Anatomic Distribution of Gadolinium Contrast Medium by High-Resolution Magnetic Resonance Imaging After Peribulbar and Retrobulbar Injections

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Objective: To examine the anatomic distribution of gadolinium contrast medium by high-resolution surface-coil magnetic resonance imaging after peribulbar and retrobulbar injection.

Methods: Comparative case series in which 4 healthy volunteers were randomized to peribulbar (n = 2) or retrobulbar (n = 2) injection of gadolinium and lidocaine hydrochloride, 2%, without epinephrine. Magnetic resonance imaging was performed before injection and at 5 minutes and 90 minutes after injection.

Results: The peribulbar injection technique resulted in contrast medium primarily in the extraconal space, with no gadolinium observed at the orbital apex; surprisingly, a small amount of contrast medium was observed in the pterygopalatine fossa immediately after peribulbar injection. The retrobulbar injection technique resulted in gadolinium signal diffusely enhancing the intraconal space, orbital apex, optic nerve sheath, and optic canal. The signal intensity was clearly observed in the cavernous sinus surrounding the cavernous portion of the internal carotid artery. A small amount of contrast medium was detected in the pterygopalatine fossa.

Conclusions: The retrobulbar injection technique localizes to the intraconal space, with access to intracranial and central nervous system structures via the optic canal, superior orbital fissure, and cavernous sinus. In contrast, the peribulbar injection technique produces a mostly extraconal distribution; however, intraconal solution may communicate with the central nervous system via the inferior orbital fissure and pterygopalatine fossa. This novel finding suggests that peribulbar anesthesia has a readily accessible route for central nervous system toxic effects. Magnetic resonance imaging with gadolinium contrast medium administration provides an important methodological advantage over previously described techniques and is a safe, reproducible, and superior method of orbital imaging.


Retrobulbar anesthesia, first described by Knapp in 1884 and modified by Atkinson in 1936, is widely used in oculoplastic surgery. The current intraconal technique involves transcutaneous puncture through the temporal lower eyelid, with perforation of the orbital septum, parallel advancement along the globe up to the equator, directional rotation and advancement toward the orbital apex, and perforation of the vagina bulbi. After approximately 38 mm of advancement, aspiration is performed to ensure that the needle is not intravascular and that the anesthetic agent is injected. The retrobulbar injection technique provides good anesthesia and akinesia; however, it carries significant risks, including retrobulbar hematoma, ocular perforation, brainstem anesthesia with cardiac and pulmonary arrest, central nervous system (CNS) toxic effects, direct lesions to the optic nerve, and, rarely, contralateral amaurosis by diffusion of anesthetic through the optic nerve sheaths.

Peribulbar injection is suggested as a safer anesthetic alternative for ophthalmic procedures, in which the injection needle is advanced only until it is parallel to the orbital roof or orbital floor. Although the retrobulbar injection technique provides inferior anesthesia and akinesia, this method is believed to carry lower rates of serious complications, such as retrobulbar hematoma or scleral perforation. Studies indicate that the risk of ocular perforation during peribulbar or retrobulbar injection is approximately 1 in 1000, 1 in 4000, or 1 in 10 000. In highly myopic eyes with an axial length of at least 26.0 mm, the risk of perforation is 10-fold to 250-fold the risk in healthy eyes.

Several groups of researchers have attempted to elucidate the in vivo anatomic differences between peribulbar and...
retrobulbar injections. Winder et al8 used high-resolution ultrasonographic B-scan imaging to demonstrate that retrobulbar injections of local anesthetic accumulate directly in the muscle cone, while peribulbar injections allow spreading of local anesthetic from the orbital fat into the muscle cone. Using computed tomography, Ropo et al9 found that both peribulbar and retrobulbar injections of local anesthetic spread rapidly in the ocular area after injection. Niemi-Murola et al10 used magnetic resonance (MR) imaging to show that local anesthetic spreads mainly around the superior and posterior parts of the globe after peribulbar and retrobulbar injections. Unfortunately, these previous studies were limited by image resolution and methods; the low resolution of B-scan ultrasonography, the poor tissue delineation described by image resolution and methods; the low resolution of B-scan ultrasonography, the poor tissue delineation of computed tomography, and the method of MR imaging subtraction proved to be suboptimal for deciphering the anatomic localization and distribution of local anesthetic after injection.8-10 The aim of the present study was to examine the distribution and localization of a local anesthetic solution mixed with gadolinium contrast medium by MR imaging after peribulbar and retrobulbar injections. To our knowledge, this is the first study to directly inject gadolinium contrast medium with local anesthetic to anatomically define the peribulbar and retrobulbar injection techniques.

