

Effect of Ibuprofen on Masking Endodontic Diagnosis

Jason K. Read, DMD MS,* Scott B. McClanahan, DDS MS,* Asma A. Khan, BDS PhD,[†] Scott Lunos, MS,[‡] and Walter R. Bowles, DDS MS PhD*

Abstract

Introduction: An accurate diagnosis is of upmost importance before initiating endodontic treatment; yet, there are occasions when the practitioner cannot reproduce the patient's chief complaint because the patient has become asymptomatic. Ibuprofen taken beforehand may "mask" or eliminate the patient's symptoms. In fact, 64%–83% of patients with dental pain take analgesics before seeing a dentist. The purpose of this study was to examine the possible "masking" effect of ibuprofen on endodontic diagnostic tests. **Methods:** Forty-two patients with endodontic pain underwent testing (cold, percussion, palpation, and bite force measurement) and then received either placebo or 800 mg ibuprofen. Both patients and operators were blinded to the medication received. One hour later, diagnostic testing was repeated and compared with pretreatment testing. **Results:** Ibuprofen affected testing values for vital teeth by masking palpation 40%, percussion 25%, and cold 25% on affected teeth with symptomatic irreversible pulpitis and symptomatic apical periodontitis. There was no observed masking effect in the placebo group on palpation, percussion, or cold values. When nonvital teeth were included, the masking effect of ibuprofen was decreased. However, little masking occurred with the bite force measurement differences. **Conclusions:** Analgesics taken before the dental appointment can affect endodontic diagnostic testing results. Bite force measurements can assist in identifying the offending tooth in cases in which analgesics "mask" the endodontic diagnosis (*J Endod* 2014;40:1058–1062)

Key Words

Analgesic, apical periodontitis, bite force transducer irreversible, pulpitis

Before initiating endodontic treatment, an accurate diagnosis is required that reproduces the patient's dental complaint. This requires the consideration of multiple variables and may involve testing methods using palpation, percussion, cold, heat, and/or an electric pulp test (EPT) (1, 2). However, there are occasions when the practitioner cannot reproduce the patient's chief complaint because the patient is no longer symptomatic upon examination. In this case, with no positive radiographic or testing indications, most clinicians will opt to defer treatment and send the patient home with instructions to return to the dental office once the symptoms have returned. One hypothesis for this situation is that medication taken preoperatively, such as ibuprofen, could "mask" or decrease the patient's symptoms. The effect of analgesics on endodontic diagnostic testing and the impact of these drugs on common endodontic testing methods are not well understood. As early as 1963, Mumford (3) suggested dental EPT as a means of comparing pain-relieving drugs. He also noted that painful pulpal inflammation alters mechanical and thermal pain thresholds; yet, EPT thresholds were not different during pulpal inflammation (4). A later prospective double-blind study evaluating the intraosseous injection of glucocorticoid for tooth pain reduction showed these patients reported less pain and less percussion pain (5).

A study looking at lay management strategies for coping with tooth pain showed that 84% of patients had tried some form of self-care strategy before seeking the care of a professional (6). Of the different strategies attempted, 64% of patients attempted to relieve their odontalgia with over-the-counter analgesics. However, this strategy only resulted in temporary relief or reduced pain intensity for half of these patients. Another study concluded that 81%–83% of emergency patients with moderate to severe pain will have taken some type of medication(s) to help control their pain, and more women than men with irreversible pulpitis will take an analgesic (7). Of the patients who did take preoperative medication, relief occurred 62%–65% of the time. This suggests that most patients presenting to the dental clinic with acute dental pain will have taken analgesics before their dental visit. The remaining patients often will be instructed by clinicians to take ibuprofen to relieve their tooth pain. In fact, the majority of endodontists will recommend 600 mg ibuprofen 4 times a day for patients in pain and not allergic to nonsteroidal anti-inflammatory drugs (NSAIDs), regardless of the patient's pain level, endodontic diagnosis, or treatment provided (8). Dental clinicians should have a flexible analgesic strategy that begins with ibuprofen if the patient health history permits. Often, this will be sufficient for mild to moderate pain of odontogenic origin (9, 10). The maximum dose of ibuprofen is 3.2 g over 24 hours, and this drug is recommended for the management of both preoperative and postoperative pain in dentistry where inflammation is involved (11). Unlike opioids, NSAIDs do not impair consciousness and are available over-the-counter, which makes them more accessible and less costly than prescription alternatives. A recent Cochrane systematic review found good evidence to support ibuprofen as an effective and safe analgesic in adults with minimal adverse effects. The 2007 league table of analgesic efficacy states that 600–800 mg ibuprofen is very effective in the management of acute pain (12).

