Cutaneous Pinprick Sensibility as a Screening Device

This method is overlooked, undervalued and critical in redefining a clinically significant threshold for protective sensation.

BY BARRY L. JACOBS, DO

Leg ulceration — a serious complication of diabetic peripheral neuropathy (DPN) — frequently leads to amputation. Several widely recommended testing methods are useful aids to predict DPN. Among these methods, established convention and clinical evidence maintain the employment of large fiber modality testing (eg, pressure/touch and vibration). It might be speculated, however, that closer scrutiny of the relevant neuropathophysiology suggests that adequately executed pinprick may still emerge as the superior choice of testing modality.

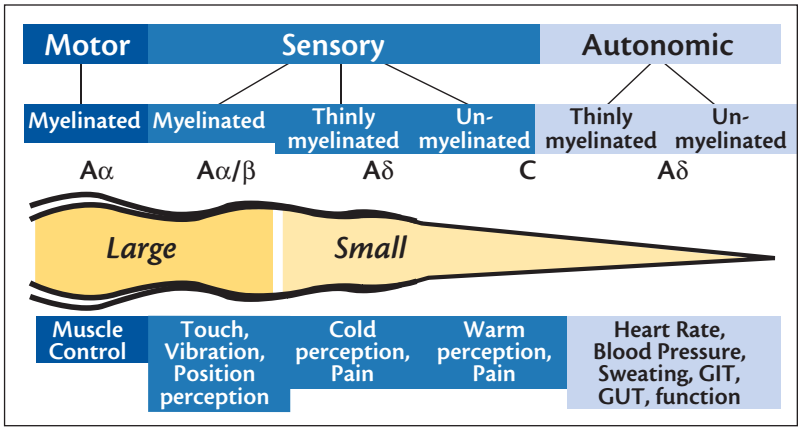

Figure 1. A simplifed view of the peripheral nervous system. (Adapted from Vinik AI. Presented at the APMA 2004 Annual Scientific Meeting, Aug 25, 2004. Available at www.cahe.com/apma2004.webcast_pres.cfm.)

The testing of cutaneous pinprick sensibility is a routine medical procedure. It has applications in family practice, diabetes, neurology, oncology, anesthesiology and in the ER. Particularly, it has ramifications for the prognosis of conditions associated with gross morbidity in which the pathophysiology is dominated by small nerve fiber destruction. The pinprick deficit produced by such small fiber population loss is commonly reported to precede that of larger fiber modalities including pressure/touch. It is hypothesized, where appropriately discernible, that cutaneous pinprick sensibility may reflect the development of clinically critical thresholds of neuropathy not revealed by testing with other modalities.

PAIN AS A PROTECTIVE MECHANISM

It may be cliché — and possibly the more powerful for it — that *The Gift of Pain* by Brand and Yancey is acclaimed as a principal medium for establishing the value of pain to provide a protective mechanism against tissue damage.[1] In context, the Latin derivative, "nocere," offers the translation[2] "to do harm." Widely quoted and almost universally venerated, the text's standpoint on the critical role of nociception as a defining element in the maintenance of health would be difficult to refute and is compatible with the definition offered by the International Group For the Study of Pain, "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."

It is reasonably assertable that the primary role of pain is to caution the central nervous mechanism against somatic insult. This is not a function generally attributed to either pressure/touch or vibration, although that is not to say the

events giving rise to tissue damage cannot overlap with them. As an advanced warning device, however, pressure/touch or vibration are not the primary physiological mechanism. In the peripheral nervous system (Figure 1), withdrawal of an extremity from noxious stimulation (damaging normal tissue) is largely initiated by cutaneous nociceptors. The central nervous system easily habituates to sustained neurological traffic, apart from that of pain. In contrast, sustained exposure to a painful stimulus frequently generates an exponential curve of sensitization.

Pain perception is a mechanism evolved to effect the preservation of normal tissue integrity in which the diminution of performance must attract a concomitant potential for reciprocal endangerment of the host tissue. One frustration for the examining practitioner is that a deficit of pain perception may be a subtle but progressive affair that seems beyond conventional primary methods of assessment. According to Marks,[3] some patients will have loss of normal sensation as a result of nerve damage and hypofunction. These patients will not be aware of disability until injury and/or ulceration occur. This occult behavior gives rise to the assertion that DPN, the most common peripheral neuropathy in advanced nations,[4,5] accounts for more hospitalizations than all other diabetic complications combined. We should appreciate that the potential for risk for damage does not represent (nor require) absolute abolition of pain perception rather than a reduction. Pain sensitivity can be represented on a scale; increments of this scale should roughly equate to a proportional risk of damage. Where some degree of pain has occurred, deficit examination of the affected tissues must be quantitative.

A CLINICALLY SIGNIFICANT THRESHOLD FOR PROTECTIVE SENSATION

Almost without exception, studies employing a pinprick as part of the DPN screening test criteria have imposed a simple presence of perception test using sharpness rather than degree. Variations of this technique have been described, however, the general principle may take for granted that a negative finding is revealed (1) by a failure to discriminate between sharp and blunt or (2) to identify sharp as compared with a predictably normal part of the body (eg, shoulder). This does not accurately represent the role of pinprick or the behavior of typical pathophysiological degeneration.