METHODS

STUDY DESIGN

The study was conducted in accord with the tenets of the Declaration of Helsinki and all provincial and federal laws and received approval from the Queen’s University, Kingston, Ontario, Canada, research ethics board committee. All individuals gave informed consent for study participation. Four healthy volunteers were randomized to peribulbar (n=2) or retrobulbar (n=2) injection of contrast medium and anesthesia. Exclusion criteria included claustrophobia, allergy to gadolinium contrast medium, the presence of any magnetic metallic implant (eg, cardiac pacemaker, orthopedic implant, and others), or a history of malignant neoplasm, significant systemic disease, or ocular or orbital disease or surgery. These exclusion criteria ensured that the participants had normal ocular and orbital anatomy and would be able to undergo successive MR imaging with gadolinium contrast medium administration.

All the participants received an injection of a 5.0-mL solution containing gadolinium (0.1 mL) mixed with lidocaine hydrochloride, 2% (4.9 mL), without epinephrine. The participants receiving retrobulbar anesthesia experienced transitory extraocular motility disturbances, diplopia, and variable ptosis. Magnetic resonance imaging was performed at baseline, about 5 minutes after injection, and approximately 90 minutes after injection. No adverse events or complications were observed, and all the participants had resolution of symptoms before discharge home at the conclusion of the study. No adverse events or toxic effects occurred from the administration of gadolinium directly into the orbit.

RESULTS

PARTICIPANT CHARACTERISTICS AND DEMOGRAPHICS

Four healthy volunteers (3 male and 1 female) of comparable age (age range, 24-28 years) were enrolled in our study. In all the participants, baseline imaging revealed normal orbital anatomy, and no individuals were excluded from the study. All the participants received injections consisting of a solution of gadolinium (0.1 mL) mixed with lidocaine hydrochloride, 2% (4.9 mL), without epinephrine. The participants receiving retrobulbar anesthesia experienced transitory extraocular motility disturbances, diplopia, and variable ptosis. Magnetic resonance imaging was performed at baseline, about 5 minutes after injection, and approximately 90 minutes after injection (mean, 103.25 minutes, range, 100-105 minutes) after injection. No adverse events or complications were observed, and all the participants had resolution of symptoms before discharge home at the conclusion of the study. No adverse events or toxic effects occurred from the administration of gadolinium directly into the orbit.

PERIBULBAR INJECTION TECHNIQUE

At 5 minutes after peribulbar injection, the solution remained primarily extracanal between the lateral rectus and orbital wall (Figure 1A and B). Most signal intensity was observed in the extracanal space (predominantly laterally and inferiorly), with near-complete...
Figure 1. Axial T1-weighted fat-saturated (A and B), coronal T1-weighted fat-saturated (C and D), and axial T1-weighted fat-saturated (E and F) magnetic resonance images at 5 minutes (E) and 90 minutes (F) after peribulbar injection. A and B. The signal is initially seen in the extraconal space between the lateral rectus (LR) and orbital wall, with elimination of the signal by 90 minutes. C and D. Most signal intensity is seen in the extraconal space laterally and inferiorly, with reduced intensity by 90 minutes. E and F. A small amount of contrast medium extends into the pterygopalatine fossa (PF) via the inferior orbital fissure (arrowheads).
clearance of contrast medium by the second postinjection MR image (Figure 1C and D). At no point was any signal intensity observed in the optic nerve sheath, posterior margin of the globe, or orbital apex. Notably, a small signal intensity was seen in the inferior intracranial space. The signal extended through the inferior orbital fissure and into the pterygopalatine fossa (Figure 1E and F). These findings were consistently observed in both participants receiving retrobulbar injections.

