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# Ulnar Neuropathy

# Incidence, Outcome, and Risk Factors in Sedated or Anesthetized Patients

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Background: Ulnar neuropathy is well-recognized as a potential complication of procedures performed on anesthetized patients. However, reported outcomes and risk factors for this problem are based on small series and anecdotes.

Methods: We retrospectively reviewed the perioperative courses of 1,129,692 consecutive patients who underwent diagnostic and noncardiac surgical procedures with concurrent anesthetic management at the Mayo Clinic from 1957 through 1991 (inclusive). The medical diagnoses of patients who had these procedures were scanned for 26 diagnoses associated with neuropathy. Persistent neuropathy of an ulnar nerve was defined as a sensory or motor deficit of greater than 3 months' duration. Risk factors anecdotally associated with persistent neuropathy were analyzed by comparing patients with an ulnar neuropathy with control subjects in a 1:3 case-control study.

Results: Persistent ulnar neuropathies were identified in 414 patients, a rate of 1 per 2,729 patients. Of these, 38 (9%) patients had bilateral neuropathies. Approximately equal numbers of the neuropathies included sensory loss only or mixed sensory and motor loss. Initial symptoms for most neuropathies were noted more than 24 h after the procedure. Factors associated with persistent ulnar neuropathy included male

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gender and a duration of hospitalization of more than 14 days (P < 0.01). Neuropathy was more likely to develop in very thin and obese patients than in patients with average body habitus. Neither the type of anesthetic technique nor the patient position was found to be associated with this neuropathy. Of the 382 patients who survived the 1st postoperative yr, 53% regained complete motor function and sensation and were asymptomatic. Of those with neuropathies persisting for more than 1 yr, most had moderate or greater disability from pain or weakness.

Conclusions: These data suggest that perioperative ulnar neuropathies are associated with factors other than general anesthesia and intraoperative positioning. Men at the extremes of body habitus who have prolonged hospitalizations are particularly susceptible to development of ulnar neuropathies. (Key words: Complications: ulnar neuropathy. Positioning: complications; ulnar neuropathy.)

ULNAR neuropathy occurring during the perioperative period is a well-known risk for anesthetized patients undergoing surgical procedures. One-third of nerve injury claims in the American Society of Anesthesiologists' Closed Claims Study involved ulnar neuropathy.<sup>1</sup> The mechanism of this problem is often unknown, although external compression of the ulnar nerve as it passes through the cubital tunnel has been considered to be a potential etiologic factor. 2,3 Because cubital tunnel compression is commonly acknowledged as a possible factor, perioperative ulnar neuropathies often have been considered to be preventable and to occur because of poor intraoperative care (e.g., improper positioning or padding).<sup>4,5</sup> This perception has significant impact on the outcomes of medicolegal cases involving these types of problems.6 Unfortunately, no previous studies have evaluated other risk factors associated with perioperative ulnar neuropathy.

To provide this information, we reviewed the perioperative courses of 1,129,692 consecutive patients who underwent diagnostic and noncardiac surgical procedures with concurrent anesthetic management at one institution during a 35-yr period. The aim of this

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study was twofold: (1) to determine the frequency and outcomes of ulnar neuropathies in this population and (2) to evaluate patient, anesthetic, and procedure factors associated with these neuropathies.

#### **Materials and Methods**

Study Subjects

During the 35-yr period from January 1957 through December 1991, 1,412,116 primary diagnostic and surgical procedures involving all surgical specialties were performed at the Mayo Clinic. For cases in which an individual patient underwent more than one procedure during an anesthetic, only the major procedure has been included in this total and used for the data presented in this study. The procedures were coded using both the Mayo-Berkson<sup>7</sup> and the International Classification of Diseases, 9th revision: Clinical Modification§ systems.8 Based on a review of procedure codes, we identified all diagnostic and noncardiac procedures for which services of the anesthesia care team (i.e., sedation and monitoring; regional or general anesthetics) historically have been used. Procedures that were performed on patients sedated by health care workers other than members of the anesthesia care team or who received only surgeon-administered local anesthetics were excluded. Cardiac procedures were also excluded because of the high incidence of brachial plexus injury associated with sternal retraction and physiologic changes common to cardiopulmonary bypass. 9-12 Based on information in our extensive institutional surgical procedure-indexed database, 282,424 procedures met these exclusion criteria. The remaining 1,129,692 procedures were performed on 873,366 patients. Because each patient encounter was considered to represent a new risk for neuropathy, all 1,129,692 encounters during this interval were included in the base population for this study.

