RETROGRADE DEGENERATION OF THE TRIGEMINAL GANGLION OF ALBINO RAT AFTER TOOTH EXTRACTION

By

**Adel Abbass Bondok, Ibrahim Attia Shaaban,
Raouf Fikri Bedir and Mohammed El-Shafie**

From

*Department of Anatomy, Faculty of Medicine,
Mansoura University*

ABSTRACT

The present study was carried out to investigate the retrograde reaction in the primary sensory neurons of the rat trigeminal ganglion after tooth extraction.

Forty adult albino rats of both sexes with average body weight were used in this study. The animals were subjected to extraction of the lower left incisor tooth and the right non-operated side was used as control. Rats were sacrificed at survival periods 2, 7, 14, 21 and 28 days after the day of tooth extraction (8 rats for each survival period). The control and experimental trigeminal ganglia, were dissected out and processed for light microscopic examination using cresyl

violet, toluidine blue. The trigeminal ganglia of two rats were removed at 2, 7, and 14 days after tooth extraction and were processed for electron microscopic examination.

Light microscopic examination of sections of the trigeminal ganglia revealed signs of degeneration in nerve cells in the form of chromatolysis and displacement of the nucleus. Signs of chromatolysis were absent in the control ganglia. Staining the trigeminal ganglia for acid phosphatase activity revealed strong acid phosphatase activity in almost all the nerve cells at all the survival periods after tooth extraction. The control nerve cells showed absence of acid phosphatase activity.

Ultrastructural examination of the trigeminal ganglion of the rat after tooth extraction revealed extensive signs of degeneration in nerve cells. The cytoplasm of some cells appeared vacuolated and the nuclei were shrunken and had irregular outline. Extensive degeneration of the axons was observed. The myelin was completely dissolved and was replaced by vacuoles and debris and the surrounding Schwann cells contained large number of degenerated materials.

Therefore, it is concluded in this investigation that tooth extraction affects the nerve terminals of the trigeminal nerve and induced retrograde changes in trigeminal ganglion cells with degeneration and death of the nerve cells, a picture of retrograde degeneration.

INTRODUCTION

Mammals have four kinds of teeth that differ in shape, function, position in the mouth and whether or not they are replaced. The four types are incisors, canines, premolars and molars. Rats have incisors (one on each side) and molars (three on each side). Rats

do not have canines or premolars. Rats have 8 teeth on the upper jaw and 8 on the lower jaw (4 on each side), a total of 16 teeth (Widdowson, 1952). Rats are monophyodont, which means that they have one set of teeth during their lifetime (they are never replaced). In comparison, humans are polyphyodont, because one set is lost and another set grows (Hilscher-Conklin, 1996). Teeth of the upper jaw are supplied by branches from the maxillary division of the trigeminal nerve. The teeth of the lower jaw are supplied by a branch from the mandibular division of the trigeminal nerve, the inferior alveolar nerve (Guglielmotti and Cabrini, 1985).

Previous investigations have shown that disturbance of the sensory input from the teeth results in a definite degeneration of the primary sensory neurons (Fried et al. 1991; Chudler et al., 1997). Kubota et al. (1988) observed signs of retrograde degeneration during the first two weeks after tooth extraction. Hansen (1980) examined the neurohistological reactions in the trigeminal ganglion after tooth extraction. He observed signs of retrograde degeneration dur-

ing the first two weeks after extraction. Fried et al. (1991) in their study on the inferior alveolar nerve lesions or tooth injury demonstrated that pulpal differentiation may change the long term cytochemical characteristics of the affected trigeminal ganglion neurons.

Extraction of teeth damages the nerve fibers innervating the teeth and induces peripheral structural, biochemical and physiological changes in the primary sensory neurons (Tsuji-no et al., 2000; Kaori et al., 2006).

Bigotte and Olsson (1987) induced selective damage to a certain branch of the trigeminal ganglion of the mouse. They detected degeneration of trigeminal ganglion neurons. Itotagawa et al. (1993) studied the effects of dental injuries of the upper first molar on the trigeminal ganglion of rats and found that dental injuries alter the primary sensory neurons in the trigeminal ganglion.

