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Ulnar Nerve Pressure

Influence of Arm Position and Relationship to Somatosensory Evoked Potentials

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Background: **Although the ulnar nerve is the most frequent site of perioperative neuropathy, the mechanism remains undefined. The ulnar nerve appears particularly susceptible to external pressure as it courses through the superficial condylar groove at the elbow, rendering it vulnerable to direct compression and ischemia. However, there is disagreement among major anesthesia textbooks regarding optimal positioning of the arm during anesthesia.**

Methods: **To determine which arm position (supination, neutral orientation, or pronation) minimizes external pressure applied to the ulnar nerve, we studied 50 awake, normal volunteers using a computerized pressure sensing mat. An additional group of 15 subjects was tested on an operating table with their arm in 30°, 60°, and 90° of abduction, as well as in supination, neutral orientation, and pronation. To determine the onset of clinical paresthesia compared to the onset and severity of somatosensory evoked potential (SSEP) electrophysiologic changes, we studied a separate group of 16 male volunteers while applying intentional pressure directly to the ulnar nerve. Data are presented as mean (median; range).**

Results: **Supination minimizes direct pressure over the ulnar nerve at the elbow (2 mmHg [0; 0–23]; n = 50), compared with both neutral forearm orientation (69 mmHg [22; 0–220]; $P < 0.0001$), as well as pronation (95 mmHg [61; 0–220]; $P < 0.0001$). Neutral forearm orientation also results in significantly less pressure over the ulnar nerve compared to pronation ($P \leq 0.04$). The estimated contact area of the ulnar nerve with the weight-bearing surface was significantly ($P < 0.0001$) smaller in the supine position (2.2 cm^2 [0.5; 0–9]; n = 50) compared with both neutral orientation (5.5 cm^2 [5.0; 0–13]) and pronation (5.8 cm^2 [6; 0–12]). With the forearm in neutral orientation, ulnar nerve pressure decreased significantly ($P \leq 0.01$; n = 15) as the arm was abducted at the shoulder from 0° to 90°. In the 16 male subjects tested, notable alterations in ulnar nerve SSEP signals (decrease ≥ 20% in N9–N9′ amplitude) were detected in 15 of 16 awake males during application of intentional pressure to the ulnar nerve. However, eight of these subjects did not perceive a paresthesia, even as SSEP waveform amplitudes were decreasing 23–72%. Two of these eight subjects manifested severe decreases in SSEP amplitude (≥ 60%).**

Conclusions: **Extrapolating these results to the clinical setting, the supinated arm position is likely to minimize pressure over the ulnar nerve. With the forearm in neutral orientation, pressure over the ulnar nerve decreases as the arm is abducted between 30° and 90°. In addition, up to one half of male patients may fail to perceive or experience clinical symptoms of ulnar nerve compression sufficient to elicit SSEP changes. (Key words: Medical malpractice; neuropathy; perioperative nerve injury; positioning; pressure mat.)**

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ULNAR neuropathy is the most common perioperative nerve injury, generating one third of nerve injury claims in the American Society of Anesthesiologists' (ASA) Closed Claims Study database.[1] These injuries result chronic pain, permanent disability, and economic damages, and frequent litigation.[1,2] Only 1 of 20 claims identified the proposed mechanism of nerve injury, and in

more than two thirds of cases the closed claim reviewers judged that the standard of care had been met. However, payments for ulnar nerve damage still ranged from $2,000 to $330,000.[1]

Although the true mechanisms of anesthesia-related ulnar neuropathy remain undefined, it has been assumed that external pressure exerted against the nerve is a likely etiologic factor.[3-5] Stoelting[5] speculated that external pressure of the ulnar nerve coursing within the rigid bony compartment of the superficial condylar groove at the elbow can produce ulnar nerve compression, resulting in nerve ischemia. Indeed, several reports suggest the ulnar nerve is more susceptible to ischemia than are the radial or median nerves within the arm.[4,6] Nevertheless, there is no consensus regarding positioning of the arm during anesthesia to minimize pressure applied to the ulnar nerve. Some authors suggest that abduction of the arm to $> 60°$ and supination of the hand and wrist put the nerve at risk.[7] Others disagree and stress the opposite—the need to avoid pronation of the forearm.[5,8] Recent editorial opinion cautions anesthesiologists against making "assumptions" and "shortcuts" when considering ulnar neuropathy and recommends a systematic investigation of factors related to positioning of the arm and interactions with the ulnar nerve.[9] Thus, our overall goal was to develop quantitative and physiologic models of peripheral ulnar nerve injury and to examine the key relationships between (1) arm position and nerve pressure and (2) paresthesia and electrophysiologic changes. If successful, these models will allow us to move beyond qualitative, inferential data provided by traditional anatomic studies, or *post hoc* analysis of case reports. We believe such an approach, although not providing instant remedies, can improve our interim decisions about clinical care and be a guide for future studies. Specifically, we therefore tested the consequences of varying arm positions in a series of investigations to determine:

Phase A: the maximal, passive, spontaneous pressure exerted directly over the ulnar groove, with the forearm (abducted at 90°) placed in supination, pronation, and neutral orientation, with the weight of the arm supported on a firm surface.

Phase B: the maximal pressure exerted over the ulnar groove, with the forearm placed in supination, pronation, and neutral orientation, and with the arm abducted at 30°, 60°, or 90°, with the weight of the arm cushioned by a standard 2-inch foam pad, secured to a operating table arm board.

