A Review of Paresthesia in Association with Administration of Local Anesthesia

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Local anesthetics allow dentistry to be practiced without patient discomfort. Serious complications associated with the use of these drugs are rare. The occurrence of paresthesia following the use of local anesthesia in dentistry, however, represents an important side effect. This article examines the occurrences of paresthesia to the lingual and inferior alveolar nerve resulting from the injection of local anesthetic agents. Paresthesia can be defined as persistent anesthesia (anesthesia well beyond the expected duration),[1] or altered sensation (tingling or itching) well beyond the expected duration of anesthesia.[1-4] Tingling and itching are included as part of the definition as these sensations are considered partial anesthesia to the dentist and patient. In general, most dentists and patients would define paresthesia as a prolonged numbness. The definition of paresthesia also includes hyperesthesia and dysesthesia[1-3] in which the patient experiences both pain and numbness. Dysesthesia is defined as, painful sensation to nonnoxious stimuli and hyperesthesia as, increased sensitivity to noxious stimuli.[2] Paresthesia can also be associated with a burning sensation, and patients can experience drooling, speech impediment, loss of taste, and tongue biting.[4] Local anesthetic-induced lingual nerve and/or inferior alveolar nerve paresthesia is generally considered a rare occurrence.[5,6] The occurrence of paresthesia in the United States has been studied, and it was found that 51% of paresthesias were related to lidocaine, 51% to prilocaine, and 8% to mepivacaine (these data were obtained prior to the introduction of articaine in the US). The total number is above 100% because twelve of the eighty-three patients studied received more than one local anesthetic agent. Estimates of the use of these local anesthetic agents in the US were 62% lidocaine, 23% mepivacaine, and 13% prilocaine. Symptoms are most commonly associated with mechanical trauma during surgical procedures.[5-7] During the administration of local anesthesia prior to treatment of mandibular teeth or their associated structures, the lingual or inferior alveolar neurovascular bundle can be traumatized by the sharp needle-tip, the movement of the needle, extraneural or intraneural hemorrhage from trauma to the blood vessels, or from neurotoxic effects of the local anesthetic.[4,6-8] The primary factor in neurotoxicity of local anesthetics appears to be the concentration of the solution.[4,9] Paresthesia may occur if, during injection, the patient complains of a sensation described as electric shock along the path of the nerve that is contacted by the needle.[1-4,6-8] This article is a review of published data about the incidence of paresthesia induced by the administration of local anesthetic, which is not related to surgical trauma.

Figure 1. The pattern of the numbers of reported cases of paresthesia in Ontario, Canada. * *Reproduced with permission of the publisher from Figure 1: The pattern of the numbers of reported cases of paresthesia in Ontario, Canada, by Haas and Lennon. [4]

LITERATURE REVIEW AND ANALYSIS

Because of voluntary medical-legal reporting of the occurrence of paresthesia in Ontario, Canada, and the publication of these data by Haas, Miller, and Lennon, information has been collected regarding the occurrence of paresthesia following administration of mandibular local anesthesia, which is not related to surgical trauma.[4,10,11] Data from three reports indicate that articaine and prilocaine show a very high rate of mandibular paresthesia (usually of the lingual nerve), while lidocaine and mepivacaine were rarely associated with this untoward side effect.[4,10,11]

In viewing the pattern of dental injection-related paresthesia in Ontario, Canada from 1973 to 1993 (Figure 1), there is an abrupt change in frequency in 1985, the year after articaine was available in Canada. Lidocaine was introduced in 1948, mepivacaine in 1960, prilocaine in 1978, and bupivacaine in 1982. There were twenty-one reported cases of paresthesia in the 11 years prior to the introduction of articaine, and never more than three cases per year. There were 123 reported cases of paresthesia in the 10-year period after articaine was introduced, with never fewer than five cases per year. Table 1 lists the location and symptoms associated with the 143 cases of nonsurgical paresthesia shown in Figure 1.

The different frequency of lingual nerve versus inferior alveolar nerve paresthesia is interesting. Why would there be twice the number of lingual nerve paresthesias (101) as compared to the number of inferior alveolar nerve paresthesias (51)? This is notable considering how most practitioners administer local anesthesia to the mandible (one fourth cartridge at the lingual nerve site and the remainder at the inferior alveolar nerve site). Also, inferior alveolar anesthesia is ineffective more often than lingual nerve anesthesia, and requires subsequent injection. If the quantity of local anesthetic were the primary causative factor, it would be expected that the inferior alveolar nerve would be affected more often.

A possible reason for the increased paresthesia to the lingual nerve is that in performing inferior alveolar injections some practitioners change direction of the needle at the approximate depth of the lingual nerve. The sharp needle tip may lacerate the lingual nerve and/or artery on the initial or subsequent path. Another possible reason is in a performing subsequent injection to anesthetize the inferior alveolar nerve the needle may traumatize the more superficial lingual nerve but without the electric shock sensation, because the nerve is usually anesthetized on the initial attempt. The cause of the paresthesia may also be a combination of neurotoxicity of the local anesthetic[9] and trauma to the nerve.