The diagnosis of pulpal and apical conditions can be very complicated and inaccurate. Previous studies have shown that some patients have a reduction in mechanical pain thresholds (mechanical allodynia), which is manifested as sensitivity to percussion, biting, or pressure (13). Most practitioners will use a mirror handle to test for sensitivity to percussion, or they will have the patient bite on a device such as a Tooth Slooth (Professional Results, Inc, Laguna Niguel, CA) (2). Unfortunately, these tests do not provide quantitative data and can yield variable results. Moreover, these tests can be subjective and produce a large margin for error (14).

From the *Division of Endodontics, University of Minnesota School of Dentistry, Minneapolis, Minnesota; [†]Department of Endodontics, University of North Carolina-Chapel Hill School of Dentistry, Chapel Hill, North Carolina; and [‡]Biostatistical Design and Analysis Center, University of Minnesota, Minneapolis, Minnesota.

Address requests for reprints to Dr Walter Bowles, Division of Endodontics, University of Minnesota School of Dentistry, 8-166 Moos Tower, 515 Delaware Street SE, Minneapolis, MN 55455. E-mail address: bowle001@umn.edu 0099-2399/\$ - see front matter

Copyright © 2014 American Association of Endodontists. <http://dx.doi.org/10.1016/j.joen.2014.05.004>

Recently, a diagnostic instrument for the measurement of mechanical allodynia was tested to measure mechanical pain thresholds on normal healthy patients (14). The results of this study indicated that this bite force transducer has substantial test-retest reliability and fair to substantial inter-rater reliability. This bite force transducer has potential for repeated clinical measurements when subjects are followed over time. The fair to substantial inter-rater reliability suggests that clinical trial designs should include only 1 examiner to collect the mechanical threshold values.

The purpose of this randomized double-blind placebo-controlled clinical trial was to quantitatively measure the effect of ibuprofen on mechanical allodynia in patients with odontalgia caused by symptomatic apical periodontitis (15) and to measure the effect of ibuprofen on endodontic diagnostic tests.

Materials and Methods

The protocol for this study was approved by the university institutional review board. Patients presenting to the School of Dentistry Graduate Endodontics Clinic seeking treatment for the relief of pain of odontogenic origin were screened for possible inclusion. It was determined that a sample size of 20 in each group would have 80% power to detect a difference in means of 0.91 standard deviation using a paired *t* test with a 0.05 2-sided significance level. Patients included in the study provided informed consent and information about all medications taken in the previous 24 hours. Inclusion criteria included patients having a premolar or molar with a clinical diagnosis of symptomatic apical periodontitis. Exclusion criteria included the following: American Society of Anesthesiologists physical status of >3, periodontal pocketing >6 mm, absence of the contralateral tooth, sensitivity to percussion in the contralateral tooth, persistent use of medication such as steroids and antidepressants (which could alter the pain report), use of NSAIDs in the previous 12 hours, and NSAID allergy.

Once enrolled in the study, patients were asked to rate their present odontogenic pain and maximum pain using a verbal numeric rating scale (VNRS) (16). Buccal and lingual gingivae were palpated over both the contralateral and affected teeth to assess sensitivity to palpation. Both the contralateral tooth and the affected tooth were percussed with a mirror handle to determine percussion sensitivity. Then, the contralateral and affected teeth were tested using Endo Ice (Hygenic Corp, Akron, OH). A large cotton pellet was sprayed for 3–5 seconds, which was similar to the procedure described by Jones (17). The contralateral uninflamed tooth, the affected tooth, and the patient's contralateral and affected adjacent 2 teeth were percussed, palpated, cold tested, and examined for mobility.

