Letters to the Editor

Work-related upper limb disorders: Can prevention and management be improved?

Sir,

Work-related upper limb disorders remain a challenge to the clinician because an estimated 75% are regarded as diagnostically unclassifiable.[1] Evidence-based prevention and treatment of these frequent and disabling disorders have therefore been limited and largely unsuccessful. It is essential to identify the involved tissues and structures as well as the responsible pathologies. To do so would require improved diagnostic approaches.

Upper limb pain appears frequently as neuropathic and may be accompanied by weakness/heaviness, tactile dysfunction and/or sensory abnormalities, e.g. paraesthesia. Although this combination of symptoms suggests a peripheral nerve-involvement, clinicians and researchers tend to attribute most pathology to muscles and tendons/insertions. Even when peripheral nerve-involvement is alleged, the focus is mostly restricted to carpal tunnel syndrome and cervical radiculopathy. The intermediate nerve receives less attention.

Rather specific neurological patterns follow focal neuropathies: Weakness in muscles innervated distally to the lesion, altered sensibility in supplied cutaneous territories and abnormal nerve trunk soreness. All physicians have been trained in an examination based on these principles. Still, a thorough neurological examination is rarely applied – in particular with respect to the more proximal portions of the upper limb nerves. It may be regarded as difficult and time consuming, and the validity may be questioned if peripheral neuropathy is not proved by electrophysiology. Although electrophysiological studies are viewed as “golden standard” for peripheral neuropathy, a mixed and partial nerve affection with few myelinated fibers intact and re-innervation taking place may result in entirely normal findings.[2]

A feasible physical examination should target the nerves from the roots to the muscular and cutaneous supply by including neurological items representative to neuropathies with various locations. It should be reproducible and preferably identify and exclude abnormalities in symptomatic and healthy subjects, respectively.

Our team has developed a detailed but still rapid semi-quantitative upper limb neurological examination comprising an assessment of the strength in selected individual muscles,[3], of sensory deviations from normal in homonymously innervated territories, and of the presence of mechanical nerve trunk allodynia.[4] Patterns of findings in accordance with the topography of the nerves and their muscular and sensory innervation were frequent – and also identified in patients that could not be diagnosed by conventional means. The patterns were reliably identified[4] and related to symptoms.[5] The infraclavicular brachial plexus was the dominant location and often combined with median and radial nerve-involvement at elbow level. Whether diagnosed by conventional diagnostic criteria or criteria developed by the authors, neuropathic upper limb disorders were also common among patients in general practice.

This low-tech examination demands no equipment beside a needle and a 256 Hz tuning fork. The manual assessment of individual muscle strength is easily learned by any physician.[3] The manual character of the examination indicates its feasibility by medical practitioners in any setting in industrialized countries as well as in the developing world.

Therefore, the developed and validated diagnostic approach may eventually constitute a step towards improved prevention and treatment of work-related upper limb disorders.

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Letters to the Editor

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Transient anisocoria: A pesky palpitation

Sir,

Anisocoria in an emergency room invariably indicates an impending life-threatening neurosurgical emergency. It may be the first and only sign of third nerve dysfunction from expanding aneurysm, or intracranial bleeding, cerebral neoplasm/compression, meningeal infiltration or tentorial herniation. Determining the cause of anisocoria is challenging in a critically ill patient, since the patient is often sedated, paralyzed or has an altered mental status. As clinical neurological examination is limited, the situation warrants advanced neuroradiologic investigations. Here, we report a case of anisocoria due to unintentional ocular exposure to nebulized ipratropium bromide.

A 65-year-old male with a history of severe head injury and a Glasgow Coma Score (GCS) of 7/10, without demonstrable changes in the initial non-contrast cerebral computer tomography (CT), was referred to our center on the second day in anticipating a neurosurgical intervention as he had dilated (7 mm), non-reacting left pupil without change in his GCS. Neurologic examination in our emergency department was essentially nonfocal with a fixed and dilated left pupil [Figure 1]. Urgent CT brain at that time did not reveal any acute or new changes. Further review disclosed that he received nebulized ipratropium bromide via a faulty nebulizer set leaking predominantly toward the left side of the face. One-tenth percent pilocarpine instilled in each eye to test cholinergic supersensitivity resulted in no pupillary constriction. One percent pilocarpine was instilled in each eye and, after 30 minutes, the right pupil constricted from 4 to 2 mm, whereas the left pupil remained dilated at 7 mm which was consistent with pharmacological cause for dilation. The patient was closely followed up. Within the next 8 hours, the anisocoria resolved and the pupil reduced to 2 mm in size and reacted normally to light. There were no subsequent episodes of anisocoria.

In the absence of a structural cause, transient anisocoria in critically ill may be due to contamination of the eye from nebulized ipratropium bromide [1-7] which paralyzes parasympathetic nerve endings as reported in the literature, [8] but is still not immediately recognized. Minimal pupillary asymmetry following ocular exposure to nebulized ipratropium in clinical practice is often not noticed or considered in practice or discussed much in teaching-training. In our patient, the repeated nebulization may have resulted in an exaggerated mydriatic effect as an animal study [9] suggests that following the use of anti-cholinergic agents, the β-receptor in the eye gets stimulated and can potentiate the mydriatic effect of beta-adrenergic agents by about twofold. Failure of the dilated pupil to constrict after...