Westrum et al., (1984) and Henery et al., (1987) demonstrated ultrastructural transganglionic degeneration in brainstem trigeminal nuclei during

normal primary tooth exfoliation and permanent tooth eruption and following the application of the potent toxin ricin to the tooth pulps of unilateral maxillary and mandibular posterior teeth. Similar observations were observed in the Clarke's nucleus after sciatic nerve crush at birth (Bondok, 1994).

Charkas et al. (2004) as well as Bryce and William (2004) have shown that when a nerve trunk is transected, certain changes take place proximal and distal to the injury. On the proximal side of the injury (toward the cell body), degenerative changes extend to the nerve cell bodies. According to the nature of the injury, nerve cell bodies either degenerate or regenerative processes are initiated (Bondok and Gawish, 1994-b as well as Junqueira and Carneiro 2003).

In the light of the previous investigations, the present study was carried out to investigate the retrograde reaction in the primary sensory neurons of the rat trigeminal ganglion after tooth extraction. The lower left incisor tooth was selected as a model.

MATERIAL AND METHODS

Animals Used :

Forty adult albino rats of both sexes with average body weight between 200-250 gm were used in this study. The animals were subjected to tooth extraction of the lower left incisor tooth and the right non-operated side was used as control. Rats were sacrificed at survival periods 2, 7, 14, 21 and 28 days after the day of tooth extraction (eight rats for each period)..

Tooth Extraction :

The rats were anaesthetized with an intramuscular injection of ketamine (70mg/kg) and xylazine (13mg/kg) according to Kaori et al. (2006). The mouth was opened; the gum was excavated around the lower left incisor tooth. The tooth was then held by forceps and moved slowly from side to side to loosen it and to avoid its fracture during extraction and the right non operated side was used as control.

Tissue Preparation :

At the assigned dates, rats were anaesthetized under deep anesthesia by intraperitoneal injection of 10% chloral hydrate (300mg/kg). For light

microscopy, the rats were perfused transcardially by a needle in the left ventricle and the right atrium was incised with formol saline. For electron microscopy, with 500 ml of 2.5% buffered glutaraldehyde in 0.2 M phosphate buffer containing 0.2% picric acid. For acid phosphatase activity the specimens were frozen by Freon, then were cut at 25 um on the freezing microtome. The sections were mounted on albumin coated slides, air dried and then stained.

Obtaining the Specimens :

A midsagittal incision through the scalp and the skull bones was made and another anterior and posterior coronal incisions were made in the parietal bones. The two parietal bones were reflected laterally. The cerebrum, the cerebellum and the brainstem were carefully exposed. The two cerebral hemispheres were reflected dorsally to expose the trigeminal ganglia with their sensory root.

A. For light Microscopy :

The trigeminal ganglia were dissected out and kept in 10% formol saline for 24 hours, dehydrated,

cleared in xylene and embedded in paraffin, sections were cut at 10 μ m and stained with cresyl violet stain. the fixative, embedded in paraffin and processed for staining with :

1. Cresyl violet for staining the Nissl granules.
2. Acid phosphatase for staining the lysosomes.

B. For Electron Microscopy :

Two rats at the age of 2, 7 and 14 days postoperatively were used. The trigeminal ganglia were immediately removed and cut into small pieces and kept in 2.5% buffered glutaraldehyde solution in the refrigerator for 24 hours. The specimens were then rinsed in phosphate buffered saline, postfixed in phosphate buffered 1.0% osmium tetroxide, dehydrated in graded series of ethanol and subsequently immersed into 100% propylene oxide. The specimens were embedded in epoxy resin (Wells and Vaidya, 1989). One micron sections were cut and stained with toluidine blue to determine the area of interest. Ultrathin sections were cut on a Reichert ultracut microtome and stained with uranyl acetate and lead citrate and examined and photographed under JEOL

JEM 1010 transmission electron microscope.