The contralateral, unaffected tooth's bite force (mechanical pain threshold) and the affected tooth's bite force were measured using the bite force transducer (GM10 Occlusal Force-Meter; Nagaro Keiki, Tokyo, Japan). The bite force transducer was modified by attaching the head of a Tooth Slooth to the end of the biting tab using acrylic resin (Fig. 1) (14). The bite force transducer was placed on the subject's contralateral (control) tooth, and the subject was instructed to bite down on the bite force transducer with instructions (Fig. 1). This procedure was repeated 4 more times for a total of 5 mechanical pain threshold measurements recorded for the contralateral tooth. In addition, the examiner obtained 2 more readings and recorded the mechanical pain thresholds of the inflamed, affected tooth using this same procedure. The method in this study is similar to previous studies for measuring mechanical allodynia (14, 18). Randomization was determined by a random digit table using even-odd numbers by a separate investigator not involved with patient treatment, with packets sequentially numbered based on randomization coding. Both the

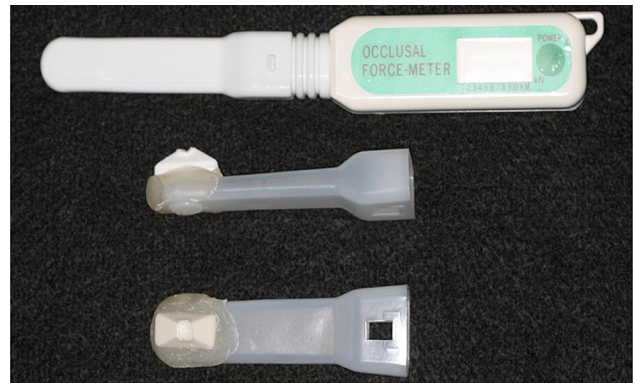


Figure 1. Bite force transducer modified by attaching the head of a Tooth Slooth to the detachable plastic sleeve. The patient was given the following instructions: “This device measures how hard you can bite down. It is similar to a scale. If you jump or move on a scale, then you will not receive a consistent reading. The same is true for this device. This device requires constant pressure to produce an accurate measurement. Therefore, I would like you to gently close until your upper and lower teeth first contact the device. When I say ‘begin,’ bite down as hard as you can with constant pressure until you hear a beep. Once you hear a beep, the device has produced a measurement. The beep usually takes 3 to 5 seconds. I will do this 5 times on the side that does not hurt and only 2 times on the side that hurts.”

treating dentist and patient were unaware of treatment allocation, and the treating dentist enrolled participants into randomized drug allocation. After baseline measurements were gathered, the examiner administered either 800 mg ibuprofen or placebo to the patient (randomized, double blind). One hour later, endodontic diagnostic testing (cold, percussion, and palpation) and mechanical pain threshold measurements for the contralateral control tooth (5 mechanical threshold measurements) and the affected tooth (2 mechanical pain threshold measurements) were repeated as described previously.

Data and the assignment of the test group (ibuprofen or placebo) were uncovered by the statistician and tabulated to summarize the averages of pre- and post-bite force measurements. Descriptive statistics were used to summarize the demographics, patient characteristics, and outcome measures. Two group *t* tests were used to compare the mean change in the outcomes from pretreatment to post-treatment between the groups. *P* values <.05 were considered statistically significant. SAS V9.1.3 (SAS Institute Inc, Cary, NC) was used for the analysis. Pearson and Spearman rho correlations were calculated to determine the comparison of mechanical pain thresholds (bite force) to percussion and palpation and to compare the association of palpation to percussion preoperatively. In addition, a Wilcoxon signed rank test was used to compare the before and after measurements of cold tests (response or no response), palpation (sensitive or not sensitive), and percussion (sensitive or not sensitive) that were assigned ordinal values.

Results

Forty-two subjects were enrolled; however, 3 subjects were unable to complete this study. One subject had an upper complete denture and was unable to bite down on the bite force transducer without dislodging his upper denture. The other 2 subjects could not bite down hard enough on the bite force transducer to produce a measurement. Therefore, they were excluded from the study, and 39 subjects were included for analysis.

Of the 39 subjects, there were 21 women and 18 men. Nineteen subjects received ibuprofen, and 20 subjects received placebo. The

TABLE 1. Summary of Palpation, Percussion, and Cold Tests by Treatment Group

	Ibuprofen (n = 19)		Placebo (n = 20)		Total (n = 39)	
	Pre	Post	Pre	Post	Pre	Post
Palpation, n (%)						
NS = 0	5 (26)	10 (53)	12 (60)	13 (65)	17 (44)	23 (59)
S+ = 1	14 (74)	9 (47)	8 (40)	7 (35)	22 (56)	16 (41)
Percussion, n (%)						
NS = 0	1 (5)	3 (16)	0	2 (10)	1 (3)	5 (13)
S+ = 1	16 (84)	15 (79)	18 (90)	16 (80)	34 (87)	31 (79)
S++ = 2	2 (11)	1 (5)	2 (10)	2 (10)	4 (10)	3 (8)
Cold, n (%)						
NR	8 (42)	9 (47)	8 (40)	9 (45)	16 (41)	18 (46)
RNL	3 (16)	4 (21)	1 (5)	0	4 (10)	4 (10)
S + NL	0	0	2 (10)	0	2 (5)	0
S + L+	8 (42)	6 (32)	8 (40)	10 (50)	16 (41)	16 (41)
S++L++	0	0	1 (5)	1 (5)	1 (3)	1 (3)

