EDITORIAL

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Is a detailed neurologic physical examination always necessary?

THE ARTICLE IN THIS ISSUE by Shikino et al[1] on a mimic of Bell palsy gives us an opportunity to discuss the question posed by the title of this editorial. The obvious short answer is "no."

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Any experienced clinician will acknowledge that the extent of the physical examination and the extent of information obtained during the history should be determined by the problem being evaluated at the time and by the setting in which it takes place. The difficulty, of course, is that this relies on the judgment of the clinician, and this may or may not pass the test of hindsight.

Every bit of information you obtain from the history or physical exam that you do not record disappears with you and is irretrievably lost

Verghese et al[2] have eloquently emphasized the hazards of an incomplete or inadequate physical examination. Their study was not designed to determine the prevalence of deficient physical examination, either in its extent or its accuracy. Their purpose was to promote the necessity of proper teaching and performance of examination technique.

The neurologic examination is one of the last bastions of physical assessment.[3] Despite remarkable advances in imaging and physiologic techniques, the neurologic physical assessment remains critical for diagnosis and management of the neurologic patient. One of my mentors in neurology used to urge residents to examine patients and record the results of the examination as if every patient would subsequently be the subject of a clinicopathologic conference. Anyone who has reviewed a case for a conference or a case report can identify with that sentiment, wishing that some missing piece of information were available. Yet everyone also recognizes the difficulties, if not the impossibility, of achieving that ideal result.

But recording information obtained during the history or physical examination is important even in the course of a daily routine evaluation. I find myself wishing that a previous examiner had commented on whether the muscle stretch reflexes were somewhat hypoactive (eg, "1+") or on the brisk side ("3+") rather than "physiologic." Was the right leg actually globally weak ("4/5"), or was there a discrepancy between proximal and distal muscles or between the physiologic flexors and the extensors?

This can make a big difference in following a patient's neurologic progress, even over a short time span. It might tell us whether we are dealing with weakness from a peripheral neuromuscular disorder (eg, Guillain-Barré syndrome) or from a myelopathy due to impending spinal cord compression.

It should be mentioned that although Guillain-Barré syndrome is characterized as an ascending paralysis, ie, beginning distally and spreading rostrally, it is one of the few peripheral neuropathies that can present with predominant proximal weakness. It is, in fact, a radiculoneuropathy. But spinal cord (upper motor neuron) disorders preferentially weaken the physiologic flexors of the lower limbs (hamstrings and ankle dorsiflexors), leading to the characteristic extensor posture of the spastic leg. Other findings that can help differential peripheral vs spinal cord disorders include distal sensory loss and hypoactive or absent muscle stretch reflexes in a peripheral neuropathy, compared with dissociated sensory loss (eg, impaired pain and temperature sensation

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in one leg with reduced vibration perception and proprioception in the other) along with hyperreflexia with cord lesions.

Therefore, a careful neurologic examination may tell us whether magnetic resonance imaging of the spine or an electrodiagnostic study should be the next step.

Shikino et al describe a patient who presented with what looked like idiopathic facial palsy (Bell palsy) but turned out to be the result of a primary central nervous system (CNS) cause. Would a more detailed neurologic examination have identified this as a CNS disorder? Would more specific information about the degree and distribution of facial paresis have facilitated earlier recognition of a progressive process, making idiopathic facial palsy less likely? How much elevation of the eyebrow occurred with voluntary activation, how many millimeters of sclera were visible with gentle eyelid closure? How much space remained between the lips on attempted lip closure?

Upper facial muscle weakness is typically not seen in CNS disorders, although facial nerve or nucleus involvement at the pontine level can impair eyelid and frontalis function. Such lesions would usually be accompanied by "neighborhood" signs such as subtle ipsilateral lateral rectus or abducens palsy, involvement of the vestibular nuclei with vertigo, or facial sensory impairment from disruption of the descending trigeminal nucleus and tract. These would be "pertinent negatives" for excluding a brainstem lesion, and ipsilateral motor, sensory, or "higher cortical" functions would obviously signal a supratentorial CNS disorder.

In the case described by Shikino et al, observation and recording of the amount of facial motor function at the initial visit, 3 days after onset, could facilitate recognition of an aberrant course even a few days later and prompt further investigation at an early follow-up visit (idiopathic palsy is almost invariably maximal by 72 hours). I would assume that no additional clinical information was available to the subsequent examiner in this case, 2 months later, rather than suggesting that such information was omitted for the sake of parsimony.

Would any of this have made a difference? Probably not, but we need all the help we can get in medicine. Remember that every bit of information you obtain from your history or physical examination that you do not record disappears with you and is irretrievably lost. ■

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