**RETOBULBAR INJECTION TECHNIQUE**

The retrobulbar injection technique produced dramatic signal intensity in the intracranial space (laterally greater than medially) at 5 minutes after injection, with contrast medium visible at the orbital apex (lateral aspect) and optic canal (Figure 2A and B). The intracranial signal intensity includes the area surrounding the optic nerve sheath and can be seen beyond the optic nerve and optic canal surrounding the internal carotid artery within the cavernous sinus (Figure 2A and B). In the cavernous sinus, the signal intensity is predominantly present surrounding the cavernous portion of the internal carotid artery, both at 5 minutes and 90 minutes after injection. Gadolinium was freely distributed within the intracranial structures, posterior aspect of the globe, and lateral intracranial space; this was significant even at 90 minutes after injection (Figure 2C and D). The axial MR images show faint signal intensity in the pterygopalatine fossa immediately after retrobulbar injection, with an increase in intensity at 90 minutes (Figure 2E and F). These findings were consistently observed in both participants receiving retrobulbar injections.

**COMMENT**

We present a small case series in which direct orbital gadolinium injection outlined important differences in the anatomic distribution of fluids using peribulbar vs retrobulbar injection techniques. Magnetic resonance imaging with contrast medium administration provides an important methodological advantage over previously described techniques in elucidating these anatomic relationships. To our knowledge, this is the first study to directly inject gadolinium into the orbit to improve image acquisition and anatomic localization. Peribulbar and retrobulbar injections of gadolinium contrast medium were well tolerated, with no adverse events or toxic effects noted.

The peribulbar injection technique produces an anatomic distribution primarily in the extraconal space. The signal intensity is predominantly found in the lateral and inferior extraconal space, with no significant distribution into the optic nerve sheath or orbital apex. In contrast, anesthetic administration via the retrobulbar injection technique results in localization of solution in the intraconal space; the lateral and medial distribution within the intracranial space after retrobulbar injection differs from the lateral and inferior accumulation after peribulbar injection. The ability to introduce anesthetic solution into the intraconal space allows it to reach the optic nerve sheath, optic nerve, optic canal, superior orbital fissure, and cavernous sinus.

Known complications of retrobulbar injection include brainstem anesthesia and CNS toxic effects; despite this, the exact route has largely remained unknown. We demonstrate that the retrobulbar injection technique allows the anesthetic solution to travel from the orbital apex, via the optic nerve and optic canal, through the superior orbital fissure to the cavernous sinus. In addition, we show direct evidence of the signal intensity surrounding the cavernous portion of the internal carotid artery within the cavernous sinus. This administration route allows communication between the intracranial space and intracranial CNS structures, present both immediately after injection and at 90 minutes after injection. The anatomic distribution into the CNS via the optic canal and superior orbital fissure is the likely route that results in brainstem anesthesia and CNS toxic effects after retrobulbar injection.

It was previously believed that peribulbar injections, because they remain parallel to the orbital floor or orbital roof, do not gain access to the intracranal space and do not carry the inherent risk of CNS toxic effects associated with retrobulbar injections. Surprisingly, we found this to be untrue. The presence of a small signal intensity within the intracranial space was observed immediately after peribulbar injection in both study participants using this technique. This suggests that, via the pterygopalatine fossa, anesthetic solution has a possible route into the CNS and neck. The pterygopalatine fossa contains the pterygopalatine ganglion, with nerve roots from the maxillary nerve (the second division of the trigeminal nerve) and the terminal third of the maxillary artery (in addition to the seventh cranial nerve, which joins with the tenth cranial nerve to the tongue). Direct access to the CNS, via the pterygopalatine fossa, is possible posteriorly via the foramen rotundum into the middle cranial fossa or via the pterygoid canal into the middle cranial fossa and foramen lacerum.