Phase C: the onset of clinical paresthesia in volunteers, compared to the onset and severity of somatosensory evoked potential (SSEP) electrophysiologic changes, during application of intentional pressure to the ulnar nerve.

Methods

After review and approval by the Clinical Research Practices Committee of Wake Forest University School of Medicine, written informed consent was obtained from all volunteer research subjects. The same device was used for determination of pressure around the ulnar nerve for the three phases (A, B, C noted previously) of the investigation and is described henceforth. Patients rested the selected arm on this device, a thin, flexible, moisture-resistant pressure sensor pad (Xsensor Technology Corporation, Calgary, Alberta, Canada). This pad contains 1,296 microsensors, each of which sample at 5 Hz (each) to record pressure between 0 and 220 mmHg (the sensor pad is calibrated at the factory and at yearly intervals thereafter; fig. 1).[10,11] Pressure readings were recorded *via* a 32-bit proprietary software program on a Dell (Dell Computer, Round Rock, TX) laptop computer, for later playback and analysis. A viscous white paste identified the precise location of the ulnar groove 4 cm superior and inferior to the olecranon, and the contact area was summated (to 1 cm^2 resolution) *via* a qualitative grading scale of minimal, moderate, or complete coverage for each cm^2. This surface map was then compared to the pressure distribution graph generated by the Xsensor Technology software program (fig. 2).

Anatomically, supination and pronation describe the radius rotating within the radial groove of the ulna in the forearm. However, the term *supination,* as conventionally applied in relationship to positioning patients for surgery, also invokes external rotation of the humerus. This external rotation of the humerus appears to anatomically rotate the postcondylar groove away from the resting surface when moving from pronation to supination. Conversely, pronation is associated with internal rotation of the humerus, and may increase the contact between the supporting surface and the contents of the postcondylar groove. With this understanding, we refer to supination and pronation *in proxy* for the clinical

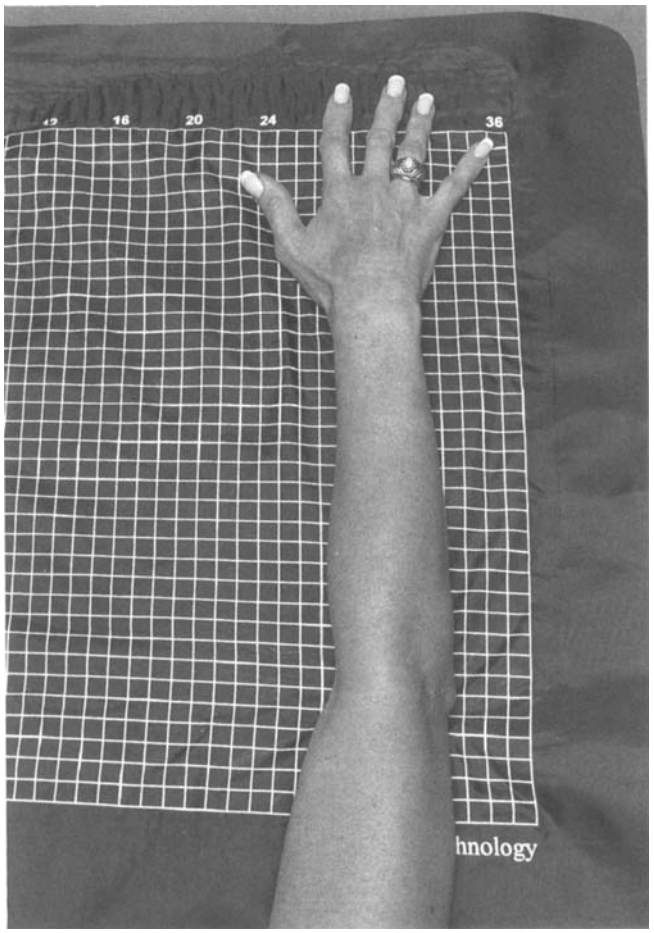

Fig. 1. Photograph of the 46 cm × 46 cm flexible, pressure sensor pad that contains 1,296 embedded microsensors, each 0.64 mm thick. The pressure mapping software is calibrated to sample each cell at 5 Hz and determines pressure between 2 and 220 mmHg.[10,11] The subject's arm shown here is resting passively with the forearm in pronation, part of the protocol described in Phase A. The viscous medical paste used to identify exactly the ulnar groove is not visible from the angle of this photograph.

application of these terms, which includes rotation of the humerus, as noted previously.

Phase A

Fifty unpaid volunteers were recruited by a random sampling of family members accompanying patients scheduled for evaluation in the Department of Anesthesiology Preanesthesia Assessment Clinic. These subjects completed a demographic questionnaire, and were rejected if afflicted with any of the following illnesses: rheumatoid arthritis, carpal tunnel syndrome, diabetes, cervical disk or nerve problems, renal failure, cirrhosis, hypothyroidism, lymphoma, multiple myeloma, polycythemia vera, porphyria, vitamin deficiencies, amyloidosis, or acromegaly. In addition, volunteers were excluded if they required any of the following medications: hydralazine, pyridoxine, amiodarone, dapsone, isoniazid, metronidazole, phenytoin, or vincristine. These seated subjects rested their dominant arm at approximately 90° abduction on the pressure sensitive pad (described previously) draped over a wooden table top, with maximal pressures recorded in each of three orientations of the forearm: full supination, neutral orientation, and full pronation. The order of positioning was alternated (subject 1: supine, neutral, pronated; subject 2: pronated, neutral, supine; and so forth) among subjects.

