A Clinical Study of the Effectiveness and Possible Mechanism of Action of the Periodontal Ligament Injection in attaining Anesthesia for Exodontia

by

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Abstract

The effectiveness and possible mechanism of action of the periodontal ligament injection in producing anesthesia were evaluated, in a clinical trial, by three criteria.

Periodontal ligament injections were performed under strong pressure on fourteen teeth with lidocaine, seven teeth with epinephrine and seven teeth with normal saline. The anesthesia attained was evaluated by; response of the teeth to the electric pulp tester, gingival anesthesia around the teeth and ability to extract the teeth without pain.

The results demonstrated that periodontal ligament injections with lidocaine are effective in producing anesthesia. Lidocaine periodontal ligament injections were statistically and clinically more effective in attaining anesthesia than epinephrine or saline injections. Epinephrine periodontal ligament injections significantly decreased sensory nerve activity in the teeth while saline injections produced no anesthesia. It appears, based on the results of the saline injections, that hydrostatic pressure has no direct effect in producing anesthesia with the periodontal ligament injection.

Résumé

L'efficacité et le principe d'action de l'anesthésie par injection du ligament péridentaire ont été évalués cliniquement, selon trois critères; réponse des dents à la stimulation du vitalomètre, insensibilité de la gencive autour des dents et l'extraction des dents sans douleur. Quatorze dents ont reçu des injections intra ligamentaire de lidocaine, sept dents ont reçu de l'épinéphrine et sept dents de la solution saline.

Les résultats ont démontré que les injections du ligament péridentaire avec de la lidocaine produisent une anesthésie adéquate. Les injections de lidocaine furent statistiquement et cliniquement plus efficaces en terme d'anesthésie que l'épinéphrine ou les injections salines. Les injections ligamentaires d'épinéphrine diminuèrent sensiblement l'activité du nerf sensitif, tandis que les injections salines ne produirent pas d'anesthésie. En se basant sur les résultats des injections salines, il ressort que la pression hydrostatique n'a pas d'effet direct sur l'anesthésie du ligament péridentaire.

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Introduction

Standard nerve block or infiltration injection techniques, given for dental procedures, may not result in adequate anesthesia. If repeating the same injection does not produce the desired level of anesthesia, a supplemental injection such as an intrapulpal, intraosseous, or periodontal ligament injection is then indicated to obtain complete anesthesia.[43]

In the continuing search for improved techniques for dental anesthesia there has been a recent growth in interest, within the dental profession, with respect to the periodontal ligament injection.[24,31] The periodontal ligament injection is a method of administering a local anesthetic agent directly into the periodontal ligament of the tooth to be anesthetized and has been employed for many years using standard dental syringes.[25,43] Recently syringes which can deliver the anesthetic solution into the periodontal ligament under great pressure have been developed.[31] The injection attempts to deliver the local anesthetic agent to the apical foramen of the tooth.[12]

Studies have shown that the periodontal ligament injection is successful in producing anesthesia for surgical, periodontal, restorative and endodontic procedures 80% to 90% of the time.[12,14,15,24,31,41,43] Strong pressure on injection was significantly related to a higher incidence of anesthesia as compared to injecting under moderate or slight pressure.[41,43] The pressure is created within the periodontal structures by the force applied to the syringe and the tissue resistance.[35]

In 1983, the American Dental Association council on dental materials, instruments and equipment stated that the actual mechanism of action of the periodontal ligament injection has yet to be determined.[19] It has been demonstrated that when using the periodontal ligament injection there is a rapid onset of anesthesia.[12,24,39,43] This could indicate that the anesthetic agent has rapid distribution and contact with sensory nerves supplying the pulp. Another factor in the rapid onset of anesthesia may be that the high hydrostatic pressure created by this injection affects the sensory nerves and reduces or prevents nerve conduction.[39]

A clinical study on the intrapulpal injection showed that the pressure created during an intrapulpal injection was the most critical factor in attaining anesthesia.[6] Another clinical study by Pashley et al[35] on the pressures created by dental injections showed that the mean tissue fluid pressure created during a periodontal ligament injection was 17,630 mm Hg while the mean tissue fluid pressure created during an intrapulpal injection was only 8,918 mm Hg. It has been demonstrated that a tissue fluid pressure of 50 mm Hg or greater will completely block sensory and motor nerve responses.[18,22,29] The pressure may inhibit nerve conduction by a direct effect on the nerve or secondarily by inhibiting blood flow to the nerve and causing ischemia of the nerve.[5,11,16,23,29,36,37] The hydrostatic pressure created with the periodontal ligament injection is much greater than 50 mm Hg.[35] Therefore, it may be that the pressure created within the periodontal ligament, by the injection, may be responsible for the anesthetic effect.

It has been shown by Olgart and Gazelius[34] that epinephrine injected supraperiosteally causes a decrease in pulpal blood flow and inhibition of sensory nerve activity of the adjacent tooth. The epinephrine decreases or prevents local microcirculation at the tooth apex. Therefore, it may be

that the decrease in sensory nerve conduction after the periodontal ligament injection is due to ischemia of the nerves secondary to epinephrine.

Therefore, it would appear that there are four possible mechanisms of action of the periodontal ligament injection in producing dental anesthesia. They are; the action of the anesthetic agent, the action of the vasoconstrictor, the effect of hydrostatic pressure or a combination of these factors. The purpose of this study was to evaluate in a clinical trial the effectiveness and possible mechanism of action of the periodontal ligament injection in attaining anesthesia for routine dental extractions.

Review of Literature

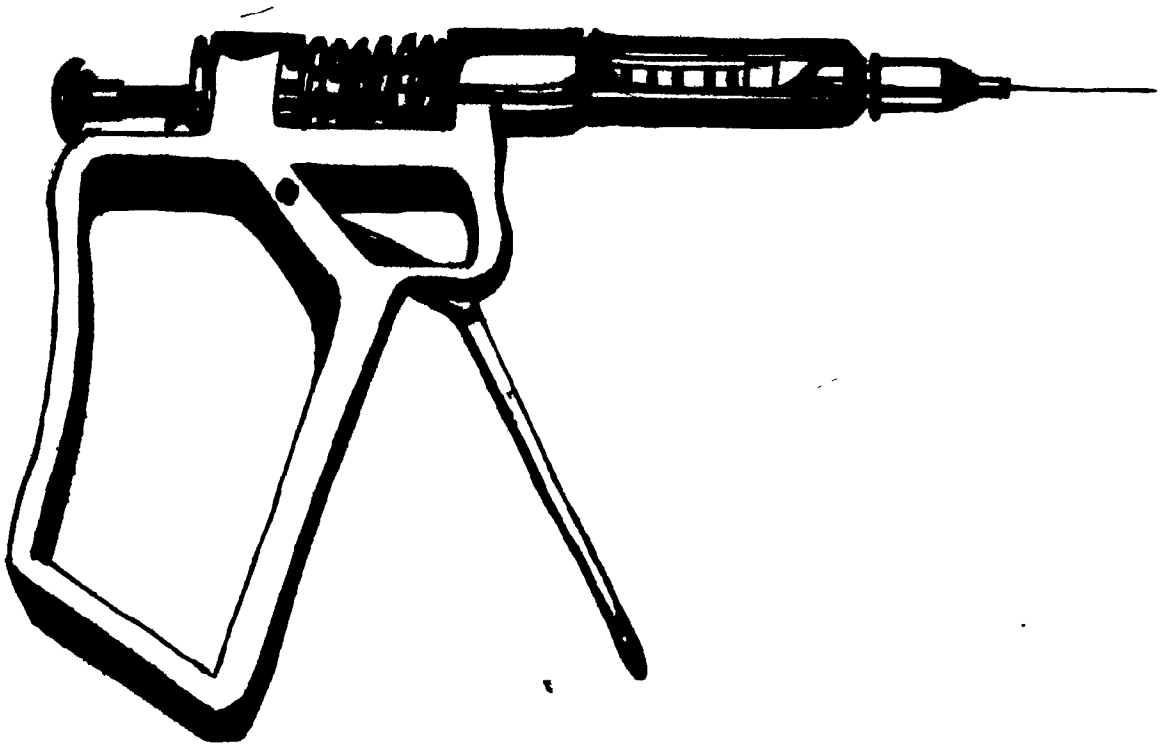
Recently there have been advances in local anesthetic technique and armamentarium which may significantly affect the practice of dental anesthesia in the future.[24,31] One of the new techniques for producing dental anesthesia is the periodontal ligament injection using either the Peri-Press* or the Ligmaject** syringes. The concept of injecting a local anesthetic solution into the periodontal ligament to produce anesthesia of a single tooth is not new. It has been referred to in the literature as the periodontal ligament injection, intraperiodontal injection or intraligamentary injection. However, until recently this procedure employed a standard dental syringe and was used primarily as a means of achieving complete anesthesia of a tooth where infiltration or block anesthesia had previously failed.[31]

The Peri-Press and Ligmaject syringes for use in the periodontal ligament injection were introduced to North America in the late 1970's.[31] These syringes are "pistol-type" devices (Figure 1) which employ a drive mechanism which facilitates the injection of anesthetic solution into the periodontal ligament under great pressure.[10] Since the introduction of these devices it has been claimed that the periodontal ligament injection could be administered as the only injection necessary for attaining profound anesthesia for a dental procedure on a single tooth.[12,31] The periodontal ligament injection would thus eliminate the need for regional nerve blocks or infiltrations when treating an individual tooth.