RESULTS

CONTROL TRIGEMINAL GANGLION

A. Light Microscope :

Examination of paraffin sections stained with cresyl violet (Figs. 1, 2) and toluidine blue (Fig. 3) revealed that the trigeminal ganglion was surrounded by a connective tissue capsule and contained nerve cells of different sizes: large, medium and small. Most of the nerve cells were located at the periphery of the ganglion (Fig. 1). The cells were arranged in groups or as elongated cords separated by bundles of nerve fibers. The nerve cell bodies appeared oval or rounded in shape. A capsule of flat satellite cells surrounded each nerve cell. The nucleus was usually large, vesicular, centrally located and contained a prominent nucleolus. The cytoplasm contained Nissl granules that appeared as coarse, irregular, basophilic granules. The Nissl granules were dispersed throughout the cytoplasm. The distribution of the Nissl granules gave most of the large cells

a light color and most of the small cells a dark color (Fig. 2).

Staining frozen sections for the demonstration of acid phosphatase activity showed absence of acid phosphatase activity in almost all the nerve cells (Fig. 4).

B. Electron Microscope :

Ultrastructural examination of the trigeminal ganglion of the rat revealed that the nerve cells contained different types of organelles such as free ribosomes, rough endoplasmic reticulum (rER), Golgi apparatus, mitochondria. The cytoplasm contained also variable numbers of lysosomes and cytoskeletal elements. It had a large nucleus surrounded by bilaminar nuclear membrane and contained scattered heterochromatin (Fig. 5). The satellite cells surrounding the nerve cells appeared darker than the nerve cells due to the condensation of the organelles in the cytoplasm (Figs. 6, 7).

The area between the nerve cells (Fig. 9) contained large number of myelinated axons. Their myelin lamellae appeared organized and regularly

arranged. The axoplasm characterized by the presence of large number of microtubules, the presence of mitochondria and profiles of smooth endoplasmic reticulum. The myelinated nerve fibers were wrapped by schwann cell cytoplasm. The nerve fibers were surrounded by the endoneurium that is formed of bundles of collagen fibers (Fig. 8).

EFFECT OF TOOTH EXTRACTION

A. Light Microscope :

Light microscopic examination of sections of the trigeminal ganglia stained with cresyl violet and toluidine blue at different survival periods after extraction of the incisor teeth revealed signs of degeneration in some nerve cells. These degenerating nerve cells showed signs of chromatolysis in the form of dispersion of Nissl granules at the periphery of the cell and the presence of eccentric nuclei (Figs. 10, 11, 12, 13, 14, 15, 16). The chromatolysis, eccentric nucleus and peripheral displacement of Nissl granules appeared on the second day after tooth extraction and persisted throughout the experimental period (Figs. 10, 11, 12, 13, 14, 15, 16). The

cytoplasm appeared vacuolated with shrunken nucleus (Figs. 14, 15).

Staining the trigeminal ganglia for acid phosphatase activity revealed strong acid phosphatase activity in almost all the nerve cells at all the survival periods after tooth extraction. The reaction appeared as coarse granules that were arranged mostly in the perinuclear region (Figs. 17, 18, 19, 20, 21, 22, 23, 24).

B. Electron Microscope :

Ultrastructural examination of the trigeminal ganglion of the rat after tooth extraction revealed the presence of signs of degeneration in nerve cells and axons.

Two days after tooth extraction (Fig. 25), the cytoplasm showed dilated rough endoplasmic reticulum (rER) cisternae in the peripheral region of the perikarion and the presence of numerous lysosomes. The nucleus appeared shrunken and had a folded membrane (Fig. 26). The cells were surrounded by satellite cells, which showed an increased number of lysosomes and dilated rER

cisternae (Fig. 27). Some axons showed vacuolation and disintegration of their myelin and their axoplasm contained electron dense bodies of different sizes (Fig. 28).

One week (Figs. 29, 30, 31, 32) and two weeks (Figs. 33, 34) after tooth extraction, extensive signs of degeneration were observed in nerve cells and axons. In the cytoplasm, the ribosomes were displaced to the periphery and the lysosomes were present in large numbers. The cytoplasm of some cells appeared vacuolated and the nuclei were shrunken and had irregular outline (Fig. 30). Some degenerating cells appeared surrounded with macrophages (Fig. 29). Extensive degeneration of the axons were observed (Figs. 31, 32). The myelin was completely dissolved and was replaced by vacuoles and debris (Fig. 31) and the surrounded Schwann cells contained large number of degenerated materials (Fig. 32). Two weeks after tooth extraction, degenerating neurons containing multiple vacuoles, irregular pyknotic nuclei and surrounded by satellite cells were observed (Figs. 33, 34).