Phase B

Fifteen unpaid, adult volunteers were placed in the supine position with their dominant arm at 30°, 60°, and 90° abduction resting on the pressure sensor pad draped over a standard padded arm board (2-inch foam pad) attached to a Quantum 3080 RC operating room table (Amsco International, Pittsburgh, PA). Maximal pressures (mmHg) over the ulnar nerve were recorded in each of the three arm positions for abduction (fig. 3), as well as with the forearm in supination, pronation, or neutral orientation.

Phase C

Nineteen unmedicated, male volunteers in the supine position rested their nondominant arm (pronated and abducted to 60°) on a firm surface. These subjects received financial compensation for their time and participation in this part of the investigation. SSEP waveforms were recorded using a Viking IV Instrument (Nicolet Biomedical, Madison, WI) with silver-silver chloride self-gelled stimulating electrode pads placed over the ulnar nerve at the wrist. Stimulus parameters included programmed stimulation at 4.7 Hz, a duration of 0.2 ms, and an intensity of 15–25 mA. The stimulus intensity was determined individually based on (1) minimizing motor movement, (2) SSEP waveform resolution, and (3) subject's tolerance to stimulation, as recommended by Chiappa.[12] N9 is a short-latency SSEP with the brachial plexus being the wave generator. This was chosen because the waveform configuration is less variable within and among subjects, especially compared to the evoked potentials of longer latencies.[13] Once set, the intensity was not changed for the duration of each study. SSEPs were recorded using silver EEG surface electrodes (10

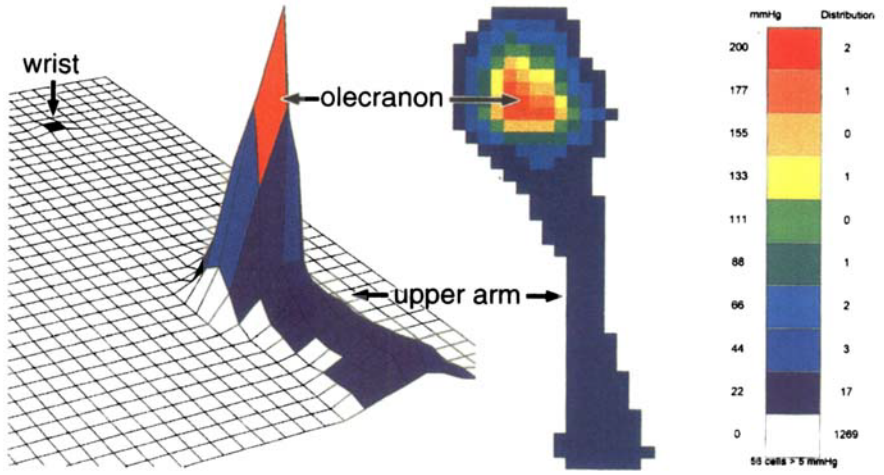

Fig. 2. Two- and three-dimensional color image of the pressure map distribution for a typical subject, with the arm resting passively in pronation on the sensor mat. Color code corresponds to various pressure ranges, as shown on the scale. The display will also specify the exact pressure exerted on each individual cell of the map. The precise location of the ulnar groove is then correlated with this pressure map. Labels identify the location of the wrist, olecranon, and upper arm on this image.

mm) and electrode paste. Using the International 10-20 System, electrode placements were as follows: Cp4-Fpz and Cp3-Fpz for cortical recordings; C5s (5th cervical spine)-Fpz for cervical and subcortical recordings; and EpL (left)-EpR (right) for peripheral recordings. All impedances were matched and maintained less than 3 kΩ, and filters were set 30-1,000 Hz. Between 500 and 1,000 stimuli were averaged for the final recording at each data collection time point. The following parameters were recorded: the EP latency of N9 (Erb's point; brachial plexus; maximal over the supraclavicular fossa), and the peak to peak amplitude of N9 to N9′. This diphasic, positive-negative waveform recorded at the Erb's point electrode after stimulation of a peripheral nerve of the upper limb is generated by the ascending volley in motor and sensory fibers as it approaches and passes through the brachial plexus. Most of this negativity is generated in sensory fibers.[12] SSEP changes were considered significant and severe if amplitude decreased ≥ 60% in the N9-N9′ waveform recordings compared to baseline values.[14,15] This threshold was chosen because it is the conventional clinical criterion for predicting neurologic complication.[14-16] In addition, intraoperative thresholds are set at this high level to limit false-positive results.[12,13,16] We wished to ascertain more subtle degrees of neurologic dysfunction, *before* the onset of irreversible injury to the nerve, and therefore we also defined an intermediate category as a decrease in amplitude of ≥ 20% and < 60%. Indeed, there is some evidence that decreases in SSEP amplitudes in the range of 20-50% result in mild to severe neurologic changes, such as numbness or deficits in other sensory modalities.[1]