*Universal Dental Implements, P.O. Box 254, Fanwood, N.J.

**I.M.A. Associates, U.S., Inc. 270 South Harvard Blvd., Los Angeles, Calif.

Figure 1. Ligmaject "pistol-type" syringe



Archer[1] and Wall[42] reported that there was actually a pressure syringe designed by Gunthorpe in 1912. Wall[42] states "A high pressure syringe was designed that used a needle within a needle concept for the administration of local anesthesia in dentistry. By squeezing on a plier mechanism and forcing the plunger forward under greater pressure than could be produced by the standard dental syringe, a considerable amount of pressure was exerted on the solution in the steel barreled syringe. The Gunthorpe syringe was probably not accepted because of the skill necessary to master the high pressure, local anesthetic injection technique." From 1912 until the introduction of the Peri-Press and Ligmaject syringes in the late 1970's no other high pressure syringes were available.

Walton and Abbott[43] define the periodontal ligament injection as "the administration of the local anesthetic agent directly into the periodontium adjacent to the tooth." The periodontal ligament provides a potential pathway to the apical foramen of each tooth by which it should be possible to deliver a local anesthetic solution and thereby produce anesthesia. At the level of the alveolar crest the width of the periodontal ligament space has been determined to be in the range of 0.10 mm to 0.35 mm depending on the location of the tooth and the occlusal forces placed on it.[12] The periodontal ligament injection technique takes advantage of the average width of the periodontal ligament space at the alveolar crest in that a 25 to 30 gauge needle can be inserted into this space.[31,41,43]

The technique for injection into the periodontal ligament is quite simple. The injection can be made at any point around the tooth but is most often described as being made at either the mesiobuccal, distobuccal, mesiolingual and/or distolingual aspects of the tooth.[24] The needle is

inserted into the periodontal ligament via the gingival sulcus at a 30 degree angle to the long axis of the tooth with the bevel facing away from the tooth. After placement the needle is forced to maximum penetration so that it is wedged between the root of the tooth and the alveolar bone.[12,19,20,24,25,31,41,43]

Following insertion of the needle the injection is performed slowly, requiring approximately 20 seconds for completion at each site.[12,19,20,25] If needle position is correct, initially there is a strong resistance to the influx of the anesthetic solution by the periodontal ligament. This resistance lasts approximately 6 seconds after which the anesthetic solution begins to flow easily.[12] Each injection should deliver approximately 0.2 cc* of anesthetic solution into the periodontal ligament.[19,20,24,25,31]

If at any stage during the injection there is a rapid return of anesthetic solution from the gingival sulcus then the needle has not been correctly placed. In this case the injection should be stopped and the needle reinserted in a new area so that proper needle placement is accomplished.[12,31] During the injection one must be careful not to exert too much pressure too rapidly as this may result in intense pressure pain for the patient.[12]

The needle is then removed and profound anesthesia of the tooth and gingiva where the injection occured should be attained within 30 seconds and last for 45 to 55 minutes.[10,19] For restorative and endodontic dental procedures it is usually sufficient to perform one injection per root.[19] When periodontal or surgical procedures are to be performed it is necessary to inject on both the buccal and lingual aspects of the tooth to be anesthetized.[31]

* 1.0 cc equals 1.0 ml.

Currently there are two instruments marketed which are specifically designed for the periodontal ligament injection. They are the Peri-Press and the Ligmaject syringes.[10,14,20,31] Some of the advantages of these "pistol-type" syringes compared to the standard dental syringe are the following. Due to their special design the "pistol-type" syringes deliver a measured amount, of approximately 0.2 cc of anesthetic solution, per injection.[19,20,24,25,31] The Peri-Press and Ligmaject syringes are designed with a special handle which allows the periodontal ligament injection to be given under high pressure without discomfort to the operators hand. The pressure syringes are also designed so that the glass anesthetic cartridge is covered and therefore the patient and operator are protected in the event that the glass cartridge breaks upon injection.[10]

The Peri-Press and Ligmaject syringes also have disadvantages compared to the standard dental syringes. The "pistol-type" syringes are larger, bulkier and are shaped like a gun. These factors could conceivably increase patient anxiety.[10,31] Malamed[31] felt that proper preinjection discussion with the apprehensive patient helped to minimize this problem. The "pistol-type" syringes also cost more than the standard dental syringe. Due to the increased pressures that can be generated with the Peri-Press and Ligmaject syringes there is a greater chance of breakage of the glass anesthetic cartridge if the anesthetic solution is administered too rapidly.[10]

Direct comparisons between the Peri-Press and Ligmaject syringes show small differences in design of the instruments. Both instruments incorporate a system to protect the patient in case of breakage of the anesthetic cartridge. In the Ligmaject this consists of a removable transparent plastic sheath which covers the anesthetic cartridge and in the Peri-Press syringe it is a solid metal barrel into which the cartridge is

placed. With the Ligmaject syringe the anesthetic cartridge can be observed by the operator during injection. However, the metal barrel holding the anesthetic cartridge is windowless with the Peri-Press syringe and therefore the operator cannot see the position of the plunger during injection. With the Ligmaject syringe the direct drive mechanism relies on a coiled spring while the ratchet principle is used for the drive mechanism in the Peri-Press syringe. Overall, the Ligmaject and Peri-Press syringes have been rated to be equally effective in administering the periodontal ligament injection.[10]

Morse,[32] in 1974, suggested that the periodontal ligament injection technique may help in attaining anesthesia in difficult endodontic treatment situations. However, no evidence of the effectiveness of the periodontal ligament injection was shown until 1981.[43] At that time, Walton and Abbott[43] reported that using the periodontal ligament injection they attained successful anesthesia in 92% of their cases. In this study they were using a standard dental syringe to administer the periodontal ligament injection in patients who had inadequate pulpal anesthesia following conventional injections for routine endodontic therapy. These investigators found that after the first periodontal ligament injection their success in attaining anesthesia was 63% but after doing a second periodontal ligament injection on the same tooth their overall success rate in attaining anesthesia was 92%.

In 1983 Smith, Walton and Abbott[41] published a study evaluating the clinical effectiveness of the periodontal ligament injection using the "pistol-type" syringe. In this study they performed periodontal ligament injections on patients who were undergoing routine endodontic procedures but had inadequate pulpal anesthesia after a conventional nerve block or

infiltration injection. Their success rate after the first periodontal ligament injection was 65%. After reinjection they had an overall success rate, in attaining anesthesia with the "pistol-type" syringe, of 83%. Comparing the results of these two studies the authors concluded that the periodontal ligament injection, using either the standard dental syringe or the "pistol-type" syringe, was effective as a supplemental injection in attaining anesthesia for endodontic procedures.[41,43]

Malamed[31] reported the findings of a clinical study on the effectiveness of the periodontal ligament injection in achieving clinically adequate anesthesia for a variety of dental procedures. He performed the periodontal ligament injections with the "pistol-type" syringes and the standard dental syringe. Malamed[31] found an overall clinical effectiveness of 88.5% with the "pistol-type" syringes and 82% with the standard dental syringe.

A clinical study of the effectiveness of the periodontal ligament injection using the Peri-Press syringe was performed by Kaufman et al.[24] They used the periodontal ligament injection as the primary means of attaining anesthesia for a variety of dental procedures. In their study successful anesthesia was attained in 84% of the teeth treated.