We propose that the route by which peribulbar injections may gain access to the CNS begins with the distribution into the intracranial space, likely inferiorly and laterally, which is then able to access the inferior orbital fissure and finally the pterygopalatine fossa. We see a predilection of the retrobulbar injection technique for CNS access via the superior orbital fissure. These findings would not be seen on ultrasonography because the pterygopalatine fossa is too deep for any meaningful resolution to be obtained with that imaging method. Similarly, computed tomography would be severely obstructed by orbital bone signal to allow localization of the anesthetic solution. Finally, the method of MR imaging subtraction would likely miss this because the signal would have been too weak for any appreciable detection sensitivity. Only with direct administration of gadolinium are we able to visualize the kinetics of the anesthetic solution in the orbital spaces after injection.

Although the predominant access point into the CNS after retrobulbar injection is via the superior orbital fissure and cavernous sinus, a small signal intensity was observed in the pterygopalatine fossa at 90 minutes after injection. This leads us to postulate that retrobulbar anesthesia likely has 2 routes of CNS access, primarily via the superior orbital fissure into the cavernous sinus but...
Figure 2. Axial T1-weighted fat-saturated (A and B), coronal T1-weighted fat-saturated (C and D), and axial T1-weighted fat-saturated (E and F) magnetic resonance images at 5 minutes (E) and 90 minutes (F) after retrobulbar injection. A, The signal intensity is seen in the intraconal space and extends posteriorly through the superior orbital fissure into the cavernous sinus (arrows), where it surrounds the cavernous portion of the internal carotid artery (arrowheads). B, The signal intensity at 90 minutes is concentrated intraconally and is seen surrounding the optic nerve (arrows). C and D, At 90 minutes, the signal intensity persists in the intraconal space; the optic nerve and surrounding cerebrospinal fluid (arrow) and the ophthalmic artery (arrowhead) are shown. E and F, A trace amount of contrast medium is seen in the pterygopalatine fossa (PF [arrowhead]) at 5 minutes after injection (E), with an increase in the signal intensity at 90 minutes (F).
also via the inferior orbital fissure into the pterygopala-
tine fossa. Clinically, this could be tested by asking the
patient about any numbness or loss of taste in the tongue
because this sensory taste pathway is transmitted through
the pterygopalatine ganglion.

Our study is limited by its lack of clinical data. For
example, no data were collected on extraocular motility
or the degree of anesthesia after injection; indeed, the only
clinical monitoring was for adverse events or toxic ef-
fec ts, none of which were observed in our study. With-
out the collection of clinical end points, we do not know
the degree of effect that the 2 different injection tech-
niques produced in each of the tested participants. Our
study was primarily designed as an anatomic investiga-
tion and not as a clinical study to evaluate the differ-
ences between peribulbar and retrobulbar methods of an-
esthesia. We believe that the latter has been adequately
researched and published in the literature. In addition,
our study relies on a small group of healthy volunteers,
and larger studies may reveal further anatomic details that
we have not analyzed. Although we are confident that
we were able to reproduce relevant findings in the in-
vigated participants, we welcome further study and
analysis of this relevant anatomic challenge.

This study illustrates the anatomic localization and dis-
btribution of anesthetic solution with gadolinium en-
hancement after the peribulbar and retrobulbar injec-
tion techniques. We demonstrate that the retrobulbar
injection technique localizes solution to the intraconal
space, with CNS access via the optic canal, superior or-
bital fissure, and cavernous sinus. In contrast, the peri-
bulbar injection technique produces a mostly extra-
conal distribution; however, a small amount of intraconal
solution may communicate with the CNS via the infe-
rior orbital fissure and pterygopalatine fossa. This novel
finding suggests that, contrary to popular belief, perib-
ulbar anesthesia has a readily accessible route for CNS
toxic effects. Direct administration of gadolinium into the
orbit is safe and, when combined with MR studies, is a
superior method of orbital imaging.

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