L+, lingering; L++, prolonged lingering; NL, non-lingering; NS, non-sensitive; Post, 1 hour after giving the test drug; Pre, before giving the test drug; RNL, responsive, non lingering; S+, mildly sensitive; S++, moderately sensitive.

age of the participants ranged from 19–77 years old, with a mean age of 48. Twenty-eight subjects were white, 6 were black, 2 were Asian, 2 were Hispanic, and 1 was Middle Eastern. The top 3 most prevalent pretreatment diagnoses of affected teeth were symptomatic irreversible pulpitis (SIRP)/symptomatic apical periodontitis (SAP) (44%), necrotic/SAP (23%), or previously treated/SAP (18%) (15). When comparing vital pulp (asymptomatic irreversible pulpitis and SIRP) versus nonvital pulp (necrotic, previously initiated, and previously treated) diagnoses, 51.3% of affected teeth contained vital pulps and 48.7% contained nonvital pulps. The subjects' mean current VNRS was 1.36, and the subjects' mean maximum VNRS was 6.72.

The responses and percentage of each categoric response for percussion, palpation, and cold test was recorded before giving the test drug and 1 hour after giving the test drug to the patient (Table 1).

Notably, of the 19 subjects in the ibuprofen treatment group, 5 of the subjects who were sensitive to percussion (S+) preoperatively changed to nonsensitive (NS) to percussion 1 hour after the administration of ibuprofen (27% decrease in S+ subjects from pre- to post-treatment). One subject changed from moderately sensitive (S++) to slightly sensitive (S+), 3 subjects changed from slightly sensitive to NS, and 1 subject changed from NS to S+. This was a net decrease in sensitivity to percussion by 11%. Two subjects in the placebo group who were S+ to percussion preoperatively changed to NS postoperatively for a net 10% decrease in sensitivity to percussion.

The Wilcoxon signed rank test was used to measure the difference in pretreatment and post-treatment for both ibuprofen and placebo. There was no statistically significant difference for palpation or percussion ($P = .07$ and $P = .66$, respectively).

As one would expect, the mechanical pain thresholds were highest among normal uninflamed teeth (RNL) and lowest among the most symptomatic teeth (S++L++). We compared the actual pretreatment mechanical pain thresholds from the contralateral healthy tooth to the affected inflamed tooth for both treatment groups (Table 2). As expected, the mechanical pain thresholds for the contralateral teeth were higher than the affected inflamed teeth. The postdrug mechanical pain thresholds were higher than the 1-hour prior predrug measurements. The mechanical pain thresholds for the contralateral (control) teeth increased an average of 24 N for the ibuprofen group and 25 N for the placebo group. Also, the mechanical pain thresholds increased an average of 20 N for the ibuprofen group and 33 N for the placebo group on the affected teeth. The paired *t* test revealed no statistically significant difference for change in mechanical thresholds on the contralateral or affected teeth ($P = .94$ contralateral, $P = .61$ affected). Because the biting force remains relatively constant compared with the control teeth values (both treatment group and control increased post-treatment), the overall biting force on the affected teeth remained lower compared with the control teeth, both pre- and postdrug.

When analyzing a subset of the data with a tooth diagnosis of SIRP/SAP, we observed that the postdrug mechanical pain thresholds of