Another study by Faulkner[15] using the "pistol-type" syringe for the periodontal ligament injection produced adequate anesthesia in 81% of the patients treated. A variety of dental procedures had been performed in this study.

These latter two studies are the only investigations which include a large number of extractions among the dental procedures performed using the periodontal ligament injection.[15,24] Kaufman et al[24] and Faulkner[15] reported successful anesthesia for exodontia in 95% of cases.

These studies all reported that, for the periodontal ligament injection

to be effective in producing profound anesthesia, the injection had to be given under strong pressure.[15,24,31,41,43] Smith, Walton and Abbott[41] and Walton and Abbott[43] stated the most critical factor in obtaining success with the periodontal ligament injection is to inject under strong pressure with firm resistance. Their clinical trials showed that injecting without strong resistance and back pressure resulted in the periodontal ligament injection not being very effective in attaining anesthesia for the dental procedure. Without the presence of strong back pressure on injection the success rate of the periodontal ligament injection in attaining anesthesia was only 30% using the "pistol-type" syringe[41] and 32% using the standard dental syringe.[43]

The periodontal ligament injection, as with any dental procedure, has its advantages and disadvantages. The advantages of this technique are the following. The periodontal ligament injection allows single tooth anesthesia and avoids numbness to the lip, tongue and other soft tissues.[10,19,20] This thereby facilitates treatment in different quadrants during the same appointment.[10,19] The patient leaves the dental office without a numb lip or tongue which could accidently be chewed upon and traumatized.[25,31] This is especially important in young children and mentally or physically handicapped patients.[31] Finally, disruption of speech and mastication, due to anesthesia of the lip and tongue, is avoided.[25]

The periodontal ligament injection can be used as a supplemental injection whenever anesthesia is difficult to obtain with conventional nerve blocks and infiltrations.[10,19,20] It can also be used when conventional techniques are contraindicated such as in patients with hemophilia or other bleeding disorders in whom the risk of postinjection bleeding may be

dangerous.[7,10,31] Bishop[7] used the periodontal ligament injection 18 times in 10 hemophiliac patients who had received no factor replacement. She reported that the hematologist was satisfied that there was no evidence of hematoma formation.

The periodontal ligament injection is less painful for most patients than conventional techniques if the injection is administered correctly.[10,12,19,20,21,31] Malamed[31] observed that many patients in his study commented on the lack of discomfort with the injection.

The periodontal ligament injection produces profound anesthesia within 30 seconds and the anesthesia lasts 45 to 55 minutes.[10,19] This is an improvement over conventional nerve blocks or infiltrations which normally require a 5 to 10 minute waiting period for profound anesthesia to occur.[25]

Local complications of mandibular block injections such as parathesia, facial nerve paralysis, hematoma formation, trismus, among others are eliminated when periodontal ligament injections are performed.[25] The periodontal ligament injection also avoids the hazards of toxicity when administering large volumes of local anesthetics or vasoconstrictors.[19,25] With each periodontal ligament injection, when using the "pistol-type" syringe, a controlled metered dose of approximately 0.2 cc of anesthetic solution is delivered into the periodontal ligament. This may be important in patients with systemic health problems where minimizing the total dose of anesthetic solution or vasoconstrictor is of concern.[10,19] The potential for allergic reactions is also greatly reduced due to the smaller quantity of anesthetic administered.[12] The periodontal ligament injection avoids direct injection of anesthetic solution into areas of large vessels[10] and Malamed[31] reported a positive aspiration rate of 0 percent.

The periodontal ligament injection can also be used as an aid in diagnosis of pain of odontogenic origin. By systematically anesthetizing individual teeth with the periodontal ligament injection it is possible to identify a specific tooth which is causing pain.[9,25,38]

It is well known that teeth with irreversible pulpitis can be extremely difficult to anesthetize with nerve blocks or infiltrations.[25] Khedari[25] states that an advantage of the periodontal ligament injection is that it is effective in attaining anesthesia of teeth with irreversible pulpitis. He feels that pulpal anesthesia occurs after the periodontal ligament injection because the anesthetic solution travels through the ligamental space and enters the root canals of the tooth through lateral foramina.

The periodontal ligament injection also has some disadvantages. The access for proper needle placement is sometimes difficult, especially at the distal aspect of posterior teeth.[10,19,20] A common complaint of patients is that they get a bitter, unpleasant taste in their mouth due to the leakage of anesthetic solution at the injection site.[10,12,19,20] The use of a suction apparatus during injection has been suggested to help eliminate this problem.[10,12]

Another disadvantage of the periodontal ligament injection is that if the anesthetic solution is injected too rapidly there is an excessive build up of pressure which can cause the glass anesthetic cartridge to break within the syringe.[10,19] This presents a problem if the standard dental syringe is being used. The "pistol-type" syringes, as previously mentioned, have a protective sleeve around the glass anesthetic cartridge which prevents the spread of pieces of glass towards the patient or operator.[10,31] Therefore, it appears that in order for the periodontal ligament injection to be safely employed it should be performed only with the Peri-Press or

Ligmaject syringes. This is a disadvantage in that a special syringe has to be purchased in order to perform the injection safely.[10,19]

The periodontal ligament injection anesthetizes single teeth only.[10,19,20] Therefore, it would not be advantageous to consider its use when multiple procedures are to be performed in a single quadrant. A conventional nerve block would be preferable in these circumstances.[10]

The possibility of introduction of bacteria during the periodontal ligament injection and a resultant bacteremia is also a concern.[10,20,43] There is at present no evidence in the literature of bacteremia occuring following periodontal ligament injections.[20] Presumably a bacteremia does occur. However, it has been stated that the occurence is probably no greater than with other dental procedures.[43]

Two authors noted that a disadvantage of the periodontal ligament injection was that the 30 gauge needle, which is recommended by manufacturers, bent on insertion into the gingival sulcus.[24,31] This problem can be eliminated by using 25 or 27 gauge needles without decreasing the success rate of the injection.[31,41,43]

The periodontal ligament injection appears to have few adverse effects but, as with any dental procedure, some complications have been reported. Due to the excessive pressure required for success of this technique some evaluators worried about focal tissue damage due to pressure necrosis.[10,19] Kaufman et al[24] reported a case in which the patient developed papillitis and marginal necrosis at the injection site after a periodontal ligament injection.

Although there is little or no pain during injecton Malamed[31] reported a case where a patient with highly inflamed gingival tissues experienced considerable discomfort during the periodontal ligament injection. A few

authors have also reported a small incidence (approximately 4%) of postinjection pain with tenderness in the gingival and mucousal area surrounding the injection site.[10,12,15,19,24] This pain lasted from a few hours to two days.[15,24]

Another possible complication is that due to the excessive hydrostatic pressure developed during the periodontal ligament injection there is a potential for extrusion or avulsion of the tooth.[19] Nelson[33] reported a case in which a mandibular first premolar was to be extracted for orthodontic reasons. He stated that five minutes after performing the periodontal ligament injection the tooth became loose and the patient was able to remove it. Furthermore, even slight extrusion of a tooth after a periodontal ligament injection can complicate establishing proper occlusion of newly placed restorations.[19] Malamed[31] reported two cases where patients who had undergone minor restorative procedures complained of discomfort of the tooth after the anesthetic effect had terminated. In both cases examination of the occlusion revealed prematurities and after the occlusion was corrected there was immediate relief of the patients symptoms.

Faulkner[15] reported two cases in which the patients returned 48 hours after a periodontal ligament injection with a discrete swelling in the area of the injection. Both patients were treated with antibiotics for five days and the infections resolved. In his study there was no antiseptic placed into the gingival sulcus prior to the periodontal ligament injection. Faulkner[15] and Grainger[20] feel that the use of antiseptic solutions may be important in preventing this complication.

A final side effect which may occur was observed by Kaufman et al.[24] They reported that five patients had anesthesia of the lower lip for approximately 10 minutes after receiving a periodontal ligament injection

to the lower premolars and first molars. This was probably due to the anesthetic solution diffusing into the mandibular canal.[17,39]

Animal studies have been performed to demonstrate the spread of the anesthetic solution through the periodontal ligament and adjacent structures after the periodontal ligament injection.[17,39] Smith and Walton[39] injected a colloidal carbon dye into the periodontal ligament in dogs. They performed the periodontal ligament injection with a standard dental syringe. Their results showed that the distribution of the dye was frequently widespread and tended to be unpredictable. The dye was usually found in the periodontal ligament, periapical tissues, medullary bone and the pulp of injected teeth. They frequently found that the dye also spread to the same tissues of adjacent teeth.