Fig. 1 : Micrograph of the control trigeminal ganglion of rat showing large number of nerve cells of different sizes that are distributed at the periphery of the ganglion. Cresyl violet, X 100.

Fig. 2 : Micrograph of the control trigeminal ganglion of rat, showing ganglion cells appear variable in size (crossed arrows). The cytoplasm of these cells filled with dispersed Nissl granules. The characteristic large, round, central and vesicular nucleus and distinct nucleolus is conspicuous. The ganglion cells are surrounded by smaller satellite cells (arrows). Between ganglion cells are numerous fibroblast (double arrows), randomly arranged in the connective tissue. Cresyl violet, X 400

Fig. 3 : Micrograph of the control trigeminal ganglion of rat, demonstrating ganglion cells of different sizes. The cells are surrounded by satellite cells (arrows) and nerve fibers. Toluidine blue, X 400.

Fig. 4: Micrograph of Frozen section of the control trigeminal ganglion, showing absence of acid phosphatase activity in almost all the nerve cells. Acid Gormori, X 400

Fig. 5 : Electron micrograph of a nerve cell in the control trigeminal ganglion of rat, revealing euchromatic nucleus (N) with distinct nucleolus (*) and surrounded by cytoplasm rich in different types of organelles: numerous mitochondria (m), golgi complex (G) in the perinuclear region and numerous. X 9000.

Fig. 6 : Electron micrograph of a nerve cell in the control trigeminal ganglion of rat, exhibiting the perikaryon containing randomly arranged organelles. Nissl bodies (white arrows) are distributed throughout the perikaryon. The initial process (P) of the nerve cell is seen containing large number of microtubules and mitochondria (dark arrows). The cell is surrounded by a satellite cell (S). X 9,000.

Fig. 7 : Electron micrograph of a nerve cell in the control trigeminal ganglion of rat, revealing the perikaryon of the nerve cell (N) contains different organelles that are randomly distributed. The cytoplasm of satellite cell (S) appears darker than that of the neuron and contains rough endoplasmic reticulum (rER) and lysosomes (L). X 11,000.

Fig. 8 : Electron micrograph of the control trigeminal ganglion of rat, showing myelinated axon in the space between the nerve cells. The axoplasm (A) contains microtubules and is surrounded by regularly arranged myelin lamellae. The nerve fiber is wrapped by Schwann cell (S). X 13,000.

Fig.9 : Electron micrograph of the control trigeminal ganglion of rat, showing myelinated axons (A) filled with microtubules. The myelin is surrounded by a Schwann cell (S). X 23,000.

Fig. 10 : Micrograph of the experimental trigeminal ganglion of rat, 2 days after tooth extraction, showing 2 degenerated nerve cells (N). These nerve cells have dispersed Nissl granules at the periphery of the cell (arrows). Other nerve cells have dispersed Nissl granules all over the cytoplasm. Cresyl violet, X 400.

Fig. 11 : Micrograph of the trigeminal ganglion of rat, 2 days after tooth extraction, showing 2 chromatolytic neurons (N) with eccentric and shrunken nuclei (arrows). The other neurons show large vesicular central nuclei. Toluidine Blue, X 400.

Fig. 12 : Micrograph of the trigeminal ganglion of rat, one week after tooth extraction, showing a degenerating nerve cell (N) with signs of chromatolysis. Nissl granules (arrow) are displaced to the periphery of the cell and the nucleus is eccentric in position. Cresyl violet, X 400.

Fig. 13 : Micrograph of the trigeminal ganglion of rat, 2 weeks after tooth extraction, revealing degenerated neurons (N). Toluidine Blue, X 400.

Fig. 14 : Micrograph of the trigeminal ganglion of rat, 3 weeks after tooth extraction, illustrating degenerated neurons (N) with vacuolated cytoplasm (arrows) and shrunken nucleus. Cresyl violet, X 400.

Fig. 15 : Micrograph of the trigeminal ganglion of rat, 3 weeks after tooth extraction, demonstrating nerve cells (N) with signs of chromatolysis. The Nissl granules (arrows) are dispersed at the periphery of the cell. Cresyl violet, X 400.