To determine the validity of our assumption that these specific procedures were consistently performed on patients who were either sedated or who received regional or general anesthetics, we reviewed the medical records of 3 randomly selected surgical patients from each month of these 35 yr (a total of 1,260 patients). This review is possible because the Mayo Clinic employs a unit medical record system, and the complete history of every patient, including outpatient as well as inpatient data, is available for review.8 We determined that 995 of the 1,260 diagnostic and noncardiac surgical procedures reviewed belonged to the group of procedures that commonly were performed on patients who received sedation or anesthetics. Of these 995 procedures, 982 (99%) were actually performed with anesthetic involvement. Overall, 1,048 of the 1,260 procedures were performed on patients who were sedated or anesthetized. The 53 patients who were sedated or anesthetized for procedures not thought by us to be historically performed with involvement of anesthetic services underwent 23 different types of procedures. Nine of these procedures involved sternotomy. Based on our clinical experience, we believe that the remaining 14 noncardiac types of procedures have not been consistently performed on sedated or anesthetized patients at Mayo, and they have not been included in our review. Because our method of identifying procedures performed with anesthetic management appears to be reliable, data for the 1,129,692 previously identified procedures were analyzed assuming they were all performed on patients who were either sedated or who received regional or general anesthetics.

Computerized patient identifiers of these 1,129,692 procedures were matched against 26 medical diagnoses for upper-extremity neuropathy. At Mayo, staff physicians determine the major medical diagnoses associated with each patient encounter, and these diagnoses have been manually or electronically recorded since 1909. During the 35-yr study period, these medical diagnoses were coded using both the Mayo-Berkson and the Hospital International Classification of Diseases, adaptation 2 systems. Based on our extensive experience with retrospective Mayo chart reviews, we believe that ulnar neuropathies persisting more than 3 months have been consistently recorded in our medical diagnoses during the 35-yr study period. We are less certain that ulnar neuropathies resolving in less than 3 months have been consistently recorded. Therefore, we strictly defined ulnar neuropathy as a sensory or mixed sensory and motor deficit persisting for more than 3 months. Brachial plexus neuropathies involving the ulnar nerve were not included. Using this definition, we identified 1,132 patients who at any point in time had a diagnosis of ulnar neuropathy.

<sup>§</sup> International Classification of Diseases. 9th revision: Clinical Modification (ICD-9-CM), Volume 3: Procedures. Ann Arbor, Commission on Professional and Hospital Activities, 1968.

<sup>||</sup> Hospital International Classification of Diseases, adaptation 2. 2nd edition. Ann Arbor, Commission on Professional and Hospital Activities, 1968.

With institutional review board approval, the medical records of these 1,132 patients were reviewed to confirm the presence of a persistent ulnar neuropathy and to determine whether it could have been temporally related to the diagnostic or noncardiac surgical procedure. Any nerve injuries described (e.g., in a surgical report) as being either planned or unplanned but part of the procedure were excluded (n = 82). To avoid the inclusion of nerves directly traumatized by a needlestick and therefore unrelated to positioning, patients in whom an ulnar neuropathy developed in an upper extremity after that extremity had been anesthetized with a regional block were excluded (n = 13). Based on these characterizations and chronologic criteria, perioperative ulnar neuropathies occurred in 414 patients.

#### Outcome Analysis

Medical records of these 414 patients with persistent neuropathies were reviewed to determine onset of initial symptoms and the extent and duration of sensory or motor deficits after their procedures. Three hundred eighty-two patients survived for more than 1 yr, and 364 (95%) received medical care at the Mayo Clinic subsequent to that time. Of these, 310 patients (81%) continued to receive care at this institution for more than 5 yr. Correspondence was attempted with the 72 patients who survived for greater than 1 yr but who received medical care at Mayo Clinic for less than 5 yr. Thirty-eight of these 72 patients or their family members responded with information related to the outcome of the patients' neuropathies. The outcomes assessed included the duration of sensory or motor deficits, major symptoms, and effect (mild, moderate, or severe) of any limitation on performance of daily activities.

### Risk Factor Analysis

To ascertain the role of various factors on the development of an ulnar neuropathy, we conducted a case—control study. Three control subjects were matched by procedure type with each patient who had a neuropathy, selecting the three patients who had their procedures closest to the date of the procedure of the patient with the neuropathy. After matching the 414 patients with ulnar neuropathy against 1,242 control

# SAS Procedures: SURVFIT, SURVDIFF, LOGIST, PHGLM, MCSTRAT, and PAIRED, SUGI Supplemental Library User's Guide. Version 5 edition. Cary, SAS Institute, 1986.

subjects, conditional logistic regression was used to assess whether any demographic variables or any other factors previously found to be associated with ulnar neuropathy were associated with this outcome.