The distribution of the dye with the periodontal ligament injection was not related to the needle location or the injection volume. However, distribution of the dye was consistently more widespread and deeper when the periodontal ligament injections were given under moderate to strong pressure. Conversely, light injection pressure resulted in passage of the dye into the gingiva or back out of the needle tract.[39] Clinically it has been shown that to obtain profound anesthesia the periodontal ligament injection must be given under strong back pressure.[41,43] Therefore, this dye study might indicate that when the periodontal ligament injection is not given with strong back pressure the anesthetic solution may be passing into the gingiva or refluxing along the needle tract rather than into the bone or periodontal ligament.[39]

Smith and Walton[39] concluded that since the dye passed through the lamina dura of the tooth socket and into the medullary bone that the periodontal ligament injection is actually an intraosseous injection.

Another study on the distribution of radiopaque material after a periodontal ligament injection in monkeys was performed by Garfunkel et al.[17] In this study the periodontal ligament injection was performed with the Peri-Press "pistol-type" syringe. Their results indicated that after the periodontal ligament injection was performed, under pressure, the radiopaque material filled the bone marrow spaces at the alveolar crest area of the interdental septum and advanced apically through the bone to the periapical region of the tooth. They found that the dye did not enter the periodontal ligament and stated that the spread of the radiopaque material is not via the periodontal ligament but rather via the alveolar bone. This differed from the results of Smith and Walton[39] who found that the dye solution spread intraosseously as well as through the periodontal ligament after a periodontal ligament injection.

Garfunkel et al[17] also reported that, with periodontal ligament injections, the dye spread to the mandibular canal or mental foramen. This could possibly explain why patients sometimes experience signs of mental nerve anesthesia following periodontal ligament injections.[17,24] They concluded that the periodontal ligament injection is really a type of intraosseous injection.[17]

Smith and Walton[39] noted that some patients complain of tachycardia and anxiety following the periodontal ligament injection. A study was therefore performed by Smith and Pashley[40] to evaluate the systemic effects of the periodontal ligament injection.

In their study they injected 0.3 cc of a variety of solutions into the periodontal ligament of dogs using the "pistol-type" syringe. They compared the systemic effects of the periodontal ligament injection with the systemic effects of other types of injections using the same quantity and type of solutions.[40]

Their results showed that when either epinephrine alone or xylocaine with epinephrine was injected intravenously, intraosseously or periodontally that similar changes occured in blood pressure and heart rate. These injections with epinephrine containing agents caused a transient decrease in blood pressure and an increase in heart rate. When epinephrine containing agents were injected intramuscularly, subcutaneously, submucosally or intrapulpally there were no changes in blood pressure or heart rate. When either xylocaine without epinephrine or saline was injected periodontally, intravenously or intraosseously there were no systemic changes.[40]

Smith and Pashley[40] concluded that the periodontal ligament injection is a type of intraosseous injection and that solutions injected into the periodontal ligament are rapidly absorbed into the systemic circulation. They suggested that after the periodontal ligament injection is performed under high pressure the anesthetic solution may flow rapidly through the alveolar lamina dura into the marrow spaces which contain venules. Further, they suggest that the high pressure developed may force the anesthetic solution into capillaries and venules so rapidly that it mimics an intravascular injection.

Lilienthal and Reynolds[28] demonstrated that intraosseous injections of anesthetics with vasoactive agents have a measurable affect on heart rate and blood pressure in human beings. Since the periodontal ligament injection really is a form of an intraosseous injection,[17,39,40] then probably catecholamine containing local anesthetic agents should not be injected into the periodontal ligament in medically compromised patients.[40] This is the same basic principle that is applied to intraosseous injections.[40]

Since the periodontal ligament injection injects the anesthetic solution directly into the periodontal ligament under strong pressure there is some concern over the detremental effects of the injection to the periodontium.[19] Two studies have been performed to show the histologic effects of the periodontal ligament injection on the periodontium.[8,44]

Walton and Garnick[44] performed periodontal ligament injections on monkeys using a 30 gauge needle and a standard dental syringe. They sacrificed the animals at different time periods following the injection and examined the histological effects of the periodontal ligament injection on the periodontium. They found that the needle puncture did not disturb the epithelial and connective tissue attachment to enamel and cementum. However, the procedure did cause slight damage to the tissues in the region of the needle penetration. This damage consisted of some inflammation in the area and slight resorption of bone in the alveolar crestal regions. There was no evidence of damage in the tissues apical to the needle penetration.

After 25 days the disruption that had occured showed repair. There was absence of inflammation and new bone had formed in the regions of resorption. The tissue anatomy and histology of the monkey periodontium is similar to humans and therefore, presumably, the same reactions would occur in humans after the periodontal ligament injection.[44] Walton and Garnick[44] concluded that the periodontal ligament injection is safe to the periodontium.

Another study was performed by Brannstrom et al[8] to determine the effect of the periodontal ligament injection using the Peri-Press "pistol-type" syringe. They injected 0.2 cc to 0.5 cc of anesthetic solution into the periodontal ligament of monkeys and sacrificed the animals at different time

periods postinjection. Histologic examination revealed that the periodontal ligament injection resulted in local tissue damage in the area of the injection. There was local inflammation and resorption of alveolar crestal bone. In no instance was there evidence of histologic change more than 1.5 mm from the area of the needle penetration.

The damage which had occured was found to be reversible and was in a state of repair two weeks after the periodontal ligament injection had been performed. They concluded that the periodontal ligament injection using a Peri-Press syringe is an acceptable technique if the periodontium is in good condition.[8]

However, Brannstrom et al[8] noted that when the periodontal ligament injection was performed on the mesial side of one tooth and on the distal side of the tooth in front of it there was increased bone loss at the alveolar crest. Although they did not study long term repair, they recommended that injections into the periodontal ligament on both sides of the interproximal alveolar bone should not be performed at the same appointment.

In order for the periodontal ligament injection to be successful in producing profound anesthesia Walton and Abbott[43] and Smith et al,[41] as previously discussed, showed that injecting under strong back pressure was critical. Malamed[31] reported that for the periodontal ligament injection to be successful there must be resistance to the deposition of the anesthetic solution. Khedari[25] also states that the two most important factors for a successful periodontal ligament injection are that the injection be given slowly and under strong pressure with firm resistance.

Birchfield and Rosenberg[6] performed a study on the role of the anesthetic solution in intrapulpal anesthesia. They wanted to determine if

the anesthesia produced by the intrapulpal injection is the result of the anesthetic solution or other factors.

They performed 56 intrapulpal injections on patients who were having endodontic treatment and had inadequate anesthesia after regional nerve blocks or infiltrations. Sterile normal saline was injected intrapulpally 37 times while the other 19 intrapulpal injections were performed with xylocaine 2% with epinephrine 1:50,000. The intrapulpal injections were made so that strong back pressure was attained during the injection. All of the intrapulpal injections performed obtained adequate anesthesia for the endodontic procedures.[6]

Birchfield and Rosenberg[6] demonstrated that there was no difference in the intrapulpal anesthesia produced when either xylocaine 2% with epinephrine 1:50,000 or sterile saline was used if the intrapulpal injection was given under strong pressure. Therefore, they concluded that pressure seems to be the major factor in producing the anesthesia.

Pashley et al[35] performed a study on dogs to measure the maximum pressures that can be produced with a standard dental syringe when giving a dental injection. They measured the pressures developed for a number of different dental injections using a pressure transducer attached to a standard dental syringe. They found that the mean tissue fluid pressure produced by the periodontal ligament injection was 17,630 mm Hg. The mean tissue fluid pressure produced by the intrapulpal injection was 8,918 mm Hg.

Pashley et al[35] also estimated the degree of tissue distensibility of various oral and extra-oral tissues. They found that the periodontal ligament is not very distensible and has the least compliance of the tissues tested. They concluded that injections which were made into the least distensible tissues developed the highest injection pressures and injections into more distensible tissues produced much lower injection pressures.

In summary, Birchfield and Rosenberg[6] have demonstrated that intrapulpal anesthesia after an intrapulpal injection is a function of pressure. Pashley et al[35] have shown that the pressure created during a periodontal ligament injection is greater than the pressure created with an intrapulpal injection. Walton and Abbott[43] and Smith et al[41] have demonstrated that for the periodontal ligament injection to be successful it must be given under strong pressure. Therefore, it is interesting to speculate on the possible role of hydrostatic pressure in producing anesthesia with the periodontal ligament injection.