It has also been theorized that pressure to the nerve from intraneural blood vessel hemorrhage[3,4,6,7] may cause damage to the nerve, with altered neurologic function being the result. It is currently believed that the least plausible cause of paresthesia is the needle actually severing the nerve, considering the relative size of the neurovascular bundle.[6,7] It is important to note that another study reporting data from the United States that was published prior to the introduction of articaine also found approximately twice the occurrence of lingual nerve paresthesia versus inferior alveolar nerve paresthesia (sixty-six cases involving the lingual nerve and twenty-seven cases involving the inferior alveolar nerve).[7]

There are a number of important consequences of lingual nerve paresthesia (see Table 1). Of the 101 reported cases of lingual nerve paresthesia, four patients experienced drooling, eight tongue biting, nine speech impediment, thirteen burning, and twenty-five loss of taste besides the general sense of the tongue feeling numb.

Table 2 lists the frequency a specific local anesthetic was used when a paresthesia was reported. Table 2 covers the 1973 to 1993 time period, and articaine was not introduced in Canada until 1984; therefore, it was available for about half of the study interval. Articaine was associated with the paresthesia 49% of the time, prilocaine 42%, lidocaine 5%, and mepivacaine 4%.

The disparity in the number of cases between Table 1 (143 cases), and Table 2 (149 times in which anesthetics were associated with paresthesia), is due to the fact that in six occurrences two different local anesthetics were used. It is not known if some of the cases associated with lidocaine and mepivacaine occurred when they were used in combination with articaine or prilocaine.

The information for 1993 alone gives a detailed accounting of the association of anesthetic usage and paresthesia (see Table 3). The authors were able to quantify local anesthetic usage by surveying Ontario dentists. The data from the 1993 study were subsequently confirmed by a study of data collected in 1994[10] and data from 1994 to 1998.[11] The authors estimated that the incidence of nonsurgical paresthesia was one in 785,000 injections (Table 3). However, it is clear from the number of cases listed and the number of cartridges used that articaine and prilocaine have precipitously higher rates of paresthesia. Analysis of the data allows the paresthesia rates for each local anesthetic to be calculated (Table 4).

Because all reported paresthesias were of the lingual nerve, inferior alveolar nerve, or both, maxillary injections are not considered in this analysis. The assumption is that half of all injections are for the maxillary arch,6 and the total number of cartridges was divided in half.

Since there were no paresthesias reported in 1993 with the use of any local anesthetics except articaine and prilocaine, a frequency of paresthesia could not be calculated for lidocaine, mepivacaine, and bupivacaine using that data. To estimate the frequency of paresthesia for these local anesthetics, the data from Tables 2 and 4 were used with two assumptions:

Assumption No. 1. Lidocaine and mepivacaine had the same number of cartridges used for each of the prior 21 years (1973 to 1993) as in the 1993 data. This is most likely a conservative estimate for these drugs, since during certain years the other local anesthetics were not available in Canada: articaine was not available from 1973 to 1983, bupivacaine from 1973 to 1981, and prilocaine from 1973 to 1977. Similarly, for bupivacaine the same number of cartridges were used each year from 1982 to 1993 as in 1993. This is most likely too high a number because one would expect that sales would increase the longer an agent has been on the market, and no other local anesthetics with similar clinical properties were available.

Assumption No. 2. When the anesthetic agent associated with paresthesia was not known (see Table 2), the distribution was the same as when the anesthetic agent was known.

For example, using these assumptions, the number of cartridges of lidocaine used on mandibular injections in 1993 (from Table 4) multiplied by 21 years equals 32,157,447 cartridges used for mandibular injections. For the number of cases of paresthesia, use the number of known cases of paresthesia from Table 2 (5). Add the ratio of known cases of paresthesia (5/102), multiplied by the number of cases of unknown paresthesia (47). This equals 2.3. The number of paresthesias would then be 5 known + 2.3 assumed = 7.3. The expected frequency of nonsurgical mandibular paresthesia with lidocaine would then be estimated as 7.3 per 32,157,447 cartridges, or 1:4,405,130. The calculated data is provided in Table 5.

By this analysis there is a twenty time greater likelihood of paresthesia resulting from a mandibular block injection with articaine as compared to lidocaine. Prilocaine is calculated to have a fifteen times higher rate of mandibular paresthesia than lidocaine. Why would articaine and prilocaine have paresthesia rates far greater than lidocaine or mepivacaine? All are amide local anesthetics. The most apparent difference between the local anesthetics is the concentration of the anesthetic. These concentrations are 0.5% bupivacaine, 2% lidocaine, 2% mepivacaine, 3% mepivacaine, 4% articaine, and 4% prilocaine.

It has been determined that local anesthetic-induced nerve injury is concentration-dependent, with injuries increasing as concentration increases. The highest concentrations of local anesthetics were associated with endoneurial edema.