The following is a common observation: Pain and temperature perception are carried by and target nerve constituents that are damaged principally and initially by metabolic disorders including diabetes.[6-11] It is well understood that these are the small-fiber A delta and small-fiber C-population. The other tactile testing modalities (eg, touch/monofilament and vibration) are carried out only by the large fiber populations, which appear to deteriorate at a later stage.

In tandem with its physiological function of providing protection against tissue damage, there is a strong implication that pain deficit is a consequence of its early occurrence in the development of neuropathic ulceration compared with the modalities carried by larger fibers. The nature of DPN is progressive; the conventional notion of protective sensation may lack sufficient discrimination for refined diagnosis and prognostication. Findings from the 10,000-patient cohort North-West Diabetes Foot Care Study[12] provided significant support for this suggestion, although even in this work, only crude pinprick technique was employed, and focus was biased toward monofilament assessment. An argument emerges for a method that describes when a patient with diabetes has become vulnerable to the effects of pain deficit. This approach requires a refinement of the stages/thresholds at which loss of protective sensation become clinically significant. The refinement would bolster the right of a normal distribution curve where time runs along the x-axis.

LIMITATIONS TO QUANTIFICATION WITH MONOFILAMENT

Testing pressure/touch is contentious; seeking to reveal early pain deficit by focusing on large fiber testing modalities would seem inconsistent with both the physiology and the pathophysiology of the nerve constituent population. Early loss of pressure/touch and the potential testing benefits provided by detection would significantly be preceded by deficit of pain or pinprick. Clinical information (derived from modest diminution rather than complete absence of cutaneous pinprick) would be extensive compared with the degree of loss simultaneously demonstrable in large fibers using pressure/touch.

The attractive aspect of assessment with the Semmes-Weinstein monofilament is based on producing a degree of objective quantification through the application of a reproducible, calibrated stimulus. As is so frequently the case in the minutiae of everyday practice, this method takes for granted a number of assumptions. Aside from concern over the lack of consistency between the available monofilaments (both commercially and offered by various pharmaceutical companies),[13] it may be erroneous to assume that the quantified stimulus of the test will be perceived as universal by the patient population. Tactile sensation is an especially idiosyncratic phenomenon that is a function of — and perpetually influenced by — factors including manifestations of neurological arousal embracing any variant between anxiety and relaxation (though it may also take into account fatigue and ambient temperature). The same patient is likely to provide two or three different responses to the same test

over several occasions simply due to variation in personal circumstance.

Between patients, and, for that matter, different practitioners, the variance will be compounded making the detection of subtle distinctions in sensitivity somewhat arbitrary. Where application of a more extreme stimulus is used (eg, one in which the magnitude gravitates significantly to the right of the normal distribution curve), the practitioner is able to provide sufficiently gross stimulation to eclipse subtle variations in circumstances and be more recognizable as uniform between individuals or for the same individual on different days.

The inability to perceive a 5.07-g monofilament represents a sensory threshold that is more than 50 times greater than normal. This implies that approximately 98% of normal sensory ability has been lost.[14] The usefulness of extreme stimulation for detecting subtle nerve damage, thus, becomes questionable by virtue of the advanced deficit required to fail to perceive it. The 10-g monofilament, therefore, is probably the least suitable instrument to detect early deficit. It is perfectly adequate for consistently demonstrating unequivocal cases. The monofilament is established in the literature as a reliable device for the prediction of neuropathic ulceration. It must be recognized, however, that this predictive value is compatible only with advanced stages of degeneration. One disability score factor that particularly betters monofilament testing is a preexisting history of ulceration itself.[12] So far, the value of the monofilament has been only to signal advanced vulnerability.

PINPRICK AS A DESCRIBER OF CHANGE

The perceived presence/absence of a sharp stimulus as an all or nothing criterion is insufficient to reveal early pain deficit. In contrast, simple recognition of a reduction in pinprick sensibility, even when early, could be critical. A redeeming feature of pinprick testing is not based on objective quantifiability (although quantification is possible with the use of adapted techniques),[15] but the principle that it can demonstrate deficit by comparison of a potentially affected area to one that is expected to display an acceptable degree of integrity elsewhere on the same subject. Findings are based upon the distinctions made by the patient between these areas, thereby dispensing with the need to compare to a predetermined marker.

Extraneous circumstances will be of little consequence; they will be constant for the same patient, on the same day, who is always compared to him or herself. All that is required of the patient is to recognize a difference in acuity of the pinprick stimulus. Visual analogue scales have shown that patients are capable of demonstrating remarkable consistency in identifying subtle distinctions in pain severity.[16] By use of a simple comparison between affected and normal areas of skin, patients are permitted to express even the earliest and most subtle differences freely. Adaptations to technique, such as those routinely employed by neurologists, can also be utilized to further refine this mode of assessment for test sensitivity, accuracy and reproducibility.

As sensitive methods for monitoring progression of DPN examination, procedures that exclude pinprick from the tactile modalities are likely to be inadequate. It is unsurprising that even in the current popular climate, pinprick is regularly recommended as a best practice for screening.[17-22] It is suggested that this method remains largely neglected by the health care provider community. There seems satisfactory evidence to effect a more reflective and potentially effective approach simply by drawing upon the resources of evidence-based physiology and revisiting with a more objective viewpoint some of the existing literature. ■

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