Fig. 16 : Micrograph of the trigeminal ganglion of rat, 4 weeks after tooth extraction, showing degenerated nerve cells (N) with peripheral displacement of the Nissl granules (arrow). Cresyl violet, X 400

Fig. 17 : Micrograph of the trigeminal ganglion of rat, 2 days after tooth extraction, illustrating peripheral distribution of the nerve cells. The cells show strong acid phosphatase activity. Acid Gomori. X 100.

Fig. 18 : Micrograph of the trigeminal ganglion of rat, 2 days after tooth extraction, showing strong acid phosphatase activity in the nerve cells. Acid Gomori, X 400.

Fig. 19 : Micrograph of the trigeminal ganglion of rat, 1 week after tooth extraction, showing strong acid phosphatase activity in the nerve cells. The nerve cells are condensed at the periphery of the ganglion.. Acid Gomori. X 100.

Fig. 20 : Micrograph of the trigeminal ganglion of rat, 1 week after tooth extraction, demonstrating strong acid phosphatase activity in the nerve cells. Acid Gomori. x 400.

Fig. 21 : Frozen section of the trigeminal ganglion of rat, 2 weeks after tooth extraction, showing strong acid phosphatase activity in the nerve cells. The nerve cells are arranged at the periphery of the ganglion. Acid Gomori, X 100.

Fig. 22 : Micrograph of the trigeminal ganglion of rat, 2 weeks after tooth, extraction illustrating intense acid phosphatase activity in the nerve cells. Acid Gomori, X 400.

Fig. 23 : Micrograph of the trigeminal ganglion of rat, 3 week after tooth extraction, showing intense acid phosphatase activity in the nerve cells. Acid Gomori, X 100.

Fig. 24 : Micrograph of the trigeminal ganglion of rat, 3 week after tooth extraction, showing intense acid phosphatase activity in the nerve cells. Acid Gomori, X 400.

Fig. 25 : Electron micrograph of a part of the trigeminal ganglion neuron (N) 2 days after tooth extraction showing dilated rough endoplasmic reticulum cisternae (rER) in the peripheral region of the perikaryon and numerous lysosomes (arrows). The nerve cell is surrounded by a satellite cell (S). X 12.000.

Fig. 26 : Electron micrograph of the trigeminal ganglion 2 days after tooth extraction, revealing degenerating nerve cell (N). The nucleus (Nu) is shrunken, irregular and folded. The cytoplasm contains distorted organelles (arrows) and many lysosomes (arrowheads). X 9.500.

Fig. 27 : Electron micrograph of a part of the trigeminal ganglion nerve cell (N) two days after tooth extraction, showing dilated rough endoplasmic reticulum cisternae (black arrows). The satellite cell (S) shows increased number of lysosomes (double white arrows) and dilated rough endoplasmic reticulum (rER). X 10.000.

Fig. 28 : Electron micrograph of trigeminal ganglion, 2 days after tooth extraction, demonstrating degenerated axons (A). The axoplasm contains electron dense bodies of different sizes (white arrows). The myelin shows vacuolation and disorganization (double yellow arrows). X 9.500.

Fig. 29 : Electron micrograph of the trigeminal ganglion one week after tooth extraction, illustrating degenerated nerve cell (N) containing free ribosomes (R) and dilated rough endoplasmic reticulum (rER) displaced at the periphery of the cell as well as numerous lysosomes (arrows). A large macrophage process (M) surrounding the nerve cell and contains vacuoles (V). X 14.000.

Fig. 30 : Electron micrograph of the trigeminal ganglion one week after tooth extraction, demonstrating degenerated nerve cell (N) with shrunken and irregular nucleus (Nu). The cytoplasm contains multiple vacuoles (V) and lysosomes (arrows). X 9.500.

Fig. 31 : Electron micrograph of the trigeminal ganglion one week after tooth extraction, illustrating dark and shrunken degenerated myelinated axon (A). The myelin is completely dissolute and replaced by vacuoles (V) and debris. X 14.000.