Variables analyzed in this study included age (years), gender, body mass index (BMI) (kilograms per meter squared), and variables previously reported to increase the risk of neuropathy or nerve ischemia. 13 Preoperative variables included smoking history (currently smoked vs. never smoked or ceased smoking more than the 30 days before the procedure) and the preoperative presence of diabetes, vascular disease, connective tissue disease, anemia, and chronic alcohol intake. Intraoperative variables evaluated included duration of sedation or anesthetic, type of anesthetic technique (i.e., sedation, regional, or general), and patient position (i.e., supine, prone, lateral decubitus, or lithotomy). The duration of hospitalization was noted. We did not analyze the type of arm-holders. padding, or arm wraps used intraoperatively because this information was unavailable.

A multivariate analysis was performed with an initial model that included as independent variables all factors found to be univariately significant.# The contribution of each factor was assessed by testing the regression coefficient against zero. Factors were removed from the model in a stepwise fashion with the factor showing the smallest contribution deleted at each step. After a factor was removed, the contribution of each risk factor previously removed from the model was reassessed to determine if it now added significantly (P < 0.05) to the model. The model building stopped when all factors remaining in the model had regression coefficients that were significantly different from zero and no other factors outside the model continued to add significantly.

The analyses were based on the entire cohort of 414 patients with an ulnar neuropathy and 1,242 control subjects. For the logistic regression models, smoking history was analyzed by comparing patients who smoked within 1 month of their procedures (current smokers) and those who never smoked or had stopped smoking more than 1 month before the procedure. The type of anesthetic technique was analyzed by comparing patients who underwent general anesthetics and those who received regional anesthetics or sedation. BMI and durations of procedures and hospitalizations were analyzed as continuous variables.

#### Results

Using the methods described above, we identified 1,129,692 diagnostic and noncardiac surgical proce-

dures performed on 873,366 sedated or anesthetized patients during this 35-yr period. Ulnar neuropathy persisting for more than 3 months developed in 414 patients, a rate of 1 per 2,729 patients. Of 798,436 patients receiving general anesthetics, a neuropathy developed in 317 (1 per 2,518). In 71 of 253,164 (1 per 3,566) patients who were sedated and 26 of 78,092 (1 per 3,004) patients who received a regional anesthetic, a neuropathy developed. These patients who had a neuropathy ranged in age from 23–89 yr with a mean age of  $58.3 \pm 18.3$  yr (table 1). Nearly 70% of the patients with an ulnar neuropathy were male. Eigh-

teen of the patients with an ulnar neuropathy had their procedures performed on an outpatient basis.

Forty-seven percent of the deficits associated with these ulnar neuropathies were sensory only, and the remaining deficits were mixed sensory and motor. The involved ulnar nerve was equally distributed between left and right in patients with unilateral involvement. Bilateral symptoms developed in 38 (9%) of the 414 patients with an ulnar neuropathy. In three of 22 patients with preexisting perioperative ulnar neuropathies who underwent subsequent procedures, their neuropathies were either exacerbated or occurred in

Table 1. Univariate Analysis of Patient Characteristics and Other Factors Associated with Ulnar Neuropathy

Characteristics	Patients with a Neuropathy (n = 414)	Controls (n = 1, 242)	Relative Risk	95% CI	P* ·
Age (yr)	58.3 ± 18.3	53.0 ± 14.8	1.0	1.01, 1.08	<0.01
Gender					
Male	289 (70)	584 (47)	4.1	2.16, 9.81	<0.01
Female	125 (30)	658 (53)			
Body mass index (kg/m²)	$25.8 \pm 3.4$	$26.2 \pm 2.8$	0.9	0.86, 0.96	NS
<24	157 (38)	154 (13)	3.7	1.62, 5.07	<0.01
≥38	120 (29)	8 (1)	15.0	3.91, 70.23	<0.01
Preexisting factors					
Diabetes	46 (11)	37 (3)	4.3	2.26, 6.73	< 0.01
Vascular disease	11 (3)	25 (2)	1.3	0.45, 6.53	NS
Connective tissue disease	4 (1)	19 (2)	0.4	0.07, 2.19	NS
Anemia	33 (8)	134 (11)	0.7	0.62, 0.91	NS
Chronic alcohol intake	25 (6)	62 (5)	1.1	0.57, 2.08	NS
Smoking history					
Current (1 month)	79 (19)	185 (15)	1.3	0.65, 3.07	NS†
Past (>1 month)	132 (32)	361 (29)			•
Never	203 (49)	696 (56)			
Surgery and other factors	• •	, ,			
Duration of sedation or anesthetic (min)	$92.1 \pm 57.0$	$82.7 \pm 47.8$	1.1	1.05, 1.16	NS
Type of anesthetic				•	
General	317 (77%)	819 (66%)	1.4	0.72, 3.21	0.025‡
Regional	26 (6%)	87 (7%)		•	•
Sedation	71 (17%)	336 (27%)			
Patient position	` ,	, ,			
Supine	340 (82%)	1013 (82%)	0.4	0.09, 2.41	NS§
Prone	8 (2%)	36 (3%)		•	·
Lithotomy	37 (9%)	74 (6%)			
Lateral decubitus	25 (6%)	100 (8%)			
Other	4 (1%)	19 (2%)			
Duration of hospitalization (days)	9.1 ± 4.2	6.2 ± 3.8	3.1	1.97, 6.13	< 0.01
Range	0-43	0-34			