Analysis of the data indicates that articaine has a 4% higher occurrence of paresthesia than prilocaine, though they are both 4% solutions. Although they may not be causative factors, articaine is the only local anesthetic with sulfur or a thiophene ring and an ester bond.[1] It is also the only local anesthetic having both an ester and amide bond. The relationship of chemical structure to the occurence of paresthesia should be investigated. A number of recent publications suggest that articaine is associated with a higher rate of paresthesia for mandibular block injections than was calculated in this paper. While the Canadian data were for voluntary reporting of paresthesia, recent data reported all occurrences. Two cases of paresthesia were reported following treatment of 13,000 patients with articaine.[12] If half of these were for mandibular procedures, the rate would be two cases for 6,500 mandibular injections, or 1:3,250. Considering that infiltration anesthesia could also have been employed for the mandible, the paresthesia rate may have even been higher. The product insert for articaine, and the publications associated with the study of articaine reported to the Food and Drug Administration (FDA), indicate a paresthesia rate much higher than one in 3,250 usages.[13-16] The FDA study reported twenty-one cases of paresthesia in 882 patient treatments, or one in every fourty-two patient treatments. The product insert[14] and one publication16 indicate a 1% paresthesia rate. Another publication[15] lists paresthesia as one of the minor adverse events found in the study.

CONCLUSIONS

Because paresthesia associated with the use of local anesthetics as part of dental care in the United States has been an infrequent event, many dentists and patients are not aware of the potential problem. Besides the range of altered sensations considered as paresthesia (perceived numbness, swelling, tingling, itching), there can be oral dysfunction4 and pain.[4,7,17] The dysfunctions include tongue biting, drooling, loss of taste, and speech impediment.[4] Pain (dysesthesia or hyperesthesia) is usually not considered when discussing local anesthesia-induced paresthesia. The pain of dysesthesia can severely impact the quality of life,[7,17] causing some patients to seek treatment at pain management clinics.[6,7,17] The life changes patients experience from these alterations are significant.[6,7,17] Minimizing the chance of paresthesia is the best approach. The analysis of data from the study in Canada presented here indicates that 4% articaine has a twenty-fold higher rate of paresthesia compared to 2% lidocaine. Data from more recent studies suggest an even greater disparity in this untoward side effect when comparing articaine and lidocaine.[12-16] These data suggest a rate of paresthesia (using articaine for lingual or mandibular block injections) as high as 2% to 4%. As evidenced by the Canadian data from 1993, there were no reported cases of paresthesia with the use of more than 3 million cartridges of lidocaine. Furthermore, there were only five confirmed cases of paresthesia with lidocaine in 21 years. One study of permanent nerve injuries associated with inferior alveolar nerve blocks found the same number of injuries associated with lidocaine (41) as with prilocaine (41). However, national sales figures indicate that lidocaine was used 4.7 times more than prilocaine.[7] It should be noted that this study[7] was conducted prior to the FDA approval of articaine in the United States.

The paresthesia rates listed for articaine, and to a lesser extent prilocaine, raise questions regarding the use of these local anesthetics:

(1) Does the risk of paresthesia warrant use of articaine and prilocaine for lingual nerve, inferior alveolar nerve, and other mandibular block injections?
(2) Does the risk of paresthesia with articaine and prilocaine for mandibular block or lingual block injections warrant the use of special informed consent? Based on this analysis of data it appears informed consent is merited in performing mandibular block and lingual block injections with articaine or prilocaine. The risk of paresthesia is so remote in all other administrations of local anesthesia that consent seems unnecessary. Previously, the medicolegal environment has not considered the issue in this context. In 1989, the Ontario High Court[18] ruled that informed consent was not necessary before administering local anesthesia because the expert witnesses said the risk was infinitesimal, minimal, extremely small, or in order of magnitude of 1:800,000. Based on the Canadian study,[4] the rate of paresthesia in Ontario for 1993 was estimated to be 1:785,000. Although this was true, ten of the fourteen cases were associated with the use of articaine in mandibular block injections and the other four were from the use of prilocaine with mandibular block injections.
(3) The Canadian data do not provide information regarding the duration of paresthesia in the cases reported. This would certainly be one important consideration when deciding on the use of these agents for local anesthesia. Analysis of the available data indicates that there are areas for future studies, and more specific data needs to be collected. For example, improved clinical monitoring/recording of extent, degree, and duration of paresthesia; clear definition of terms; the specific type of injections administered, and; differentiating paresthesia from minor adverse events related to administration of anesthesia. These data would improve our understanding of the problem. In summary, the incidence of paresthesia of the lingual nerve and inferior alveolar nerve should be considered when selecting a local anesthetic agent for anesthetizing the mandible and its associated structures.

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Dr. Dower is an associate professor in the Department of Restorative Dentistry at University of the Pacific's School of Dentistry. He has been director of the Local Anesthesia courses since 1979 and implemented a local anesthesia curriculum at UOP that includes three rotations in local anesthesia during students clinical years. Dr. Dower has presented local anesthesia courses, lectures, and table clinics to regional, state, and national dental meetings. His publications and research articles have appeared in *Anesthesia Progress, Journal of the American Dental Association, Quintessence International, Journal of General Dentistry,* and *Journal of the California Dental Association*. Dr. Dower also holds a master's degree in Educational and Counseling Psychology.