Fig. 32 : Electron micrograph of the trigeminal ganglion one week after tooth extraction, illustrating a myelinated axon (A) containing degenerated mitochondria (m). The cytoplasm of the surrounding Schwann cell contains multiple large vacuoles (V). X 14.000

Fig. 33 : Electron micrograph of the trigeminal ganglion two weeks after tooth extraction, showing part of a normal nerve cell (N1) and another degenerating nerve cell (N2) containing multiple vacuoles (V) and a pyknotic nucleus (Nu). The cell is surrounded by a satellite cell (S). X 9,000.

DISCUSSION

The structural changes in sensory ganglion cells following tooth extraction have been the subject of numerous light and electron microscopical studies. Electron microscopic studies showed the existence of retrograde neuronal degeneration in sensory ganglia following tooth extraction (Kubota et al., 1988). Andres (1961) observed cells with nuclear and cytoplasmic changes that were interpreted as signs of cell degeneration.

Fig. 34: Electron micrograph of the trigeminal ganglion two weeks after tooth extraction, illustrating degenerated nerve cell (N). The nerve cell is vacuolated and contains remnants of organelles (arrows). X 12,000.

In the present study, the results revealed that tooth extraction in adult rats induced a retrograde neuronal degeneration in trigeminal ganglion cells. These changes included peripheral displacement of the nucleus, indentation and folding of the nuclear membrane, dilatation of the rough endoplasmic reticulum (rER) and appearance of numerous lysosomal structures. Neurons in the trigeminal ganglia from the experimental side showed alterations similar to those described in previous studies on axot-

omized sensory ganglion cells (Andres, 1961; Pannese, 1963; Kirkpatrick, 1968; Barron et al., 1970; Price and Porter, 1972; Bondok and Sansone, 1984-a).

Similar observations have been previously described in association with the retrograde cell reaction (Pannese, 1963; Barron et al., 1971; Price and Porter, 1972). Apart from all these changes, the present results demonstrated neurons displaying marked degeneration, reflecting stages of inevitable cell death. These neuronal changes were not observed on the control side. The chromatolytic neurons appeared among normal neurons. Changes in satellite cells reflect their high metabolic activity and indirectly suggest the presence of nerve cell damage. The increase in the number of satellite cells in sensory ganglia following peripheral nerve lesions has been previously described (Leech, 1967; Humbertson et al., 1969). It has been reported that the satellite cells play an active role in the metabolic activity of the nerve cells and that materials are transported in both directions between the nerve cells and the satellite cells

(Gawish, 2004) and that the satellite cells act like Schwann cells in phagocytosis of the degenerated nerve cells (Bondok and Gawish, 1994-b).

Light microscopic studies of spinal and trigeminal ganglia have shown a substantial neuronal loss after lesions of peripheral spinal and trigeminal nerve branches and that the incidence of cell loss is highly variable (Galich et al., 1976; Aldskogius and Arvidsson, 1978; Risling et al., 1983; Bondok and Sansone, 1984-a; Ygge and Aldskogius, 1984; Devor et al., 1985; Arvidsson et al., 1986). This variability in the retrograde cell death and the ultimate fate of the axotomized neurons towards either death or regeneration have been attributed to several factors such as age of the animal (Grant et al., 1981 and Devor et al., 1985), axon collateralization (Langford and Coggeshall, 1981) and cell type (Kristensson, 1981).

Neurons undergoing degeneration were surrounded by reactive satellite cells. Pieces of neuronal cytoplasm were segregated inside satellite cells that showed signs of increased lysosomal activity. Aldskogius and Arvids-

son (1978) observed clusters of satellite cells containing debris of degenerated neurons in the trigeminal ganglion after infraorbital nerve transection. Leech (1967) described a conspicuous increase in the number of reactive satellite cells in spinal ganglia following peripheral nerve lesions. He suggested that this might be a reflection of the neuron dependence on satellite cells for their survival.

The results of the present study are of interest from two points of view. First, they extend our knowledge on the ultrastructure of the retrograde neuronal degeneration. Second, they add new information relevant to the reaction of primary sensory neurons to tooth extraction. In this respect, several pathological changes were observed in the majority of the degenerated neurons. The most prominent of these changes were nuclear alterations and dilatation of the cisternae of the RER with dissociation of ribosomal clusters to free single ribosomes and their displacement to the periphery of the cell. Disintegration and vacuolation of cell organelles indicated a stage of "no return" and inevitable cell death. These

neuronal changes were not observed in the control ganglia of the unoperated side.