Values are given as mean  $\pm$  SD or No. (%). CI = confidence interval; NS = not significant.

<sup>\*</sup> Two-tail P value associated with univariate test of the null hypothesis of no association using conditional logistic regression and the 1:3 matched set feature of 414 patients with a neuropathy and 1, 242 controls. This value cannot be computed directly from the information provided.

<sup>†</sup> Analysis comparing current smoker with those who are not current smokers.

<sup>‡</sup> Analysis comparing general anesthesia with other anesthetic techniques.

<sup>§</sup> Analysis comparing supine position with other positions.

the contralateral arm despite apparent efforts to avoid further problems through the use of adequate elbow and arm padding and careful upper-extremity positioning.

The initial symptoms of ulnar neuropathy were noted more than 24 h after the procedure in 57% of patients undergoing general anesthetics and 72% of patients receiving sedation or regional anesthetics. Less than 10% of the initial symptoms were noted in a postanesthesia recovery unit (PARU) (fig. 1). The initial symptoms appeared within 7 days in more than 90% of these patients with a neuropathy. Pain, tingling, and numbness were the most commonly documented initial symptoms.

#### Outcomes

Of the 382 patients who survived the 1st postoperative yr, 53% had regained complete motor function and sensation and were asymptomatic. An additional six percent had regained complete motor function and sensation but still had pain as a symptom. The remaining 41% of the patients had persistent deficits with or without pain at 1 yr. In general, pain was perceived in the ulnar nerve distribution; however, 23% of patients with persistent pain for more than 1 yr described intermittent episodes of generalized forearm and hand discomfort or aching.

Early identification of ulnar neuropathy symptoms in these patients did not appear to result in a greater chance of improvement for these symptoms. Approximately 60% of patients who were known to have neuropathy symptoms in the PARU were asymptomatic in 1 yr, a percentage similar to that of patients who had their symptoms noted later after their procedure. Eighteen of the 414 patients underwent ulnar nerve transposition or cubital tunnel decompression. Ten of these 18 patients noted improvement after these procedures.

Most of the patients with persistent deficits at 1 yr reported that their motor dysfunction or symptoms, especially pain that was often characterized as an intermittent ache, restricted their daily activities moderately or greater. The outcomes of 296 patients with unilateral neuropathies who survived for at least 5 yr after their procedures and who reported (or their family members reported) their postneuropathy courses are shown in table 2. Patients with only sensory deficits 3 months after their procedures had a greater chance of complete recovery at 1 yr than did patients with mixed sensory and motor deficits. Of the 141 patients with only sensory deficits at 3 months, 113 (80%) regained complete

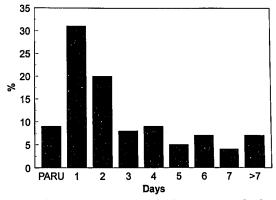


Fig. 1. Distribution of patients with ulnar neuropathy by time from conclusion of the procedure to notation of first symptoms. The onset of symptoms was first noted more than 24 h after the procedure in more than one half of all patients. PARU = postanesthetic recovery unit.

sensation within 1 yr. In contrast, only 54 (35%) of the 155 patients with mixed sensory and motor deficits at 3 months regained complete function and sensation within 1 yr.

A common complaint among patients with persistent deficits or pain was their inability to grip tools and sporting equipment because of either a loss of grip strength or discomfort. Although numbness did not contribute to restriction of activity, it was noted as the most bothersome symptom in more than half of the patients who were still symptomatic at 1 yr.