TABLE 2. Outcomes by Treatment Group

	Ibuprofen (n = 19)	Placebo (n = 20)	Total (n = 39)
Contralateral healthy tooth bite force, mean (SD)			
Pre	197.19 (133.94)	160.08 (110.97)	178.16 (122.51)
Post	221.11 (147.26)	185.08 (110.00)	202.63 (129.06)
Change*	23.92 (48.01)	25.01 (35.54)	24.47 (41.51)
Affected tooth bite force, mean (SD)			
Pre	99.42 (51.16)	114.43 (94.47)	107.12 (75.89)
Post	119.13 (73.16)	147.58 (147.65)	133.73 (116.80)
Change*	19.71 (45.66)	33.15 (106.32)	26.60 (81.77)
Delta value, mean (SD)			
Pre	-97.77 (123.24)	-45.65 (51.16)	-71.04 (98.99)
Post	-101.97 (114.08)	-37.51 (73.16)	-68.91 (116.51)
Change*	-4.21 (71.20)	8.15 (109.21)	2.13 (91.67)
Palpation, mean (SD)			
Pre	0.74 (0.45)	-45.65 (51.16)	0.56 (0.50)
Post	0.47 (0.51)	-37.51 (73.16)	0.41 (0.50)
Change*	-0.26 (0.45)	8.15 (109.21)	-0.15 (0.37)
Percussion, mean (SD)			
Pre	1.05 (0.40)	1.10 (0.31)	1.08 (0.35)
Post	0.89 (0.46)	1.00 (0.46)	0.95 (0.46)
Change*	-0.16 (0.50)	-0.10 (0.31)	-0.13 (0.41)
Cold, mean (SD)			
Pre	2.53 (1.47)	2.65 (1.27)	2.59 (1.35)
Post	2.53 (1.61)	2.95 (1.00)	2.74 (1.33)
Change*	0.00 (1.15)	0.30 (0.92)	0.15 (1.04)

Post, 1 hour after giving the test drug; Pre, before giving the test drug; SD, standard deviation.

*Two-group *t* test *P* values are .94, .61, and .68, respectively. Exact Wilcoxon test *P* values for palpation, percussion, and cold, respectively (.09, .54, and .22). No statistically significant differences were found between the groups.

affected teeth did not increase compared with the predrug measurement, whereas mechanical pain thresholds of the contralateral teeth increased an average of 33 N (Table 3). This would be beneficial for the diagnosis of inflamed vital teeth because no masking after ibuprofen was seen with the bite force, whereas on contralateral teeth the bite force increased 20%–28%.

In this group of teeth with inflamed vital pulps, ibuprofen affected palpation and percussion values by masking palpation 40% and percussion 25% in patients (SIRP/SAP, Fig. 2). In cold testing, which could only be tested with this group, ibuprofen treatment resulted in masking the cold response by 25%, with the placebo having no effect on masking the cold response in inflamed teeth. When comparing the masking effects of ibuprofen on all diagnostic groups versus this subset of SIRP/SAP, ibuprofen masked palpation the most (36.5% vs 40%) and percussion less (11% vs 25%) on affected teeth. During this clinical trial, no masking effect in the placebo treatment group on palpation and percussion values was observed. Although bite force values did increase in patients with a diagnosis of SIRP/SAP after treatment with ibuprofen, these values were still much lower than the bite force values of control teeth, clearly indicating which tooth presented with mechanical allodynia. This suggests that bite force is a more sensitive diagnostic measurement not affected by ibuprofen pretreatment.

In this present study, 19 changes from preoperative to 1 hour after drug administration were observed in the combined cold test, palpation test, and percussion test. After reviewing the *t* tests, there was no significant difference of mechanical allodynia between men and women pre- or post-treatment ($P = .45$ and $P = .08$, respectively).

Discussion

It has been hypothesized that analgesics taken before the dental appointment could affect the endodontic diagnostic testing results and thus endodontic treatment. In healthy teeth (no pulpal or apical inflammation) tested with EPT and cold, 10 mg hydrocodone/1,000 mg

TABLE 3. Outcomes by Treatment Group (symptomatic irreversible pulpitis/symptomatic apical periodontitis subset)

