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Perioperative Ulnar Neuropathy:

Are We Ready for Shortcuts?

How do we prevent adverse outcomes? At first glance, the question seems too broad to permit any meaningful answer. But for purposes of understanding an important study that appears in the current issue of ANESTHESIOLOGY,[1] let us provide a basic conceptual framework.

Recognition is the first step in prevention. This is straightforward for outcomes that have a stereotyped presentation, but for those with variable expression or features that mimic other entities, the process of recognition may be difficult.

Once an adverse outcome is recognized, it must be deemed *suitable for preventive efforts.* This second step represents a cost-benefit estimate that requires consideration of the frequency of the adverse outcome, the severity of the associated injury, and the anticipated costs and side effects of preventive efforts. Although precise numeric values may be difficult to obtain, decisions often can be based upon assessments of *relative magnitude.* For example, catastrophic and costly adverse outcomes, such as death and brain damage, usually warrant preventive efforts, even if they are rare.

The third step is the formulation of hypotheses about *cause-and-effect relationships* between the processes of medical care and the adverse outcome. Some hypotheses follow directly from observing the temporal sequence of events. In other situations, the appearance of an adverse outcome may be so delayed or variable that simple observation does not permit the identification of useful hypotheses. Under these circumstances, suggestive relationships or associations may not emerge until a large collection of cases is available for study. It is important to remember that this third step merely identifies possibilities.

After a plausible hypothesis has been identified, it must be *validated.* For this step, well controlled animal models and clinical trials often are required. Ultimately, it should be possible to describe the genesis of an adverse outcome in terms of a stepwise sequence of reproducible biologic processes. As a shorthand, we will refer to this as the *mechanism of injury.*

The fifth and final step involves the *design and confirmation of preventive measures.* This can be challenging. First, the mechanism of injury must be amenable to prevention. Second, we must have resources and technology that make prevention feasible. Third, we must demonstrate that the preventive strategy is effective. And finally, the benefits of the preventive measures must not be overshadowed by excessive costs or injurious side effects.

The foregoing discussion, of course, represents an ideal process. In reality, we often try shortcuts. Shortcuts are especially attractive when we identify potential cause-and-effect relationships or statistical associations that suggest easy and obvious modifications in practice. A well known example is the statistical association between "cardiac risk factors" (*e.g.,* recent myocardial infarction and signs of congestive heart failure) and adverse cardiac outcomes in the perioperative period.[2] These associations have generated a wide variety of preventive strategies, including delay of surgery, coronary revascularization before noncardiac surgery, use of invasive monitors, and specialized treatment protocols for anesthesia and postoperative recovery. Even if a shortcut seems to work, eventually we may face a question that makes us retrace our steps and renew the search for specific mechanisms: Did the observed benefit derive from the shortcut or some other cause-and-effect relationship that has not yet been appreciated? This issue gains particular importance when we wish to expand the success obtained with an existing preventive strategy. An excellent example of the problematic nature of shortcuts can be found in Rao *et al.*'s[3,4] study of perioperative care for patients with recent myocardial infarction.

In this issue of ANESTHESIOLOGY, Warner *et al.* present a fascinating study that might tempt us to pursue shortcuts in the prevention of ulnar neuropathy.[1] Admittedly, the temptation is hard to resist. This is the single largest investigation of perioperative ulnar neuropathy, and it is based upon information obtained from the

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meticulous medical record system of the Mayo Clinic. The authors have taken particular care to assure accuracy and specificity of data. From a descriptive standpoint, the study reinforces many of the distinctive and disconcerting features of perioperative ulnar neuropathy that have been perceived in smaller series for the past several decades.[5–7] These features include an apparent predilection for injury compared to other peripheral nerves, delayed onset or recognition of injury in the postoperative period, persistence of pain and disability beyond 1 yr in approximately half of patients, and limited benefit from surgical remedies such as ulnar nerve decompression and transposition. The use of a large population base and excellent longitudinal tracking also provides one of the first believable estimates of incidence (conservatively, about one case per several thousand noncardiac surgeries). To these data, we can add the liability perspective provided by the ASA Closed Claims Project. Perioperative ulnar neuropathy accounts for approximately 5% of all claims in the current database.[8,*] Payment occurs in 51% of cases, with a median cost of $17,500 and a range of $1,400–$330,000.* Clearly, we have passed the first and second steps in our conceptual framework for accident prevention.