## Factors Associated with Ulnar Neuropathy

In univariate analyses, factors associated with ulnar neuropathy included patient characteristics, the use of general anesthetics, and length of hospitalization. Of the patient-related characteristics, male gender was a powerful risk factor for ulnar neuropathy (table 1). Other patient-related risk factors for ulnar neuropathy included increasing age and preexisting diabetes. The risk of developing a motor neuropathy increased 35% for each decade of life. Diabetes was present in 11% of patients in whom an ulnar neuropathy developed, compared with 3% of the control subjects (P < 0.01). A greater percent of patients with neuropathies compared with control subjects had BMIs associated with either a thin or obese body habitus. This difference was particularly true of patients with BMIs greater than or equal to 38; 29% of patients with a neuropathy had a BMI greater than or equal to 38 compared with only 1% of control subjects (fig. 2).

Table 2. One- and Five-year Outcomes of 296 Patients with Unilateral Ulnar Neuropathies\*

Type of Neuropathy		
Sensory (n = 141)	Mixed Sensory and Motor (n = 155)	
28 (20%)	101 (65%)	
21	89	
20	79	
25 (17%)	96 (62%)	
18 ′	87	
16	74	
	Sensory (n = 141) 28 (20%) 21 20 25 (17%) 18	

<sup>\*</sup> These data do not include information on 18 patients who underwent surgical procedures to correct their ulnar neuropathies and 35 patients who did not report their five year outcomes.

The use of general anesthetics and a prolonged hospital stay were univariately associated with the development of an ulnar neuropathy (table 1). A greater percentage of patients with ulnar neuropathies compared with control subjects underwent general anesthetics (P=0.025). Regarding length of hospital stays, patients with neuropathies spent greater than 50% more time in the hospital than control subjects ( $9.1\pm4.2$  vs.  $6.2\pm3.8$  days, P<0.01). Ninety-seven (23%) of the 414 patients with an ulnar neuropathy were hospitalized more than 14 days compared with 101 (8%) of the 1,242 control subjects. The duration of anesthetic care and intraprocedure patient position were not found to be associated with this type of neuropathy.

When the factors identified by univariate analyses were considered in a multivariate analysis, only the factors of male gender, low and high BMI, and duration of hospital stay were found to have independent predictive effects for the development of persistent ulnar neuropathy. Increased age, preexisting diabetes, and the use of general anesthetics were not strongly predictive of ulnar neuropathy after adjusting for other factors.

#### Discussion

Although the etiology of perioperative ulnar neuropathy is usually unknown, 1,13-15 improper patient positioning during general anesthesia is often assumed to

be the cause of ulnar neuropathy in litigations. Our data clearly support the conclusion that perioperative ulnar neuropathies can be unrelated to patient positioning during general anesthesia. In a large number of our patients, ulnar neuropathies developed in the absence of general anesthetics. Initial symptoms were noted in three of every five patients with ulnar neuropathy more than 24 h after the conclusion of their procedures. These findings, plus strong associations of several patient characteristics with these problems, suggest that not all perioperative ulnar neuropathies are preventable by improved intraoperative positioning and padding of patients' arms.

Why, in patients receiving sedation or regional anesthetics, do ulnar neuropathies develop? An easy conclusion would be that sedation, like general anesthesia, leads to loss of normal protective responses to early signs of nerve compression and ischemia. If local ischemia and the resultant paresthesias are prolonged, focal demyelination of the nerve, especially the superficial sensory fibers of the nerve as it passes under the arcuate ligament, 16 may lead to sustained axonal damage. 17 Many of our patients, however, underwent short diagnostic procedures with minimal sedation, and their initial symptoms developed more than 24 h later. Compressive and stretch forces on ulnar nerves are not unique to the operating room; it is possible for these forces to occur while patients are resting in beds or easy chairs. Our finding that prolonged duration of hospitalization is associated with an increased risk of this neuropathy further

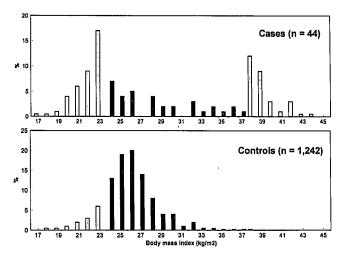


Fig. 2. Distribution of body mass index (BMI) for patients with ulnar neuropathies and control subjects. Open bars = patients and control subjects with a BMI less than 24 or greater than or equal to 38.