If tissue fluid pressure is a factor in producing anesthesia with the periodontal ligament injection there are two possible explanations of the role of pressure in attaining anesthesia. One is that the increased tissue fluid pressure causes direct injury and/or compression of nerves supplying the tooth. Another possible mechanism of action may be that the elevated tissue fluid pressure may stop local tissue blood flow at the apex of the tooth producing pulpal ischemia with a resultant pulpal anesthesia.[19,35] Olgart and Gazelius[34] have demonstrated that decreasing or preventing local microcirculation at the apex of the tooth produced pulpal ischemia and resulted in inhibition of sensory nerve activity. Kim[26] suggested that one of the mechanisms of the periodontal ligament injection is a temporary cessation of blood flow to the pulp.

A number of researchers have performed studies, in areas other than dentistry, to show the effects of pressure on nerve conduction.[2,3,5,11,18,22,23,29,36] The mechanism by which pressure causes a decrease in nerve conduction is not fully understood. Some authors have shown that ischemia, secondary to the compression of the nerve, was the cause of the nerve conduction block[11,29,36] while others have shown that nerve conduction

block results from mechanical deformation of the nerve fibers.[5] Still other studies have suggested that the mechanism of action of pressure on nerve conduction is a combination of ischemia and mechanical deformation of the nerve.[16,23,37]

Bentley and Schlapp[5] reported a simple experiment by Weir Mitchell in 1872 which showed that conduction in the sciatic nerve of the rabbit could be interrupted in 20 to 30 seconds by the pressure of a column of mercury 18 to 20 inches* high. In their study, published in 1943, Bentley and Schlapp[5] demonstrated that when pressures between 130 and 200 mm Hg were applied to a peripheral nerve there was a decrease in nerve conduction after 40 minutes. The conduction through the peripheral nerve was completely blocked in 2½ to 3 hours.

Gelberman et al[18] performed a study to investigate the pressure threshold for peripheral nerve dysfunction. They applied pressure to the median nerve in the carpal tunnel of healthy human volunteers. Their results indicate that between 40 mm Hg and 50 mm Hg there exists a critical tissue fluid pressure threshold at which peripheral nerve function is impaired. They found that tissue fluid pressures of 50 mm Hg or greater resulted in complete sensory nerve block within 25 to 40 minutes.

A similar study on median nerve compression in the carpal tunnel and nerve function was performed by Lundborg et al.[29] They concluded that between 30 and 60 mm Hg there is a critical pressure level where nerve fiber function is impaired. They also found that compression of the median nerve at 60 mm Hg and 90 mm Hg resulted in complete sensory nerve conduction block within 20 to 40 minutes.

Another study on peripheral nerve conduction block by high muscle compartment pressure was conducted by Hargens et al.[22] They examined the

* 1.0 inch equals 2.54 cm.

effects of increased tissue fluid pressure on the function of the peroneal nerve in dogs. They demonstrated that a complete blockade of nerve conduction occurs at tissue fluid pressures of 50 mm Hg or more.

These studies seem to show that tissue fluid pressures of 50 mm Hg or greater block sensory nerve conduction.[5,18,22,29] The periodontal ligament injection, as previously mentioned, creates a mean tissue fluid pressure of 17,630 mm Hg.[35] This is much greater than pressures which the proceeding authors have shown are needed to block nerve conduction. This high tissue fluid pressure may occur at the injection site only and dissipate toward the apical region of the tooth. However, it is possible that a tissue fluid pressure of a magnitude great enough to block nerve conduction may occur within the periodontal ligament at the tooth apex. Therefore, hydrostatic pressure may play a role in producing anesthesia with the periodontal ligament injection.

In dentistry most injections involve the administration of a local anesthetic agent and a vasoconstrictor to produce sufficient anesthesia for the dental procedure. The vasoconstrictor is used to delay the absorption of the local anesthetic agent into the bloodstream.[30]

Kim et al,[27] in 1984, demonstrated in a study on dogs that epinephrine 1:100,000 administered by various dental injection techniques significantly decreased pulpal blood flow. They found that the intraosseous injection of epinephrine caused the most severe reduction in pulpal blood flow.

Edwall and Scott[13] showed that an increase in sympathetic nerve activity caused a reduction in pulpal blood flow and a marked depression in the excitability of the sensory unit in the tooth. They suggested that the excitability of sensory units in the tooth is strongly modulated by changes in pulpal microcirculation.

Olgart and Gazelius,[34] in 1976, reported the results of an animal study on the effects of epinephrine on blood flow and sensory nerve activity in the tooth. After supraperiosteal injections with epinephrine, either alone or with lidocaine, they found that the adjacent tooth had almost complete inhibition of pulpal blood flow within a few minutes. This was followed by a total inhibition of sensory nerve activity in the tooth. They showed, that by decreasing or preventing local microcirculation at the tooth apex and causing pulpal ischemia, that sensory nerve activity could be inhibited. Olgart and Gazelius[34] concluded that the excitability of sensory neurons in the tooth is decreased by the reduction of pulpal blood flow.

The above articles demonstrate that epinephrine injected adjacent to a tooth or sympathetic nerve stimulation inhibit pulpal blood flow and thereby, result in decreased sensory nerve activity within the tooth.[13,27,34] Therefore, it is possible to speculate that epinephrine may play a role in achieving anesthesia when it is injected into the periodontal ligament.

When the periodontal ligament injection is performed the dentist usually injects a local anesthetic agent with or without a vasoconstrictor under strong pressure in the hope of attaining profound anesthesia. To this point the possible role of hydrostatic pressure and epinephrine in producing local anesthesia for dental procedures, utilizing the periodontal ligament injection, have been discussed. A brief review of the mechanism of action of local anesthetic agents producing dental anesthesia is in order at this time.

Local anesthetic agents exert their pharmacological actions at the nerve membrane. The most favored theory today is the specific receptor hypothesis which proposes that local anesthetic agents act by attaching themselves to

specific receptors in the nerve membrane. The indication is that the local anesthetic receptor is located at or near the sodium channel in the nerve membrane.[30]

Local anesthetic agents are available as salts for clinical use. In solution the anesthetic salts exist as uncharged molecules (ie. base) and as positively charged molecules (ie. cation). The relative proportion of cation or base depends on the pH of the solution or surrounding tissues and the pKa or dissociation constant of the specific local anesthetic agent.[4,30]

It appears that both the charged ionic and the uncharged base form of local anesthetic agents are involved in the total process of nerve conduction block. The uncharged base, which is fat soluble, is believed responsible for optimal diffusion through the nerve sheath. After penetration of the nerve sheath reequilibration occurs between the base and cationic form. The charged cation then binds to a specific receptor site on the nerve membrane. This results in a blockage of the sodium channel and the failure in the development of an action potential. The result of this is the suppression of nerve transmission. Nerve blocks produced by local anesthetics are called nondepolarizing blocks.[4,30]

After a thorough review of the literature it is possible to speculate about the mechanism of action of the periodontal ligament injection. Previous studies have shown that the periodontal ligament injection attains adequate anesthesia in 80% to 90% of cases.[12,14,15,24,31,41,43] The question then arises, is the anesthesia produced with the periodontal ligament injection due to the local anesthetic agent, the vasoconstrictor, the hydrostatic pressure created when the injection is performed or a combination of these factors? No previous studies have investigated the

mechanism of action of the periodontal ligament injection.

A clinical study was therefore undertaken to investigate the possible mechanism of action of the periodontal ligament injection and its effectiveness in attaining anesthesia for exodontia.

Materials and Methods

Twenty eight patients between the ages of 16 and 75 with unremarkable medical histories were selected for the study. These patients had all presented to the emergency department at the Montreal General Hospital and had been referred to the department of oral surgery when it was determined that they required an extraction.

Included in the study were teeth which were judged to be simple, non surgical extractions which could be removed routinely with elevators and forceps. No third molars or surgical extractions were included in the study. Teeth with acute apical infections or drainage of pus from the gingival sulcus or surrounding tissues were also not included in the study. All teeth included in the study had a mobility index of M_1 (less than 0.5 mm in any direction). Since one criterion of pulpal anesthesia in this study was response of the tooth to the electric pulp tester, all teeth which did not respond initially to the Ritter electric pulp tester were excluded. Prior to the periodontal ligament injections being given no other means of attaining anesthesia of the tooth to be extracted had been performed. If the tooth was judged to be eligible for inclusion in the study then consent was received from the patient to use the periodontal ligament injection.

