important early clinical finding.³ We describe the “goosebump sign” as a hitherto undescribed objective clinical finding of sympathetic dysfunction in axillary nerve injury.

Case Report

A 54-year-old man was referred with axillary nerve palsy 9 months after undergoing shoulder hemiarthroplasty following a fracture dislocation. Clinical examination demonstrated no muscle activity in the deltoid and numbness in the “regimental badge” area. In addition, it was noted that

although the hair follicles were erect in the rest of the arm, there was a noticeable lack of pilomotor activity in the region that corresponded to the area of sensory deficit (►Fig. 1). This “goosebump sign” could be elicited by exposure to a cold stimulus or by lightly stroking the skin of the area concerned.

Fig. 1 Clinical photograph of the right shoulder demonstrating the goosebump sign—where there is an obvious absence of “goosebumps” in the regimental badge area compared with the surrounding skin.

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Electrophysiologic testing confirmed a severe axillary nerve lesion. The patient underwent exploratory surgery that revealed a nonconductive neuroma-in-continuity and received a radial-to-axillary nerve transfer.

Discussion

A peripheral nerve is not a homogeneous structure. The most heavily myelinated fibers are the large motor neurons (type A α), followed by afferent muscle spindles (type A β). Nerve conduction velocities in these neurons are approximately 30 to 120 m/s. Unmyelinated neurons (type C), such as the sensory neurons involved in transmitting pain and temperature, and postganglionic sympathetic fibers are the slowest, conducting at approximately 1 to 2 m/s.⁴ Sympathetic innervation of the arm is provided by nerve fibers that leave the spinal cord at T2–4 segments. They traverse the stellate ganglion to join the somatic branches of the brachial plexus before reaching their final destination carried by the individual peripheral nerves.⁵ They are responsible for the sudomotor, vasomotor, and pilomotor activities of the skin, which can be affected when there is injury to the accompanying nerve.⁶

In the assessment of potential nerve injury, the clinician relies on a battery of clinical signs that can be easily and quickly ascertained. Objective sensibility examinations such as the Semmes-Weinstein's monofilament test and two-point discrimination can be difficult to perform in the acute trauma setting.⁷

Several clinical tests for autonomic function have been described for the assessment of peripheral nerve injuries, which rely directly or indirectly on the presence of sudomotor activity, that is, sweating. The "ballpoint pen" test relies on the loss of tactile adherence in an anhydrotic area when a pen is lightly drawn across the skin.⁸ Although useful when assessing the digit, it has limited value in the examination of axillary nerve palsies. The "dye test" involves coating the skin with a dye, and the patient is made to sweat either with the aid of heat or with different drugs.⁹ When sweating sets in, the areas with normal sudomotor function would become colored while the abnormal area remains unchanged. This test, however, is impractical and can cause considerable discomfort. O'Riain described the "wrinkle test" in which the arm is immersed in warm water for 30 minutes and the area where there is lack of skin wrinkling corresponds to the territory of peripheral nerve injury.¹⁰ This is because with the loss of sweating following nerve injury, there is reduction in the salt content of the skin, which then leads to reduced capacity of the skin keratin to bind water.⁵ Although useful

in the hand, this test is also impractical in the assessment of axillary nerve injuries.

The "goosebump sign" reflects the loss of pilomotor activity of the denervated skin. This sign is easy to observe in the upper arm, where there is an abundance of hair follicles compared with the hand. It may be useful in the comatose, young, or otherwise uncooperative patient, and offers the clinician an early clue to the presence of a peripheral nerve injury.

Although appearance of a "goose-flesh" phenomenon is well known in the presentation of autonomic dysreflexia due to abnormal activation of the sympathetic nervous system,¹¹ the "goosebump sign" is caused by the loss of sympathetic activity following injury to a peripheral nerve. This is previously undescribed for the assessment of axillary nerve injury and is a useful complementary finding in addition to the standard examinations.

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Conflict of Interest

None.

References

- 1 Visser CP, Coene LN, Brand R, Tavy DL. The incidence of nerve injury in anterior dislocation of the shoulder and its influence on functional recovery. A prospective clinical and EMG study. *J Bone Joint Surg Br* 1999;81(4):679–685
- 2 de Laat EA, Visser CP, Coene LN, Pahlplatz PV, Tavy DL. Nerve lesions in primary shoulder dislocations and humeral neck fractures. A prospective clinical and EMG study. *J Bone Joint Surg Br* 1994;76(3):381–383
- 3 Seddon HJ. Three types of nerve injury. *Brain* 1943;66(4):237–288
- 4 Waxman SG. Determinants of conduction velocity in myelinated nerve fibers. *Muscle Nerve* 1980;3(2):141–150
- 5 Braham J, Sadeh M, Sarova-Pinhas I. Skin wrinkling on immersion of hands: a test of sympathetic function. *Arch Neurol* 1979;36(2):113–114
- 6 Gibbons CH, Wang N, Freeman R. Capsaicin induces degeneration of cutaneous autonomic nerve fibers. *Ann Neurol* 2010;68(6):888–898
- 7 Strauch B, Lang A, Ferder M, Keyes-Ford M, Freeman K, Newstein D. The ten test. *Plast Reconstr Surg* 1997;99(4):1074–1078
- 8 Harrison SH. The tactile adherence test estimating loss of sensation after nerve injury. *Hand* 1974;6(2):148–149
- 9 Moberg E. Objective methods for determining the functional value of sensibility in the hand. *J Bone Joint Surg Br* 1958;40-B(3):454–476
- 10 O'Riain S. New and simple test of nerve function in hand. *BMJ* 1973;3(5881):615–616
- 11 Karlsson AK. Autonomic dysreflexia. *Spinal Cord* 1999;37(6):383–391