<sup>\*\*</sup> Self-reported as being "painful" daily.

<sup>\*\*\*</sup> Self-reported as limiting daily activities moderately or greater.

suggests that the mechanisms of nerve compression and stretch contribute to the development of this problem outside of operating and diagnostic procedure rooms. Prolonged hospitalization is likely to be a marker of various perioperative factors (e.g., complicated postoperative course or poor general health). We did not assess the reasons for our patients' prolonged hospitalizations because of the difficulties of making these determinations by retrospective review.

We were surprised that the initial symptoms of greater than half of our patients were noted more than 24 h after their procedures. In some cases, the patients likely complained of tingling and numbness in their fingers, and their physicians ignored or underestimated the symptoms until later. In others, patients may have been heavily sedated by analgesics or otherwise inhibited from complaining (e.g., during intensive care management). Although analgesics do not affect numbness, the sedative effects of most analgesics may have prevented patients from complaining of this symptom. Still other patients may have been preoccupied with their primary illness and may not have mentioned their ulnar nerve symptoms until later in their recovery. 18 Nonetheless, in a large number of patients, symptoms of neuropathy appear to have developed late in their postprocedure hospital stay.

Delayed onset of neuropathic symptoms have been previously reported. 1,18,19 If an intraoperative event was primarily responsible for the onset of an ulnar neuropathy, it seems unusual that the symptoms of most of our patients were noted more than 24 h after the intraoperative period. For example, intraoperative compression of the ulnar nerve is considered to be a major etiologic factor for this problem. Compressioninduced ischemia in peripheral nerves usually is associated with a rapid decrease in conduction velocity and onset of symptoms, especially pain and numbness.20-22 Delayed or tardy onset of symptoms, especially in previously anesthetized patients who were awake and alert in the PARU or patients who received minimal sedation for diagnostic procedures, suggests that a number of perioperative factors distinct from intraoperative malpositioning contribute to the development of these neuropathies. The identification of risk factors in this study provides a starting point for prospective studies to elucidate further the chronology, mechanisms, and causes of perioperative ulnar neuropathies.

Our patients who were noted to have symptoms of ulnar neuropathy soon after their procedures did not have more favorable outcomes compared with patients with symptoms documented later after their procedures. Why? In general, diagnostic evaluations and medical or surgical interventions offer little chance for improvement to patients with neuropathies. Electrodiagnosis of the problem is confirmatory or may pinpoint the location of nerve insult, but no therapeutic procedure has proven to be uniformly successful in improving the symptoms of ulnar neuropathy. Cubital tunnel decompression, reconstructive nerve repair, or other surgical interventions may be useful in selected cases, but there currently are no well-developed criteria to determine which patients might benefit from these surgical maneuvers.

Despite this finding that early identification of ulnar neuropathy did not lead to improved outcome, we believe that there are several reasons that patients should have their ulnar nerve status routinely assessed and documented before leaving postanesthesia recovery areas. First, early detection of perioperative ulnar neuropathies may lead to improved modalities for their treatment. Although improved modalities might include complex care such as new surgical interventions, they may also be as simple as the use of elbow padding during postoperative bedrest for patients with earlyonset neuropathies. Second, nine percent of our patients with neuropathies had bilateral involvement of their ulnar nerves. Early detection of a neuropathy in one extremity might lead to practice changes (e.g., warning patients to avoid prolonged flexion of their elbows to more than 90° or pressure on their elbows) that would decrease the incidence of contralateral ulnar neuropathy. Third, examination of ulnar nerves in PARUs is simple and inexpensive. Finally, PARU examinations would establish any temporal relation between ulnar neuropathies and surgery and anesthesia. A better appreciation of this relation would assist efforts to understand the mechanisms responsible for this problem.

Male gender and extremes of body habitus may predispose to perioperative ulnar neuropathy. Approximately three fourths of our patients with neuropathy were men, which is similar to the findings of other studies. 1,3,18,19 Stoelting speculates that the cubital tunnel may be more shallow in men than in women. 14 Men may have an increased risk of ulnar nerve compression by external forces or from acute flexion of the elbow. In cadavers, the tension on the arcuate ligament covering the ulnar nerve in the cubital space increases with elbow flexion of more than 90°. 23 This tension could increase ulnar nerve compressive forces, especially in individuals with shallow cubital tunnels. Regardless of mechanism, men are clearly more predisposed to this problem than are women. We also found that thin and obese patients were predisposed to ulnar neuropathy. The peripheral nerves of thin patients may be unusually susceptible to compression or direct nerve damage. Conversely, at least during surgical procedures in which the arms are positioned at the side, it may be more difficult to pad and protect the elbows of obese individuals without a compressive or constricting effect. Efforts to prevent intraoperative mechanisms of ulnar neuropathy in patients undergoing noncardiac procedures should be especially focused on thin and obese men.