Of special interest, Warner *et al.* take their study beyond a purely descriptive level by using a case-control design that permits a statistical assessment of relative risk. Four significant associations emerge from multivariate analysis: male gender, low body mass index, high body mass index, and prolonged hospitalization. Of additional note, there does not seem to be a clear association with patient position during surgery, duration of surgery, or type of anesthetic.

Does this new information improve our ability to implement preventive strategies? Have we advanced beyond the third step? Not yet. These associations certainly represent a commendable advance in the scientific study of ulnar neuropathy, but they are so *nonspecific* in nature that it remains difficult to hypothesize about cause-and-effect relationships that are clinically useful or amenable to experimentation. Consider the association with male gender. Does this risk arise from body habitus, occupational exposure, hormonal effects, or a combination of these and other relationships? Consider the association with high and low body mass index. Is the mechanism that predisposes to ulnar nerve injury the same at both extremes of body weight, or are two distinct mechanisms at work? Finally, consider the association with prolonged hospital stay. This suggests that factors *outside or beyond* the operating room environment may be involved—but which ones?

Despite the uncertainties, the urge to do something is difficult to resist. In particular, the superficial course of the ulnar nerve in the cubital tunnel of the elbow[9] makes the use of extra padding or special protective devices seem logical and attractive. Before doing so, we should carefully consider our situation. At present, we have no evidence that extra padding produces benefit, nor do we have a validated model that tells us *how* to apply extra padding in the most effective way. The current study by Warner *et al.* provides no additional insights on this topic, because information about armholders, arm-wraps, and padding was not available to the investigators.[1] (For an excellent general review of the unsubstantiated nature of various preventive strategies, we recommend a recent editorial by Stoelting.[10]) We also know that ulnar nerve injury can occur *despite* the specific application of padding: In 14 of 77 (18%) ulnar nerve injuries reported from the ASA Closed Claims Database, reviewers explicitly noted that padding had been placed over the affected nerve.[8] If the use of extra padding adds cost without accompanying benefit, we are simply wasting health-care resources. Of equal concern, extra padding may result in unfavorable outcomes and increased liability. A case that recently entered the ASA Closed Claims Database illustrates this point*: An anesthesiologist used extra arm padding for a patient who was placed in the prone position. The patient exhibited an upper extremity neuropathy in the postoperative period, which some experts attributed to *compression produced by extra padding.*

Of note, Warner *et al.* succumb to temptation. But they do so by means of a suggestion that eventually may lead to a useful contribution. They suggest that patients receive a routine ulnar nerve examination before discharge from the recovery room. Their primary rationale is the belief that early detection may lead to improved outcomes by permitting the prompt initiation of therapeutic modalities such as padding and physical activity restriction. As the foregoing discussion indicates, we regard this as speculative treatment that may introduce an accompanying set of unnecessary risks and costs. Given the small proportion of cases that are symptom-

* ASA Closed Claims Project: Unpublished data.

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atic in the recovery room, we also wonder whether this is the most effective way to conduct a screening effort. However, the investigators offer another rationale that is more compelling: Routine ulnar nerve examination may help advance our understanding of perioperative ulnar neuropathy. Here we agree, but with a note of caution. Screening activities are unlikely to have much impact when conducted in an unstructured manner by isolated groups of practitioners or hospitals. If we wish to gain new insights from the early detection of perioperative ulnar neuropathy, we will need well defined and standardized protocols for nerve examination, data coding, reporting, and intervention. This type of activity is best suited to large institutions and multicenter trials.

On the whole, however, congratulations should outweigh complaints. The current study by Warner *et al.* makes an important contribution by redirecting attention to the critical issue: Perioperative ulnar neuropathy is a clinical entity for which we still have no useful understanding of cause-and-effect relationships. Some clues are accruing, but more research and better answers are needed. Collaborative approaches may prove particularly effective, with clinical investigators, such as Warner *et al.*, identifying general relationships that basic scientists can use to expand the search for specific mechanisms of injury.

Shortcuts are worth considering, but they can lead to confusing detours and unwanted delays when the territory is unfamiliar. At present, a conventional route seems best suited to the exploration of perioperative ulnar neuropathy.

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