	Ibuprofen (n = 8)	Placebo (n = 9)	Total (n = 17)
Contralateral healthy tooth bite force, mean (SD)			
Pre	140.53 (81.63)	136.98 (44.26)	138.65 (62.44)
Post	168.70 (97.40)	174.24 (48.78)	171.64 (73.13)
Change*	28.18 (55.21)	37.27 (42.01)	32.99 (47.30)
Affected tooth bite force, mean (SD)			
Pre	109.75 (46.98)	103.78 (52.11)	106.59 (48.30)
Post	106.56 (43.78)	97.39 (46.10)	101.71 (43.86)
Change*	-3.19 (36.85)	-6.39 (63.29)	-4.88 (50.99)
Delta value, mean (SD)			
Pre	-30.78 (96.84)	-33.20 (69.37)	-32.06 (80.69)
Post	-62.14 (84.34)	-76.86 (58.88)	-69.93 (70.02)
Change*	-31.36 (74.48)	-43.66 (47.20)	-37.87 (59.84)
Palpation, mean (SD)			
Pre	0.63 (0.52)	0.33 (0.5)	0.47 (0.51)
Post	0.38 (0.52)	0.33 (0.5)	0.35 (0.49)
Change	-0.25 (0.46)	0.00 (0.00)	-0.12 (0.33)
Percussion, mean (SD)			
Pre	1.00 (0.53)	1.00 (0.00)	1.00 (0.35)
Post	0.75 (0.46)	1.00 (0.00)	0.88 (0.33)
Change	-0.25 (0.71)	0.00 (0.00)	-0.12 (0.49)
Cold, mean (SD)			
Pre	2.00 (0.00)	2.11 (0.33)	2.06 (0.24)
Post	1.50 (0.93)	2.11 (0.33)	1.82 (0.73)
Change*	-0.50 (0.93)	0.00	-0.24 (0.66)

Post, 1 hour after giving the test drug; Pre, before giving the test drug; SD, standard deviation.
*Two-group *t* test *P* values are .71, .90, and .69 respectively. Exact Wilcoxon test *P* values for palpation, percussion, and cold, respectively (.21, .15, and .21).

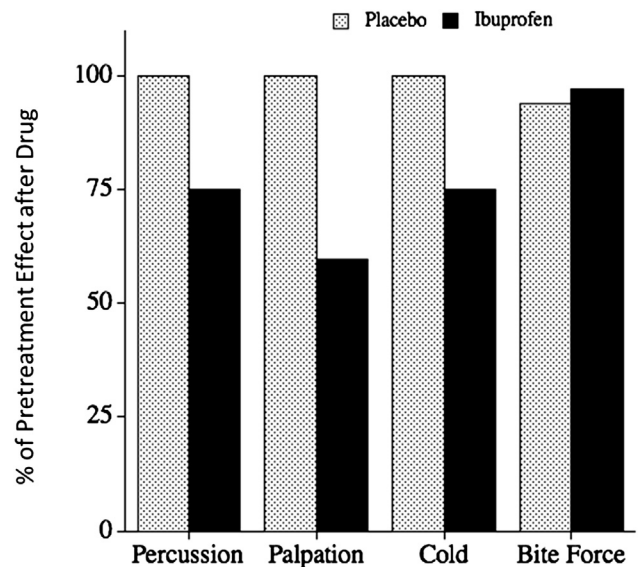


Figure 2. Percent of pretreatment effect after taking ibuprofen or placebo in vital teeth (SIRP/SAP subset). $n = 8$ –9/group.

acetaminophen did not affect diagnostic pulp tests (19). It would be interesting to see the results of a similarly designed study analyzing the effects of a commonly prescribed narcotic (10 mg hydrocodone/650 mg acetaminophen) on cold and EPT in male and female subjects with symptomatic teeth.

Others have researched the effect of a narcotic, NSAID, acetaminophen, and placebo on the pain thresholds measured by EPT on symptomatic teeth (20). They concluded that acetaminophen was the only treatment drug that had a significant difference on EPT pain thresholds between preoperative administration and 45 minutes later. Although there was a numeric difference in EPT readings, this cannot be considered clinically meaningful. The results from the EPTs were 33 preoperatively and 36 forty-five minutes later. Clinically, the results from the EPT are diagnostically the same. The presence of a response indicates vital tissue is present, whereas the absence of such a response usually indicates pulp necrosis. The exact number of the reading is of no significance and does not detect subtle degrees of vitality nor can any EPT indicate inflammation (2).

Although the intraobserver reliability reported for the bite force transducer has been reported as satisfactory (0.63–0.68), it is suggested that only 1 examiner collect the mechanical pain thresholds values because of the fair to substantial inter-rater reliability (0.3–0.64) (14). This previous study showed higher average contralateral mechanical pain thresholds preoperatively than those found in this study (277.1 ± 44.4 vs 178.16 ± 122.51 N, respectively). In addition, the preoperative measurements of the affected teeth were slightly lower compared with this study (83.9 ± 16.7 vs 107.12 ± 75.89 N). Because of the previously mentioned inter-rater reliability, it could be inaccurate to compare mechanical pain thresholds obtained in this study with those measurements gathered by a different examiner in previous studies (13, 18). We observed a difference in bite forces depending on the placement of the bite force transducer. Higher mechanical pain thresholds could be recorded when the long axis of the bite force transducer was aligned mesial to distal on the occlusal table. If the bite force transducer is positioned in a slight buccal to lingual orientation to the clinical crown, then the device engaged a single cusp and resulted in slightly lower measurements.