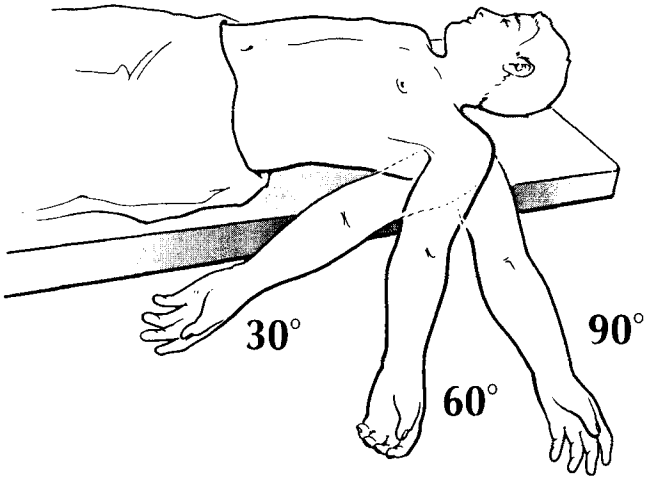

Fig. 3. Superimposed image demonstrating the three arm positions tested: 30°, 60°, and 90° abduction. During the experimental protocol in 15 adult supine volunteers, the order of arm position was alternated between subjects.

After electrode placement and stable baseline recordings were established, a 7- or 8-mm diameter wooden dowel was selected for each subject (chosen to fit snugly into the ulnar groove) and anchored in the ulnar groove with tape, allowing the weight of the arm to rest directly on the wooden block. Pressure on the ulnar nerve was monitored *via* the pressure sensor pad interposed between the arm and the inflexible surface supporting the arm. Subjects were specifically instructed to report the first symptoms of tingling, numbness, paresthesia, weakness, or altered temperature sensation distal to the elbow in their test extremity. In addition, these subjects were prompted at 5-min intervals to confirm or deny the existence of these symptoms. SSEP and pressure measurements were recorded every 5 min until subjects complained of significant hand paresthesia, or for a max-

Table 1. Pressure Recorded over the Ulnar Nerve of 50 Volunteers with the Forearm in Three Positions

	Total Arm Pressure (mmHg)			Total Arm Contact Area (cm^2)			Ulnar Nerve Pressure (mmHg)			Ulnar Nerve Contact Area (cm^2)			Number of Subjects with No Pressure on the Ulnar Nerve
Arm Position	Mean	Median	Range	Mean	Median	Range	Mean	Median	Range	Mean	Median	Range	
Supination	1,020	950	220–2,300	36	35	12–67	2	0	0–23	2.2	1	0–9	44
Neutral	1,000	890	190–2,900	42	41	13–101	69	22*	0–220	5.5	5*	0–13	14
Pronation	1,010	970	50–2,400	41	39	10–97	95	91*†	0–220	5.8	6*	0–12	7

*$P \leq 0.0001$ by Mann-Whitney U test (supine *vs.* pronated and neutral).

†$P \leq 0.05$ by Mann-Whitney U test (pronated *vs.* neutral).

imum of 60 min. Maximal decreases in N9-N9′ wave amplitude (compared with baseline), as well as the corresponding mean ulnar nerve pressure were recorded and analyzed. Analysis focused on Erb's point (a recording electrode superficial to the brachial plexus that records a deflection occurring 9-10 ms after the stimulation), consistent with investigations of the ulnar nerve in previous reports.[4,15,16]

Statistical Analysis

All data are presented as mean (median; range) because some continuous variables (*e.g.*, ulnar nerve pressure) were not normally distributed. Data for Part A were analyzed using the Mann-Whitney U nonparametric test. Total arm pressure was the arithmetic sum of all cells on the pressure mat detecting pressure $\geq$ 2 mmHg (threshold set to eliminate mat artifact). The upper pressure limit of this system is 220 mmHg. Total arm contact surface was the numeric sum of all cells detecting pressure, as noted previously. The ulnar nerve contact area was the sum of contact cells identified by the viscous paste localizing the ulnar nerve. Data for Part B were analyzed using the nonparametric Friedman two-way analysis of variance (ANOVA). When a factor was found significant ($P \leq 0.05$), subgroups were compared with the Mann-Whitney U-test. For Part C, the maximal percent change in the SSEP decrease in wave (N9-N9′) amplitude (compared with baseline), as well as pressure on the ulnar nerve, were analyzed with the Mann-Whitney U-test. In all analyses, data were entered into STATVIEW 4.1 (ABACUS Concepts, Berkeley, CA), and a $P \leq 0.05$ considered significant.

Results

Part A

Fifty subjects, mean age 41 (40; 26-78) yr, volunteered to participate in the investigation of the relationship between arm position and ulnar nerve pressure. These volunteers, weighing 76 (73; 55-105) kg, were right-hand dominant in 40 of 45 cases (5 subjects did not specify hand dominance). The subjects' relaxed arm exerted nearly identical total pressure (sum of all cell pressures) against the pressure sensing mat whether the forearm was in supination, neutral orientation, or pronated (table 1, column 1). Thus, the consistency of our methodology was internally validated. Likewise, the total contact area of the arm against the mat was the same whether the arm was in supination (36 [35; 12-67] cm^2), neutral orientation (42 [41; 13-101] cm^2), or pronated (41 [39; 10-97] cm^2). Conversely, pressure localized over the ulnar nerve was greatest with the forearm pronated, especially compared to supination ($P < 0.0001$; table 1). Indeed, with the forearm in supination, only 6 of 50 subjects manifested *any* pressure over the ulnar nerve (range, 10-23 mmHg). This was true despite the significantly smaller ulnar nerve contact with the mat compared to either neutral orientation or pronation ($P \leq 0.0001$). Interestingly, there was no significant correlation between nerve contact area and ulnar nerve pressure in supine ($P = 0.19$), neutral ($P = 0.97$), or pronated position ($P = 0.92$). In addition, there was no significant correlation between ulnar nerve pressure and subjects' height (correlation coefficients of -0.15 to 0.29; $P > 0.05$) or subjects' weight (correlation coefficients of -0.03 to 0.12; $P > 0.05$).