Using data from this study for guidance, we have approximated the proportional risks of male gender, BMI less than 24 or greater than or equal to 38, and prolonged hospitalization for development of an ulnar neuropathy (table 3). BMIs of 24 and 38 were chosen for these approximations because of the biphasic changes in risk for thin and obese patients at these BMIs (fig. 2). The high risk of heavy men for development of ulnar neuropathies, especially those who may be hospitalized for more than 2 weeks, indicates that preventive efforts should focus on this patient group.

Patients with preexisting subclinical neuropathies may be particularly susceptible to ulnar neuropathies. 13,14 Miller 24 and Alvine and Schurrer 18 found asymptomatic contralateral ulnar neuropathy (defined by nerve-conduction studies) in nearly all patients with a newly diagnosed postoperative ulnar neuropathy. Neuropathy occurred bilaterally in nine percent of our patients. Obviously, preanesthetic identification by nerve-conduction study of individuals with subclinical ulnar neuropathy would be cost-prohibitive, and it is not clear that this information would be useful to reduce the occurrence of this problem. It would seem prudent in the preanesthetic period, however, to alert any patients with mild ulnar neuropathy symptoms such as nocturnal paresthesia or dysesthesias that these symptoms may be exacerbated in either arm during the postoperative period.

Medicolegal actions against anesthesiologists for ulnar neuropathy are infrequent but persistent and costly. In a review of the American Society of Anesthesiologists Closed Claims Study, Kroll *et al.*<sup>1</sup> reported one third of claims for all neuropathies to involve the ulnar nerve. Of the 1,541 claims for all types of outcomes reviewed in the Closed Claims Study at that time, ulnar neurop-

Table 3. Approximate Proportional Risk of Ulnar Neuropathy after Procedures on Sedated or Anesthetized Patients\*

Characteristic or Factor†	Procedures (n)	Cases (n)	Proportional Risk
None present	475,000	29	1:15,500
Male gender Plus	375,000	69	1:5,400
BMI <24 or ≥38	85,000	158	1:500
Or	50,000	0.4	4.4.700
Hospitalized >14 days	56,000	34	1:1,700
BMI <24 or ≥38	58,000	61	1:1,000
Plus			
Hospitalized >14 days	12,000	20	1:600
Hospitalized >14 days	31,000	5	1:6,200
All factors present	8,000	38	1:200

<sup>\*</sup> These risks are based on the study's multivariate analysis and identification of risk characteristics or factors. They are estimates that have been calculated using assumptions made on data collected at defined intervals during the 35-yr period studied. These assumptions relate to the surgical population and include: (1) 46% are male, (2) 14% have a body mass index (BMI) <24 or ≥38, and (3) 8% are hospitalized >14 days. These assumptions do not necessarily accurately describe the current surgical population. For example, although only 3% of current patients are hospitalized >14 days, approximately 14% of Mayo surgical patients were hospitalized >14 days in 1960. The proportional risk calculations are approximated to the nearest 100.

athies represented five percent of total claims. Although ulnar neuropathies are a small part of medicolegal claims, their long-term consequences are significant. More than 40% of our patients with initial sensory or motor deficits lasting more than 3 months continued to have deficits at 1 yr. This low recovery rate was especially evident in patients with mixed sensory and motor deficits. Four of every five patients with pure sensory deficits had complete recovery at 1 yr compared with only one in every three patients who had mixed deficits. Nearly all patients with residual deficits at 1 yr reported pain and moderate or greater restriction of activities related to their neuropathies.

The utility of this study depends on our ability to identify the occurrence of ulnar neuropathy and associated risk factors in Mayo patients during a 35-yr period. The retrospective nature of this study precluded accurate assessment of all ulnar neuropathies, especially transient neuropathies. To increase our chances of identifying clinically important neuropathies, we included only those patients who had long-lasting sensory or motor neuropathies. To assess the accuracy of identifying procedures in which sedation or anesthetics were given, we reviewed 1,260 randomly

<sup>†</sup> See Methods for definition.

selected procedures performed during the 35-yr study period. Based on this review, we believe our denominator of 1,129,692 cases with sedation or anesthetics to be quite accurate. We evaluated associations between a variety of factors and the occurrence of these motor neuropathies. Unfortunately, other frequently proposed factors such as type of arm boards, padding, arm positions, and preexisting ulnar neuropathy symptoms could not be determined retrospectively, thereby limiting the scope of our evaluation.