By means of random allocation the patients were placed into one of three groups. These groups were based on the solution injected into the periodontal ligament of the tooth to be extracted. The solutions injected were; in group 1 lidocaine 2%, in group 2 epinephrine 1:100,000 and in group 3 normal saline. The anesthetic cartridges were supplied containing the

above solutions by Astra Pharmaceuticals.* The randomization and appropriate code, revealing which solution was in each cartridge, were supplied by the pharmaceutical company.

This study was a double blind study in which the identity of the drug received by each patient was known only to the two registered nurses who dispensed the drug and maintained records. Subjects who satisfied all entry criteria were given a periodontal ligament injection with a solution from a coded cartridge. This protocol resulted in the formation of three randomly allocated study groups, whose treatment and treatment evaluation followed the criteria of a true, double blind randomized clinical trial design.

Although the study employs three treatment groups, as previously outlined, prior assumptions about the success of periodontal ligament injections using solutions with and without lidocaine permitted a design more economical in the use of subjects. Specifically, it was believed that a standard solution with lidocaine, could result in acceptable local anesthesia in 90 percent of cases. Hence, although three agents would be used (lidocaine, epinephrine and saline), the patients receiving the last two agents can be pooled to afford a "two sample" comparison, those patients submitting to an injection with lidocaine versus those patients injected without lidocaine.

The sample size calculations then became relatively straight forward. In the two sample case, the null hypothesis which claims no difference in the success rate of the agents (P_C-proportion success in the lidocaine group; P_T-proportion success in the lidocaine deficient group) leads to

*Astra Pharmaceuticals Canada Ltd., 1004 Middlegate Road, Mississauga, Ont.

the equation;

$$n = \left[\frac{Z_\alpha \sqrt{2\bar{\pi}(1-\bar{\pi})} - (-Z_\beta)\sqrt{\pi_T(1-\pi_T) + \pi_C(1-\pi_C)}}{\Delta} \right]^2$$

where n = sample size per group to be estimated.

Z_α = 1.96, based on two-tailed test at the 95% significance level.

$\bar{\pi}$ = estimated by P_C.

Z_β = 0.8, based on 20 percent change of β error from one-tailed normal distribution.

π_T = estimated by P_T. Since the study wanted to detect a 40% difference in success rate, and since lidocaine might be successful in about 90% of cases, the detectable P_T was set at 0.5.

π_C = estimated by P_C. The control group (lidocaine) was believed to have a 90% chance of obtaining anesthesia.

Δ = represents the difference in success rates the study wanted to detect. For the present study this was 40% or 0.4.

Calculations revealed the following;

$$n = \left[\frac{1.96\sqrt{2(.1)(.9)} - (-.8)\sqrt{(.5)(.5) + (.1)(.9)}}{.4} \right]^2$$

$$n = 10.36 \doteq 11$$

Since, however, the group not receiving lidocaine was to be subdivided into two, it was desirable to arrive at a sample size with an even number. Hence the sample size was increased to 12. This led to the following

arrangement;

Group	Projected Sample Size
#1 - Lidocaine	12
#2 - Epinephrine	6
#3 - Saline	6

During the actual experiment additional subjects became available and two people were added to the first group while each of the last two groups were enlarged by one.

Prior to the periodontal ligament injections the tooth was tested with the Ritter electric pulp tester. A positive response from 1 to 10 inclusive was documented.

The periodontal ligament injections were then performed using the Ligmaject "pistol-type" syringe with a 30 gauge short disposable needle. No topical anesthetic or antiseptic solution was placed into the gingival sulcus. The technique for injection was the same as described earlier in this paper. The needle was placed in the gingival sulcus at a 30 degree angle to the long axis of the tooth with the bevel facing away from the tooth. Apical pressure was applied until the needle was wedged into the periodontal ligament between the tooth and the alveolar crest of bone. The injection of approximately 0.2 cc of solution was then performed slowly over 20 seconds under strong pressure. Each tooth was injected at the mesiobuccal, distobuccal, mesiolingual and distolingual aspects of the tooth.

Thirty seconds after the last periodontal ligament injection had been performed the tooth was tested with the electric pulp tester. A positive

response between 1 to 10 inclusive or no response was recorded. The gingiva around the tooth was then tested for anesthesia by placing a number twelve periosteal elevator into the gingival sulcus and applying apical pressure. If the tooth did not respond to the pulp tester and the gingiva was completely anesthetized then an attempt was made to extract the tooth with elevators and forceps.

If the tooth still responded to pulp testing, the gingiva was not completely anesthetized or the tooth could not be removed due to patient discomfort then the periodontal ligament injections were repeated. The injections were made at the same four sites around the tooth using the same solution as the first injections. All injections again delivered approximately 0.2 cc of solution per injection site under strong pressure with the Ligmaject syringe.

Thirty seconds after the last periodontal ligament injection the tooth was again tested with the electric pulp tester. The gingiva was also tested again for anesthesia, as described earlier. If there was no pulpal response to the pulp tester and the gingiva was completely anesthetized then an attempt was made to extract the tooth with elevators and forceps.

If the pulpal response of the tooth to the electric pulp tester was still positive, the gingiva was not completely anesthetized or the tooth could not be extracted due to patient discomfort then a standard dental nerve block or infiltration procedure was performed with lidocaine 2% with epinephrine 1:100,000. Once adequate anesthesia was achieved the tooth was removed in routine fashion.

If the tooth was successfully removed after the periodontal ligament injections then the evaluation of the anesthesia obtained was subjectively assessed by the patient. The patient was asked if the extraction was

performed without discomfort or pain. If after the periodontal ligament injections the pulpal response of the tooth was still positive, the gingiva around the tooth was not completely anesthetized or the tooth could not be removed due to patient discomfort or pain then the tooth was placed into the category of unsuccessful extraction due to lack of anesthesia. The results for each tooth were recorded on a data collection sheet (Figure 2).

Figure 2. Data collection sheet: periodontal ligament injection study

Tooth #_____ Cartridge #_____

Electric pulp tester response _____

1st periodontal ligament injections

Electric pulp tester response _____

Anesthesia of gingiva YES _____

NO _____

2nd periodontal ligament injections

Electric pulp tester response _____

Anesthesia of gingiva YES _____

NO _____

Extraction of tooth

Successful with no discomfort or pain _____

Successful with minimal discomfort or pain _____

Unsuccessful due to lack of anesthesia _____

Operator Comments

Results

The average age of the patients was 43.3 years and the range was 16 to 74 years. Seventeen of the patients were males (60.7%) and 11 were females (39.3%) (Table 1).

Only one complication occured during this study. In one instance the glass anesthetic cartridge broke during the injection due to the pressure created by the Ligmaject syringe. This resulted in no problems as the Ligmaject syringe has a transparent plastic sheath that covers the anesthetic cartridge and protects the patient and operator in case of glass breakage. A new anesthetic cartridge of the same solution was obtained from the registered nurse and the periodontal ligament injections were performed.

Very few patients complained of pain on injection. No patients returned to the dental department complaining of pain or local complications at the sites of the periodontal ligament injections. There were no complaints of systemic complications, such as cardiac palpitations, when injecting any of the experimental solutions into the periodontal ligament.

In this study an attempt to attain anesthesia with the periodontal ligament injection, for exodontia, was performed on 28 teeth in 28 patients. The periodontal ligament injections were performed on 14 teeth using lidocaine 2%, 7 teeth using epinephrine 1:100,000 and 7 teeth using normal saline (Table 2).

The data collected for the lidocaine, epinephrine and normal saline groups is shown in tables 3, 4 and 5 respectively.

When the periodontal ligament injections using lidocaine 2% were

performed 11 of 14 teeth were successfully anesthetized and removed without discomfort or pain to the patient. This was a success rate of 79% (Table 6). All 14 teeth treated with lidocaine periodontal ligament injections had complete anesthesia of the gingiva around the tooth (Table 7) and did not respond when tested with the electric pulp tester (Table 8).