Part B

These 15 subjects ranged in age between 24 and 44 yr (mean = 33; median = 33) and weighed 65 (62; 47-93) kg. Two subjects were left-hand dominant. All enrolled subjects completed the protocol lying supine on a standard operating room table with an attached arm board, both of which were padded with standard conductive foam pad. Pressures (mmHg) over the ulnar nerve are summarized in figure 4. Identical to Part A above, pronation of the forearm significantly increased ulnar nerve

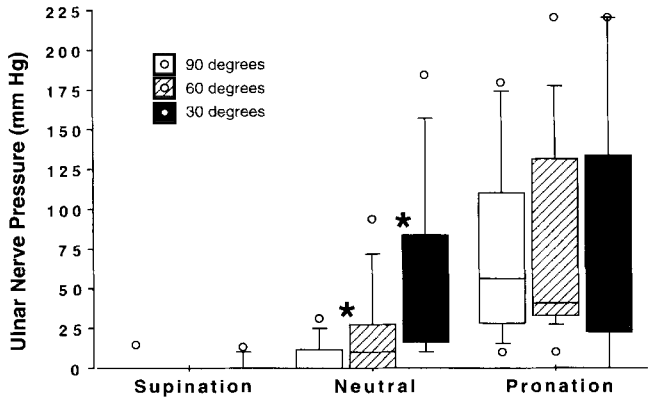

Fig. 4. Box and whiskers plot of peak ulnar nerve pressure with the forearm in supination, neutral, or pronation orientation, concurrent with the arm abducted 30°, 60°, or 90° at the shoulder. *$P < 0.01$ comparing 90° abduction with both 30° and 60° abduction with the forearm in neutral orientation. The horizontal lines for each plot represent the 10th, 25th, 50th, 75th, and 90th percentile of ulnar nerve pressure in each group. The small open circles represent outliers beyond the bounds of the 10th and 90th percentiles of each data set. In supination, the pressure over the ulnar nerve is uniformly low, and most of the data are clustered around the zero line.

pressure (*i.e.*, pronated > neutral > supine [$P < 0.0001$]). In neutral orientation, pressure on the ulnar nerve decreased as the arm was abducted from 30° to 90° ($P < 0.01$). However, with the forearm in supination, the mean pressure over the ulnar nerve was uniformly low (1 [0; 0-15] mmHg), regardless of the degree of abduction of the arm at the shoulder.

Part C

Sixteen male subjects, aged 31 (26; 19-51) yr, weighing 79 (80; 59-93) kg, completed the study protocol. Three additional subjects did not complete the study. One subject was unable to cooperate during placement of the wooden block in the ulnar groove, whereas technical or computer software problems resulted in unusable data from two other subjects. Eight subjects complained of a progressive hand paresthesia 37 (33; 20-59) min after placement of the wooden block in the ulnar groove, which exerted a mean ulnar nerve pressure of 80 (69; 41-153) mmHg. All eight of these subjects also manifested SSEP changes with a mean decrease in the N9-N9′ amplitude of − 44% (−45; −20- −71%). Two of these subjects manifested severe changes. There was not a significant correlation between ulnar nerve pressure and change in SSEP N9 amplitude in these subjects ($P = 0.30$). By contrast, eight volunteers reported no ulnar paresthesia even after 60 min ($P = 0.0003$; table 2) of pressure from the wooden block in the ulnar groove (mean ulnar nerve pressure = 59 [40; 14-167] mmHg). Despite the absence of a perceived paresthesia, these eight subjects had similar SSEP decrease in the N9-N9′ waveform amplitude of −44 (−45; −19- −72)%. Again, two of these eight subjects manifested severe changes. Note, however, one subject had erratic SSEP signals (subject #9), and the changes in SSEP amplitude for subject #11 failed to reach either the severe or intermediate thresholds for significance. Again, there was not a significant correlation between ulnar nerve pressure and decrease in SSEP amplitude ($P = 0.66$) in this group.

Discussion

Peripheral neuropathies may result from excessive pressure, stretch, ischemia, or laceration of a nerve during surgery and perhaps from other as yet unknown causes.[18] Perioperative ulnar neuropathy may occur as infrequently as 0.04% after noncardiac surgery,[2] or as often as 33% after cardiac surgery.[19] The most recent, prospective data defines the incidence as 1:215 in adults undergoing noncardiac surgery.[20] Numerous factors have been observed coincident with perioperative ulnar nerve injury, including induced or prolonged hypotension,[6,21] automated blood pressure cuffs,[22] subclinical diabetes or other unrecognized medical illness,[21,23] local anesthetic toxicity,[24] manipulations of the brachial plexus during cardiac surgery,[6,25] and, of course, positioning during anesthesia.[7] In addition, factors such as extremes of body habitus, prolonged hospitalization, and male gender are associated with increased risk of ulnar neuropathy.[2] Nonetheless, most authors acknowledge that ulnar neuropathy remains a clinical entity for which we still have minimal understanding of cause-and-effect relationships,[3] nor whether it is always a preventable complication.[5] Indeed, accumulating evidence suggests ulnar nerve injury can occur at any time during hospitalization.[2,20]