In this study, the mechanical pain thresholds for affected teeth with a diagnosis of SIRP/SAP were 23%–55% lower when compared with the contralateral (control) teeth, which one might expect for a tooth with apical periodontitis. In addition, the mechanical pain thresholds for all affected teeth (both vital and nonvital) in this study were reduced by 29%–50% compared with the contralateral (control) teeth.

Preoperative bite force values were similar for men and women in our patients. Statistically, there was no difference in preoperative or postibuprofen mechanical allodynia measurements between men and women. According to Nusstein and Beck (7), more men than women received pain relief after taking analgesics for acute dental pain. Therefore, the potential for ibuprofen to mask endodontic diagnoses would likely be higher in a group of men than women. However, this effect was not observed in this study.

It should be noted that of the 39 symptomatic patients who participated in this study, 19 changes from preoperative to 1 hour after drug administration were observed in the combined cold test, palpation test, and percussion test, suggesting that ibuprofen can cloud some of the diagnostic issues, although the overall need for endodontic therapy was often the same. However, in 1 patient, the predrug diagnosis was completely different from their postdrug diagnosis. The change in the predrug diagnosis (SIRP/SAP) to the postdrug diagnosis (normal/normal) would have changed the advised treatment from proceeding with root canal therapy to monitoring the previous symptomatic tooth for future treatment. Therefore, this patient would have been instructed to go home and return to the dental clinic when symptoms reappear without taking any analgesics before the dental visit. Ibuprofen treatment resulted in a normal cold response and normal palpation and percussion tests within 1 hour of drug administration to this patient with the previous diagnosis of SIRP/SAP. In this particular patient, the contralateral control tooth bite force before ibuprofen treatment was 248 N, and after ibuprofen, it was 238 N. The bite force of the affected tooth before ibuprofen was 97 N, and after ibuprofen, it was 131 N, which is still significantly below the control tooth bite force and indicative of mechanical allodynia. This tooth had been sensitive to percussion before drug treatment; yet, after ibuprofen, it tested not sensitive to percussion and the pulp tissue still tested vital. This reinforces the need for a more quantifiable test and one that is not “inherently variable in their force vectors and subjective endpoints” (18). One earlier study looking at bite force reductions after wisdom tooth removal found ibuprofen to increase bite force, but this was not significant until 3 hours after giving ibuprofen at a dose of 400 mg (21).

Mechanical allodynia is seen frequently with irreversible pulpitis and can be a factor in increased patient pain (13). Several theories have been presented for apical mechanical allodynia, including pulpal mechanoreceptive neurons, inflammatory mediators/bacterial byproducts, and even central sensitization (13). Because there is a subpopulation with continued pain after endodontic treatment (22), more research in this area would assist in the treatment of mechanical allodynia.

In this clinical trial, it appears that ibuprofen has a greater ability to mask the results of diagnostic tests in patients with inflamed vital pulps. This could be explained by the ability of NSAIDs to be preferentially distributed into inflamed pulps (23). NSAIDs have the ability to significantly suppress local production of prostaglandins to provide an analgesic effect (24). Moreover, there is a 100-fold greater level of prostaglandins in an irreversibly inflamed pulp, such as one with a diagnosis of SIRP/SAP, than there is in a normal control tooth pulp (25). This reduction in peripheral inflammatory mediators may produce a masking effect in endodontic diagnostic testing.

The bite force transducer has exciting research potential and may be refined to improve endodontic diagnosis for teeth with a question-

able diagnosis. Competent clinicians develop an accurate diagnosis with a variety of endodontic diagnostic tests using a system of checks and balances. If this information does not coincide, then patients are encouraged to return to the dental office when their symptoms resume. Because results from common endodontic testing can be masked by a high, single dose of ibuprofen, patients should be advised not to take ibuprofen the day of their dental visit to aid in the proper diagnosis.

Acknowledgments

The authors deny any conflicts of interest related to this study.