Seven teeth received periodontal ligament injections with epinephrine 1:100,000. There were no successful extractions achieved in this group due to lack of anesthesia (Table 6). In all 7 cases the gingiva was not anesthetized around the tooth (Table 7). However, there was a change in the pulpal response of each tooth when tested with the electric pulp tester before and after the periodontal ligament injections. The average decrease in pulpal response, in the epinephrine group, was 2.14 points when tested with the electric pulp tester (Table 8).

The 7 teeth which received periodontal ligament injections with normal saline could not be extracted due to lack of anesthesia (Table 6). Gingival anesthesia was not obtained for any of the 7 teeth in this group (Table 7). In this group a mean change of 0.14 points in the pulpal response to the electric pulp tester was observed (Table 8).

Tables

Table 1. Summary of age and sex distribution of patients

	Age							
	10-20	21-30	31-40	41-50	51-60	61-70	71-80	Total
Male	0	8	0	4	1	3	1	17
Female	1	2	1	3	3	0	1	11
Total	1	10	1	7	4	3	2	28

Average age 43.3 years

Table 2. Number of teeth in each of the experimental groups

Lidocaine 2%	Epinephrine 1:100,000	Normal Saline	Total
14	7	7	28

Table 3. Data collected for lidocaine 2% group patients response to electric pulp tester[a]

Tooth #	preinjection	postinjection	gingiva anesthetized	successful extractions
31	7	no response[b]	yes	yes
23	7	no response	yes	no
26	6	no response	yes	no
34	7	no response	yes	yes
35	5	no response	yes	yes
14	4	no response	yes	yes
37	3	no response	yes	yes
14	6	no response	yes	yes
33	2	no response	yes	no
12	2.5	no response	yes	yes
27	3	no response	yes	yes
46	6	no response	yes	yes
25	4	no response	yes	yes
13	8	no response	yes	yes

(a) the Ritter electric pulp tester delivers an electrical stimulus which increases on a scale from 1-10 (10 equals 1.5 volts AC).

(b) no response indicates that the patient had no perception of the strongest stimulus (#10) delivered by the Ritter electric pulp tester.

Table 4. Data collected for epinephrine 1:100,000 group

Tooth #	patients response to electric pulp tester[a] preinjection	postinjection	gingiva anesthetized	successful extractions
46	5	7.5	no	no
11	3	5.0	no	no
26	8	no response[b]	no	no
17	8.5	no response	no	no
37	7.5	no response	no	no
22	2	4.0	no	no
17	5	7.5	no	no

(a) the Ritter electric pulp tester delivers an electrical stimulus which increases on a scale from 1-10 (10 equals 1.5 volts AC).

(b) no response indicates that the patient had no perception of the strongest stimulus (#10) delivered by the Ritter electric pulp tester.

Table 5. Data collected for normal saline group

Tooth #	patients response to electric pulp tester[a] preinjection	postinjection	gingiva anesthetized	successful extractions
37	8	8	no	no
31	7.5	8	no	no
46	2	2	no	no
26	7.5	8	no	no
45	9	9	no	no
35	5	5	no	no
27	2	2	no	no

(a) the Ritter electric pulp tester delivers an electrical stimulus which increases on a scale from 1-10 (10 equals 1.5 volts AC).

Table 6. The efficacy of the periodontal ligament injections in obtaining adequate anesthesia for exodontia using three different solutions

Treatment Group	Number of teeth	Successful extraction yes	no	% success
Lidocaine 2%	14	11	3	79%
Epinephrine 1:100,000	7	0	7	0%
Normal saline	7	0	7	0%

X^2=18.12 df=1 Sig. $p < .01$

Note: this chi-square result is based on an analysis after collapsing the above table to a 2 x 2 format to eliminate low expected frequency counts (see discussion page 47).

Table 7. Anesthesia of gingiva obtained by periodontal ligament injections using three different solutions

Treatment Group	Number of teeth	Gingiva anesthetized yes	no	% success
Lidocaine 2%	14	14	0	100%
Epinephrine 1:100,000	7	0	7	0%
Normal saline	7	0	7	0%

X^2=28 df=1 Sig. $p < .01$

Note: this chi-square result is based on an analysis after collapsing the above table to a 2 x 2 format to eliminate low expected frequency counts (see discussion page 47).

Table 8. Mean stimulus required from electric pulp tester[a] to elicit a response among three groups of patients receiving periodontal ligament injections.

Treatment Group	Number of teeth	Mean baseline stimulus	Mean stimulus change
Lidocaine 2%	14	5.04(1.95)[b]	4.96[c](1.95)
Epinephrine 1:100,000	7	5.57(2.52)	2.14(0.38)
Normal saline	7	5.86(2.90)	0.14(0.24)

Using analysis of variance (ANOVA), mean baseline stimulus scores not statistically different among groups. $F=0.32$ $df=24$ $p > .05$

Using ANOVA, the differences of mean stimulus change among groups are significant. $F=28.8$ $df=24$ $p < .01$

(a) the Ritter electric pulp tester delivers an electrical stimulus which increases on a scale from 1-10 (10 equals 1.5 volts AC).

(b) standard deviation in brackets

(c) there was no response of the patient to maximum stimulus (ie. level 10) with the Ritter electric pulp tester.

Discussion

The periodontal ligament injection using the Ligmaject or Peri-Press "pistol-type" syringes is becoming more popular in dentistry. In 1981, de Shazer and Coffey[12] said that they expect the mandibular block to take a back seat to the periodontal ligament injection in attempting to attain anesthesia for a variety of dental procedures. This relatively new injection technique is advantageous in that it is painless, easy to administer, quick acting, has patient acceptance and is suitable for most dental procedures. The periodontal ligament injection also causes no numbness of cheeks or tongue, no nerve damage and the anesthetic effect is of short duration.[12]

A number of authors, as previously discussed, have shown that the periodontal ligament injection is able to produce adequate anesthesia, for a variety of dental procedures, approximately 80% to 90% of the time.[12,14,15,24,31,41,43] In those studies where some extractions were performed adequate anesthesia for exodontia was reported to occur in 95% of cases.[15,24]

The present study differs from the previous studies in that it used pulpal response of the teeth to the electric pulp tester, gingival anesthesia and dental extractions to evaluate the effectiveness of the periodontal ligament injection. Previous studies had used surgical, restorative, endodontic and periodontal procedures to assess effectiveness.[12,14,15,24,31,41,43]

In this study it was found that when the periodontal ligament injections were performed with lidocaine 2% gingival anesthesia was obtained in all

instances (Table 7). The periodontal ligament injections with lidocaine also eliminated the pulpal response, to the electric pulp tester, in all 14 teeth (Tables 3 and 8).

It is assumed, since all 14 teeth had profound pulpal and gingival anesthesia after periodontal ligament injections with lidocaine, that restorative procedures could have been performed without discomfort or pain to the patient. However, when dental extractions were attempted only 11 of 14 teeth could be extracted without pain or discomfort to the patient. This is a success rate of the periodontal ligament injections with lidocaine, in producing anesthesia for extractions, of 79% (Table 6). This compares favourably to the overall success rates obtained in previous studies where the periodontal ligament injections were performed using a "pistol-type" syringe for a variety of dental procedures.[12,14,15,24,31,41] However, it is lower than the 95% success rate achieved by others for extractions.[15,24]

This difference in success rate may be due to the fact that the 3 teeth which could not be extracted were all difficult extractions where a great deal of force had to be applied to remove each tooth. Two of the teeth that could not be extracted after periodontal ligament injections with lidocaine were cuspids. Kaufman et al[24] noted that they had a success rate of only 46% when trying to obtain anesthesia of cuspids with periodontal ligament injections. They noted that the length of the root apparently influenced the success of intraligamentary anesthesia. It was also noted by de Shazer and Coffey[12] that it was difficult, when using periodontal ligament injections, to anesthetize cuspids for dental procedures. The other tooth that was not successfully removed after periodontal ligament injections with lidocaine was a maxillary first molar. This tooth had long roots that were very divergent. Therefore, it may be that in instances where an extraction

is difficult, the anesthetic obtained with periodontal ligament injections is not profound enough to permit removal of the tooth.

The American Dental Association council on dental materials, instruments and equipment reported in 1983 that the actual mechanism of action of intraligamentary anesthesia has yet to be determined.[19]

Studies have shown that epinephrine decreases pulpal blood flow which results in partial or complete inhibition of sensory nerve conduction.[13,27,34] Therefore, it is possible to speculate that epinephrine plays a role in attaining anesthesia with the periodontal ligament injection.