Consistent with previous observations,[8] we found forearm position to be a robust and significant factor in determining pressure over the ulnar nerve. Supination minimized direct pressure exerted over the ulnar nerve, even when one accounted for (or perhaps because) the fact that this position produces the least contact area between the ulnar nerve and the weight-bearing surface. Although supination minimized direct pressure, pronation of the forearm produced the largest pressure to the ulnar nerve, regardless of the abduction of the arm between 30° and 90°. With the forearm in neutral ori-

Table 2. Data from 16 Subjects with Somatosensory Evoked Potential Monitoring during Intentional Application of Pressure to Ulnar Nerve*

Subject	R/L Handed	Age (yr)	Weight (kg)	SSX	Time SSX	Ulnar Pressure (mmHg)	Ulnar Area (cm^2)	Pressure per cm^2	Category of N9–N9′ Change	%N9–N9′
1	Left	33	78	Yes	30	61	7	8.8	Intermediate	−58
2	Left	25	84	Yes	30	110	7	15.7	Severe	−71
3	Right	24	73	Yes	59	41	7	5.8	Intermediate	−44
4	Right	25	66	Yes	35	90	7	12.9	Intermediate	−20
5	Left	35	75	Yes	20	153	6	25.4	Severe	−60
6	Right	39	73	Yes	45	77	7	11.0	Intermediate	−28
7	Right	51	82	Yes	25	59	8	7.4	Intermediate	−45
8	Right	43	93	Yes	50	47	6	7.9	Intermediate	−27
Mean		34	78		37	80	6.9	11.9		−44
Median		34	77		33	69	7.0	9.9		−45
Range		24–51	66–93		20–59	41–153	6–8	5.8–25.4		−20 to −71
9	Right	40	91	No	60	62	7	8.8	Intermediate (erratic SSEP signal)	−34
10	Right	23	84	No	60	167	6	27.9	Intermediate	−45
11	Right	19	71	No	60	14	8	1.8	No change	−19
12	Right	26	74	No	60	103	8	12.8	Intermediate	−51
13	Right	22	82	No	60	26	7	3.8	Intermediate	−45
14	Right	23	89	No	60	21	8	2.7	Intermediate	−23
15	Right	26	59	No	60	23	5	4.7	Severe	−66
16	Right	34	93	No	60	53	7	7.6	Severe	−72
Mean		27	80		60	59	7.0	8.8	No change - 1	−44
Median		25	83		60	40	7.0	6.2	Intermediate - 11	−45
Range		19–40	59–93		—	14–167	5–8	1.8–27.9	Severe - 4	−19 to −72
P value†		0.09	0.49		0.0003	0.21	0.57	0.17		0.92

SSX = verbal confirmation of symptoms of ulnar nerve paresthesia by the subject; No change = 0–19%; Intermediate = 20–59%; Severe = 60% decrease in amplitude.

* Subjects are grouped by those who reported ("Yes") or denied ("No") ulnar nerve paresthesia during the protocol. The protocol was then terminated.

† Mann-Whitney U test comparing the group who reported ulnar nerve paresthesia (n = 8) with those who denied symptoms of ulnar nerve paresthesia (n = 8).

entation, pressure over the ulnar nerve actually decreased as the arm was abducted from 30° to 90°.

Significant alterations in ulnar nerve SSEP signals were detected in 15 of 16 awake, male volunteers in response to application of direct pressure to the ulnar nerve. Two of the four subjects with severe SSEP changes, and 5 of 11 subjects with intermediate changes (7 of 15), did not perceive a paresthesia, even as the decrease in N9-N9′ amplitude ranged between 23% and 72%. Extrapolation to the clinical setting would suggest that up to one half of male patients who experience pressure on peripheral nerves (sufficient to precipitate electrophysiologic changes in nerve function) may be "at risk" because they do not perceive a concurrent paresthesia of that ulnar nerve.

Although it is generally recognized that most SSEP components are mediated by large myelinated fibers, some secondary, negative peaks may be carried by smaller fibers. The potential recorded from Erb's point is perhaps the most sensitive to ischemia,[26] and therefore we and others[4,16] chose this marker as the primary analysis endpoint in characterizing the waveform changes occurring during the experimental period. We also focused on amplitude changes because they are believed to be a more sensitive and valid measure of changes in nerve condition, compared to changes in latency.[17,27] We assumed that the intense local pressure of the arm resting directly on a small wooden peg results in local compression and ischemia of the ulnar nerve. The superficial ulnar nerve is trapped between the two rigid surfaces of the bony tunnel of the olecranon and the hard wooden dowel (fig. 5). Recent investigations support the concept that ulnar nerve ischemia may be a potent mechanism for, and a final common pathway of, ulnar nerve dysfunction, and perhaps perioperative neuropathy.[4,6,26,28,29] The axial magnetic resonance image in figure 5 also identifies the close proximity of the ulnar collateral artery and vein to the ulnar nerve itself, and one could envision external pressure (simulated in fig. 5B) affecting arterial inflow, venous outflow, or both, of the endoneural vasculature. Nonetheless, we do recognize that some other potentials subserved by large my-