Remarkable dilatation of the rER and dissociation of ribosomes from polysomes and rER to single ribosomes have been described in degenerating neurons in the facial nucleus of adult mice following facial nerve transection (Torvik and Skjorten, 1971) and in the trigeminal ganglion of adult rats following infraorbital nerve transection (Aldskogius and Arvidsson, 1978) and in the lumbar dorsal root ganglia after sciatic nerve crush in newborn rats (Bondok and Gawish, 1994-a).

Other changes observed in the degenerative process included vacuolation of the cytoplasm, disintegration of the mitochondrial bodies. Previous reports on adult animals have described similar changes after axotomy and have assumed that these changes indicate internal degeneration of the cytoplasmic organelles (Torvik and Skjorten, 1971).

Therefore, it is concluded in this investigation and from the results of

previous investigations that tooth extraction affects the nerve terminals of the trigeminal nerve and induces retrograde changes in trigeminal ganglion cells with degeneration and death of the parent nerve cells and their central branches.

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الملخص العربي

الاضمحلال الراجع فى العقدة العصبية الهلالية بعد خلع الأسنان فى الجرذ الأبيض

عادل عباس بندق - ابراهيم عطية شعبان

رؤوف فكرى بدير - محمد الشا

قسم التشريح ، كلية طب المنصورة

أجريت هذه الرسالة لدراسة تأثير خلع الأسنان على الخلايا العصبية الحسية بالعقدة العصبية الهلالية فى الجرذ الأبيض البالغ. استخدم فى هذا البحث أربعون جرذاً بالغاً من كلا الجنسين وقد تم خلع القاطع السفلى على الناحية اليسرى وتركزت الناحية اليمنى دون تدخل جراحي واستخدمت كضابط للتغيرات المحتملة وفى الوقت المحدد بعد ٢، ٧، ١٤، ٢١، ٢٨ يوماً من خلع الأسنان تم التضحية بالجرذان وأخذت عينات من العقدة العصبية الهلالية والجذر الحسى للعصب الجمجمى الخامس من الناحية الضابطة ومن الناحية التجريبية وتم صبغها بصبغة كريسيل البنفسجى وتولودينى الأزرق وصبغة الفوسفاتيز الحمضى. كما أخذت عينات من العقدة العصبية الهلالية من أعمار ٢، ٧، ١٤ يوماً بعد خلع الأسنان وتم تجهيزها للفحص بالميكروسكوب الإلكتروني.

وبفحص شرائح العقدة العصبية الهلالية المصبوغة بكريزيل البنفسجى وتولودينى الأزرق بالميكروسكوب الضوئى وجدت تغيرات اضمحلالية فى الخلايا العصبية شملت التحلل الصبغى وتغير مكان النواة و لم تلاحظ هذه التغيرات فى الناحية الضابطة. وصبغة الشرائح لإظهار تفاعل الفوسفاتيز الحمضى لوحظ وجود تفاعل قوى فى كل الخلايا فى كل الفترات بعد خلع الأسنان و لم تلاحظ مثل هذه التفاعلات فى الناحية الضابطة.

وبدراسة التركيبات فائقة الدقة للخلايا العصبية والمحاور العصبية بالعقدة العصبية الهلالية بعد خلع الأسنان ظهرت علامات للاضمحلال فى السيتوبلازم مثل إزاحة الريبوسومات لأطراف

الخلايا العصبية وظهور الليسوسومات بأعداد كبيره وانكماش النواة وظهور تعرجات فى غلافها الخارجى. كما ظهرت بعض الخلايا الضامرة محاطة بخلايا آكله. ولوحظ اضمحلال شديد بألياف العصب الجمجمى الخامس.

وقد استخلص من هذا البحث أن خلع الأسنان يؤثر على النهايات الطرفية للعصب الجمجمى الخامس ويحفز حدوث تغيرات اضمحلالية فى خلايا العقدة العصبية الهلالية مما قد يؤدى إلى موت هذه الخلايا وضمور المحاور العصبية بالجذر الحسى للعصب الجمجمى الخامس.