In summary, this study found the frequency of ulnar neuropathy with sensory or motor deficits persisting more than 3 months after procedures performed on sedated or anesthetized patients to be low. Male gender, extremes of body habitus, and prolonged hospitalization were associated with an increased risk of developing these neuropathies. The initial symptoms associated with more than one half of these neuropathies were noted more than 24 h after the procedures. The identification of these specific risk factors may assist investigators in their search for the basic mechanisms and causes of perioperative ulnar neuropathies.

#### References

- 1. Kroll DA, Caplan RA, Posner K, Ward RJ, Cheney FW: Nerve injury associated with anesthesia. Anesthesiology 73:202–207, 1990
- 2. Wadsworth TG, Williams JR: Cubital tunnel external compression syndrome. Br Med J 1:662–666, 1973
- 3. Perreault L, Drolet P, Farney J: Ulnar nerve palsy at the elbow after general anesthesia. Can J Anaesth 39:499–503, 1992
- 4. Britt BA, Gordon RA: Peripheral nerve injuries associated with anaesthesia. Can Anaesth Soc J  $11:514-536,\ 1964$
- 5. Dornette WHL: Compression neuropathies: Medical aspects and legal implications. Int Anesthesiol Clin 24:201–209, 1986
- 6. Dornette WHL: Identifying, moving, and positioning the patient, Legal Issues in Anesthesia Practice. Edited by Dornette WHL. Philadelphia, FA Davis, 1991, pp 120–123
- 7. Berkson J: A punch card designed to contain written data and coding. Journal of the American Statistical Association 36:535-538, 1941

- 8. Kurland LT, Molgaard CA: The patient record in epidemiology. Sci Am 245:54-63, 1981
- 9. Lederman RJ, Breuer AC, Hanson MR, Furlan AJ, Loop FD, Cosgrove DM, Estafanous GF, Greenstreet RL: Peripheral nervous system complications of coronary artery bypass graft surgery. Ann Neurol 12:297–301, 1982
- 10. Roy RC, Stafford MA, Charlton JE: Nerve injury and musculoskeletal complaints after cardiac surgery: Influence of internal mammary artery dissection and left arm position. Anesth Analg 67: 277–279, 1988
- 11. Vander Salm TJ, Cereda J-M, Cutler BS: Brachial plexus injury following median sternotomy. J Thorac Cardiovasc Surg 80:447–452, 1980
- 12. Wey JM, Guinn GA: Ulnar nerve injury with open-heart surgery. Ann Thorac Surg 39:358–360, 1985
- 13. Dawson DM, Krarup C: Perioperative nerve lesions. Arch Neurol 46:1355–1360, 1989
- 14. Stocking RK: Postoperative ulnar nerve palsy: Is it a preventable complication? Anesth Analg 76:7–9, 1993
- 15. Williams JR: Postoperative ulnar neuropathy. JAMA 243:1525, 1980
- 16. Sunderland S: The intraneural topography of the radial, median and ulnar nerves. Brain 68:243-299, 1945
- 17. Aguayo A, Nair CPV, Midgley R: Experimental progressive compression neuropathy in the rabbit: Histologic and electrophysiologic studies. Arch Neurol 24:358–364, 1971
- 18. Alvine FG, Schurrer ME: Postoperative ulnar-nerve palsy: Are there predisposing factors? J Bone Joint Surg 69:255-259, 1987
- 19. Cameron MGP, Stewart OJ: Ulnar nerve injury associated with anaesthesia. Can Anaesth Soc J 22:253–264, 1975
- 20. Fullerton PM: The effect of ischaemia on nerve conduction in the carpal tunnel syndrome. J Neurol Neurosurg Psychiatry 26: 385-397, 1963
- 21. Ochoa J, Fowler TJ, Gilliatt RW: Anatomical changes in peripheral nerves compressed by a pneumatic tourniquet. J Anat 113: 433–455, 1972
- 22. Trojaborg W: Rate of recovery in motor and sensory fibers of the radial nerve: Clinical and electrophysiological aspects. J Neurol Neurosurg Psychiatry 33:625-638, 1970
- 23. Wadsworth TG: The external compression syndrome of the ulnar nerve at the cubital tunnel. Clin Orthop 124:189–204, 1977
- 24. Miller RG: The cubital tunnel syndrome: Diagnosis and precise localization. Ann Neurol 6:56–59, 1979