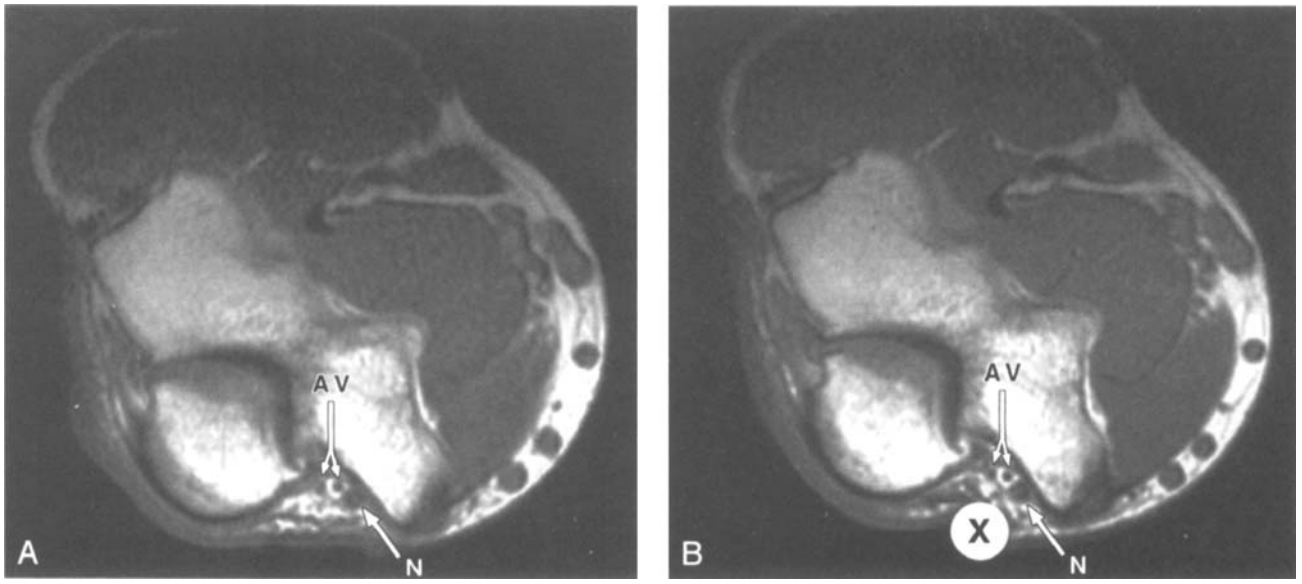

Fig. 5. (*A*) Magnetic resonance image in the axial plane of the ulnar nerve (N) as it courses through the rigid, superficial condylar groove at the elbow in a 90-kg, 34-yr-old man who was a subject in Part C of the experiments. Also identified is the ulnar collateral artery and vein (AV). (*B*) For illustration, a test tube filled with oil ("X") is added to simulate a possible position of the wooden dowel described in Part C of the experimental protocol, wherein intentional pressure to the ulnar nerve is induced by allowing the weight of the arm to rest on a rigid object affixed to the ulnar groove. These conditions may create tissue pressure that could induce direct compression of the nerve against the bony prominence of the medial epicondyle or interrupt nutrient blood flow to the nerve *via* compression of the closely approximated ulnar collateral artery and vein (AV).

elinated fibers (such at P9, P14, and N18) may also be sensitive markers to ischemia, and debate continues in characterizing SSEP waveform alterations during various pathologic states.[16,26]

Lorenzini and Poterack[16] tested neurologic symptoms as a surrogate endpoint for stretch related injury in a group of 14 awake volunteers (13 of 14 were males). These subjects were tested while they were in the prone position, with ulnar (and median) nerve stress produced by progressive cephalad movement of the arms over their head. Similar to our results, those authors found 7 of 14 subjects reported arm paresthesias, but only four of them manifested simultaneous SSEP changes. They required an increase of latency of 10% or a decrease in amplitude of 60% for significance. They therefore concluded that the lack of SSEP changes in three of these seven symptomatic volunteers rendered SSEP signals an imperfect monitor for positioning injury.[16] Several differences exist between their study and ours. The mechanism of their ulnar nerve "injury" would most likely be stretch as the arms are extended up over the head, although it is unclear exactly how this prone positioning stresses the ulnar nerve. We believe we were investigating ulnar nerve compression with endoneural vascular pressure, producing nerve ischemia.[28] In addition, the site(s) of their injury was multifactorial at the brachial plexus, the humeral head, and so forth. By contrast, our study isolated a single nerve (the ulnar) with a focused stress (compression) within a discrete, reproducible location (the condylar groove at the elbow). Thus, different mechanisms of ulnar nerve injury (*i.e.*, ischemia *vs.* stretch), different nerves, and different anatomical sites of injury may have different propensity for producing changes in SSEP waveforms. Although SSEP monitoring may be an imperfect monitor for stretch injury of the brachial plexus, we believe it remains a valid, sensitive, and early indicator of nerve ischemia induced by compression (as in our experiment) or complete interruption of arterial blood flow (as in tourniquet techniques used in other experiments) of the ulnar nerve at the elbow.[26]

We chose to study male volunteers exclusively in Phase C of this study. Men are four times (95% confidence intervals, 2.2–9.8) more likely to suffer perioperative ulnar neuropathy.[1,2] Therefore, it seemed justified to focus these initial investigations on the population known to be most at risk. Discrete differences in anatomy around the ulnar nerve may contribute to the increased susceptibility of men to external pressure at the elbow. Contreras *et al.*[30] demonstrated that the medial

aspect of the elbow in females is enveloped by 2–19 times more fat content compared with males. In addition, the tubercle of the coronoid process of the medial epicondyle is 150% larger in men compared with women, a likely area for external pressure to elicit a nerve injury perioperatively.[30]