No previous investigation has studied the effect of epinephrine in attaining anesthesia with the periodontal ligament injection. In this study 7 teeth were injected with epinephrine 1:100,000 by means of the periodontal ligament injection. These periodontal ligament injections with epinephrine did not produce gingival anesthesia in any instance (Table 7). The epinephrine injections also did not produce adequate anesthesia to permit any dental extractions in this group (Table 6). It is interesting to note that after the periodontal ligament injections with epinephrine there was a change in the pulpal response of each tooth when tested with the electric pulp tester. The pulpal response of the teeth which received periodontal ligament injections with epinephrine diminished, on the average, 2.14 points. Using the Student-Newman-Keuls procedure for multiple comparisons this decrease in response to stimulus, relative to injections with saline solution only, is statistically significant at the $p < .05$ level (Table 8). This demonstrates that epinephrine causes a decrease in sensory nerve activity within the tooth. Olgart and Gazelius,[34] as previously discussed, also demonstrated a decrease in sensory nerve activity in teeth following supraperiosteal epinephrine injections. Therefore, when the periodontal

ligament injection is performed with a local anesthetic agent with epinephrine it is possible that the vasoconstricting action of epinephrine is synergistic with the local anesthetic agent in achieving maximum anesthesia of the tooth.

Walton and Abbott,[43], Malamed,[31] Kaufman et al[24] and Smith et al[41] have all reported that periodontal ligament injections, in order to be successful, must be administered under strong pressure with firm resistance. Pashley et al[35] demonstrated that the mean tissue fluid pressure created with the periodontal ligament injection was 17,630 mm Hg while the mean tissue fluid pressure created with the intrapulpal injection was 8,918 mm Hg. Birchfield and Rosenberg,[6] in 1975, demonstrated that intrapulpal anesthesia is a function of pressure. They found that there was no difference in attaining anesthesia when either normal saline or lidocaine 2% with epinephrine 1:50,000 was injected intrapulpally under pressure. Therefore, it is possible to speculate that pressure is a factor in attaining anesthesia with the periodontal ligament injection.

Pressure has been shown, by a number of authors, to decrease or block nerve conduction.[2,3,5,11,18,22,23,29,36] Hargens et al,[22] Lundborg et al[29] and Gelberman et al[18] demonstrated that tissue fluid pressures of 50 mm Hg or greater completely blocked sensory nerve conduction. The exact machanism by which pressure blocks nerve conduction is not completely understood. Some authors feel that the pressure causes ischemia of the nerve and this results in a decrease in nerve conduction.[11,29,36] Others say that the pressure causes direct injury or mechanical deformation of the nerve.[5] Still other authors feel it is a combination of the two.[16,23,37] Since the periodontal ligament injection has been shown to create a mean tissue fluid pressure of 17,630 mm Hg[35] these studies[18,22,29] would tend to support the

concept that hydrostatic pressure is a factor in the anesthesia obtained with the periodontal ligament injection.

No other studies have investigated the effect of pressure in producing anesthesia with the periodontal ligament injection. The periodontal ligament injections performed in this study were all done under strong pressure.

It is apparent from tables 6 and 7 that there was no difference in the epinephrine and saline groups in producing gingival anesthesia or adequate anesthesia for dental extractions. These two groups were therefore combined to permit a 2 x 2 chi-square analysis to test the association of anesthetic with hydrostatic pressure and/or lidocaine 2% when used in a periodontal ligament injection.

Table 6 shows that while there was a 79% success rate in performing extractions in the lidocaine group, no extractions could be performed in either the saline or epinephrine groups. When this was analyzed using the 2 x 2 chi-square test, it is obvious that there is a statistically and clinically significant relationship between anesthesia and the agent being employed for the periodontal ligament injection. It may also be seen in table 7 that there was no gingival anesthesia in either the epinephrine or saline groups while in the lidocaine group gingival anesthesia was obtained in all instances. When this relationship was analyzed using the 2 x 2 chi-square test, it is also obvious that there is a statistically and clinically significant relationship between anesthesia and the agent being employed for the periodontal ligament injection. Evidently it is absolutely essential, for extractions and gingival anesthesia, that the solution used for the periodontal ligament injection contain a local anesthetic agent (eg. lidocaine). The hypothesis that, with the periodontal ligament

injection, lidocaine and hydrostatic pressure are equally effective in producing anesthesia cannot be supported from these results.

Lidocaine 2% and epinephrine 1:100,000 periodontal ligament injections have been shown previously in this paper to reduce the pulpal response of the tooth to the electric pulp tester. Only a minimal decrease in mean pulpal response occured after the saline injections. The saline periodontal ligament injections did not result in a statistically significant change in pulpal response based upon the paired t-test ($t=-1.55$, not significant, $df=6$) (Table 8).

Therefore, based on the three criteria (gingival anesthesia, pulpal response and dental extractions) used to assess anesthesia in this study it appears that after clinical and statistical analysis the hydrostatic pressure generated with the periodontal ligament injection does not decrease nerve conduction and cause anesthesia. The high pressure, which is known to occur at the injection site,[35] is either not transferred directly to the apex of the tooth or if the pressure does reach the apex of the tooth it is insufficient to inhibit nerve conduction. This may be due to the fact that the anesthetic solution spreads to the surrounding tissues, as shown in the dye studies,[17,39] and results in dissipation of the tissue fluid pressure. The only definitive method of determining if any hydrostatic pressure reaches the apex of the tooth after a periodontal ligament injection would be to place a pressure transducer at the tooth apex in an animal model.

Smith and Walton,[39] in 1983, demonstrated that the distribution of the injected material with the periodontal ligament injection depended on the pressure created during the injection. They found that if the periodontal ligament injection was given under strong back pressure that there was spread of the injected material throughout the periodontal ligament,

periapical tissues, medullary bone and pulp of injected and adjacent teeth. Conversely, they found that if the periodontal ligament injection was given under little pressure that there was no spread or penetration of the injected material apically into the deeper tissues. It has been shown in the present study that pressure itself does not cause anesthesia with the periodontal ligament injection. Therefore, it is reasonable to speculate that, for the periodontal ligament injection to be successful, pressure on injection is required only for the spread of the local anesthetic agent throughout the periodontal ligament and medullary bone to the apical region of the tooth.

Summary

This study was undertaken to evaluate the effectiveness and possible mechanism of action of the periodontal ligament injection in attaining anesthesia for exodontia.

The study was double blind and conducted on 28 teeth. Periodontal ligament injections under strong pressure were performed on all teeth using one of three different solutions. The periodontal ligament injections were performed with lidocaine 2% on 14 teeth. Seven teeth had injections with epinephrine 1:100,000. The other seven teeth received periodontal ligament injections with normal saline.

The only periodontal ligament injections which were able to produce profound anesthesia so that the tooth could be extracted were the injections performed with lidocaine 2%. Eleven out of fourteen teeth were successfully removed without discomfort or pain to the patient. This is a success rate of 79%. All 14 teeth in the lidocaine group had complete gingival anesthesia around the tooth and lack of pulpal response when tested with the electric pulp tester.

Anesthesia adequate enough to permit dental extractions was not obtained following periodontal ligament injections with epinephrine 1:100,000 or normal saline. The injections with epinephrine or saline produced no gingival anesthesia. The pulpal response of the teeth, to the electric pulp tester, had no significant change after periodontal ligament injections with normal saline. The periodontal ligament injections with epinephrine significantly diminished the pulpal response of the teeth, to the electric pulp tester, by an average of 2.14 points.

Conclusions

It was concluded from this study that periodontal ligament injections, when performed with the Ligmaject syringe and lidocaine 2%, were effective in attaining anesthesia for routine exodontia.

This study also demonstrates that hydrostatic pressure created by the periodontal ligament injection does not have a direct effect, through ischemia or mechanical deformation of the nerve fibers, on sensory nerve conduction. However, it appears that with the periodontal ligament injection pressure is required on injection for the spread of the anesthetic solution to the apical region of the tooth. Therefore, the mechanism of action of the periodontal ligament injection in producing anesthesia depends on the local anesthetic agent with pressure on injection being required for distribution of the anesthetic agent.

This study also demonstrates that epinephrine, injected into the periodontal ligament, decreases sensory nerve activity within the tooth. Therefore, it may be possible that when the periodontal ligament injection is performed with a local anesthetic agent with epinephrine there may be a synergistic effect in achieving maximum anesthesia.

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