References

- Rowe AH, Pitt Ford TR. The assessment of pulpal vitality. *Int Endod J* 1990;23:77–83.
- Walton RE, Torabinejad M. *Principles and Practice of Endodontics*, 4th ed. St Louis, MO: WB Saunders; 2009.
- Mumford JM. Pain perception threshold and adaptation of normal human teeth. *Arch Oral Biol* 1963;8:493–501.
- Mumford JM. Thermal and electrical stimulation of teeth in the diagnosis of pulpal and periapical disease. *Proc R Soc Med* 1967;60:197–200.
- Gallatin E, Reader A, Nist R, Beck M. Pain reduction in untreated irreversible pulpitis using an intraosseous injection of Depo-Medrol. *J Endod* 2000;26:633–8.
- Stoller EP, Gilbert GH, Pyle MA, Duncan RP. Coping with tooth pain: a qualitative study of lay management strategies and professional consultation. *Spec Care Dentist* 2001;21:208–15.
- Nusstein JM, Beck M. Comparison of preoperative pain and medication use in emergency patients presenting with irreversible pulpitis or teeth with necrotic pulps. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2003;96:207–14.
- Mickel AK, Wright AP, Chogle S, et al. An analysis of current analgesic preferences for endodontic pain management. *J Endod* 2006;32:1146–54.
- Hargreaves KM, Cohen S. *Pathways of the Pulp*, 9th ed. St Louis, MO: Mosby; 2006.
- Khan AA, Hargreaves KM. Pharmacologic control of dental pain. In: Hargreaves KM, Goodis HE, Tay FR, eds. *Seltzer and Bender's Dental Pulp*, 2nd ed. Chicago: Quintessence Publishing; 2012:190–200.
- Gage TW, Pickett F, eds. *Mosby's Dental Drug Reference*, 7th ed. St Louis, MO: Mosby; 2005.
- Oxford league table of analgesics in acute pain. Bandolier: “Evidence based thinking about health care.” Available at: <http://www.medicine.ox.ac.uk/bandolier/booth/painpag/acutrev/analgesics/leagtab.html>. Accessed July 18, 2012.
- Owatz CB, Khan AA, Schindler WG, et al. The incidence of mechanical allodynia in patients with irreversible pulpitis. *J Endod* 2007;33:552–6.
- Khan AA, McCreary B, Owatz CB, et al. The development of a diagnostic instrument for the measurement of mechanical allodynia. *J Endod* 2007;33:663–6.
- Newton CW, Hoen MM, Goodis HE, et al. Identify and determine the metrics, hierarchy, and predictive value of all the parameters and/or methods used during endodontic diagnosis. *J Endod* 2009;35:1635–44.
- McCarthy PJ, McClanahan S, Hodges J, Bowles WR. Frequency of localization of the painful tooth by patients presenting for an endodontic emergency. *J Endod* 2010;36:801–5.
- Jones DM. Effect of the type carrier used on the results of dichlorodifluoromethane application to teeth. *J Endod* 1999;25:692–4.
- Khan AA, Owatz CB, Schindler WG, et al. Measurement of mechanical allodynia and local anesthetic efficacy in patients with irreversible pulpitis and acute periradicular periodontitis. *J Endod* 2007;33:796–9.
- Kardelis AC, Meinberg TA, Sulte HR, et al. Effect of narcotic pain reliever on pulp tests in women. *J Endod* 2002;28:537–9.
- Carnes PL, Cook B, Eleazer PD, Scheetz JP. Change in pain threshold by meperidine, naproxen sodium, and acetaminophen as determined by electric pulp testing. *Anesth Prog* 1998;45:139–42.
- Norhold SE, Aagaard E, Svensson P, Sindet-Pedersen S. Evaluation of trismus, bite force, and pressure algometry after third molar surgery: a placebo-controlled study of ibuprofen. *J Oral Maxillofac Surg* 1998;56:420–7.
- Nixdorf DR, Moana-Filho EJ, Law AS, et al. Frequency of persistent tooth pain after root canal therapy: a systematic review and meta-analysis. *J Endod* 2010;36:224–30.
- Bunczak-Reeh MA, Hargreaves KM. Effect of inflammation on the delivery of drugs to dental pulp. *J Endod* 1998;24:822–5.
- Roszkowski MT, Swift JQ, Hargreaves KM. Effect of NSAID administration on tissue levels of immunoreactive prostaglandin E2, leukotriene B4, and (S)-flurbiprofen following extraction of impacted third molars. *Pain* 1997;73:339–45.
- Nakanishi T, Matsuo T, Ebisu S. Quantitative analysis of immunoglobulins and inflammatory factors in human pulpal blood from exposed pulps. *J Endod* 1995;21:131–6.