Several limitations are evident in our current investigation. Our novel methodology using the pressure sensing mat was limited by the spatial resolution of 1 cm^2. We could only monitor surface pressure over the nerve, and we must assume these pressures were transmitted to the nerve similarly among different subjects. Although the research nurse was not blinded to the subjects' arm positions, she was the single observer consistently recording the contact area on the mat. Although ulnar neuropathy is the most common perioperative nerve injury,[1,2] it is still uncommon enough (1:215 to 1:2,500) that a prospective, randomized study would be impractical. Injury to a peripheral nerve may occur through multiple mechanisms, including pressure, stretch, vascular ischemia, or direct crush or laceration of a nerve or other as yet unknown ways. This study was limited to issues surrounding arm positioning and pressure, which we assumed to be an important and common mechanism of perioperative nerve injury.[5,7,8] We assumed that direct pressure on the ulnar nerve is harmful, and if uninterrupted, would lead to nerve dysfunction and injury. However, one could reasonably assume that pressure on the ulnar nerve of short duration will not lead to permanent sequelae. In addition, we recognize that in certain situations, more than one mechanism may actually be involved, such as with "double-crush" syndrome.[31,32] Also, there are multiple possible sites besides the olecranon where "excessive" ulnar nerve pressure can occur.[33] Finally, one can argue that the intrinsic stability of SSEP recordings over time is important to the interpretation of our results. Therefore, after the original protocol was complete, N9–N9′ SSEP waveforms (Erb's point) were recorded from two subjects, without any external pressure in the ulnar groove. The maximal spontaneous decreases in amplitude over 60 min were 12.5% and 17.3%, respectively. One of these subjects (#5, table 2) previously demonstrated a 60% decrease in SSEP amplitude and experienced ulnar nerve paresthesia 20 min into the original protocol.

Other assumptions are also evident. We recognize that the subjects enrolled in this study were basically healthy young adults, with no known preexisting neuropathy, neurologic or metabolic disease, or detectable anatomical abnormalities of the ulnar nerve or condylar groove at the elbow. In clinical practice, patients are encountered with various risk factors (obesity, diabetes, metabolic derangements, ulnar nerve dislocation, and so on) that may place them at greater risk for developing nerve dysfunction in the operating room environment. Also, it would obviously not be reasonable or ethical to actually induce permanent nerve injury in volunteers. We assumed, based on experimental evidence, that the conditions in Part C that induced SSEP changes and paresthesia, if allowed to persist, could produce a permanent nerve injury. It seems inconceivable that permanent nerve injury would occur in the absence of SSEP changes, and that persistent neurologic injury is likely related to the duration of SSEP alterations. Although SSEP measurements document alterations in sensory transmission along the ulnar nerve, they do not detect alterations in the motor components of the nerve. But, the ulnar nerve is a relatively homogenous nerve, and there is no evidence for a difference in susceptibility between sensory and motor components. We did find a deficiency of perception of paresthesias associated with ulnar nerve compression in half of our male subjects using the severe and the intermediate criteria. Current investigations are underway to determine if there is a gender difference in perception generally, or perhaps specifically of the ulnar nerve.

It has been argued that "but for negligence" (*i.e.*, improper positioning of the affected arm in an anesthetized patient), ulnar neuropathies should not occur. Contrary to these assumptions, ulnar neuropathy can and does occur in the absence of predisposing conditions, depression of consciousness, or trauma.[34] In addition, our data provide evidence that changes in ulnar nerve function can occur in the absence of clinical paresthesia in awake, unsedated men. Furthermore, the ASA Closed Claims Study investigators were unable to define a breech of the standard of care in 94% of patients with perioperative ulnar neuropathies.[1] Warner *et al.*[2,20] found that certain demographic factors (gender, body size, time in hospital, and so forth) predisposed patients to perioperative ulnar neuropathy. Interestingly, we have shown that certain arm positions that have been advocated as safe for the ulnar nerve[7] (and therefore within the standard of care) may actually increase the pressure over the ulnar nerve. Finally, we have also demonstrated that a substantial number of volunteers, even when prompted at regular intervals, fail to report symptoms of ulnar nerve paresthesia, even with direct pressure on the nerve sufficient to reduce the N9 SSEP amplitude by 23–72%. In summary, our data confirm that

ulnar nerve compression and dysfunction can occur in the absence of symptoms. Nevertheless, we cannot exclude that patients might spontaneously move in response to the ulnar nerve pressure, even if they do not experience a paresthesia.

Our data provide clear evidence that supination of the forearm minimizes pressure over the ulnar nerve during positioning of the supine, adult, surgical patient. In the neutral position, ulnar nerve pressure decreases as the arm is abducted between 30° and 90°. Half of our male subjects failed to experience clinical symptoms of ulnar nerve paresthesia during intentional nerve compression pressure sufficiently severe to elicit SSEP changes. Indeed, the "delayed" reporting of symptoms of perioperative ulnar nerve palsies may be unrelated to the vigilance of the anesthesiologist, but rather due to unperceived nerve compression that takes place after the patient leaves the operating room.[20]

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