CLINICAL REPORTS

Ronald D. Miller, M.D., Editor

Anesthesiology
63:688–690, 1985

Brain Stem Anesthesia Following Retrobulbar Blockade

ROBERT C. HAMILTON, M.B., F.R.C.P.(C), F.F.A.R.C.S.(I)*

Retrobulbar block with local anesthetic drugs is widely used for ophthalmic surgery of various types.[1] Several reports of brain stem anesthesia following this and other head and neck blocks have appeared in the literature.[2–7]

In a busy outpatient ophthalmic surgery practice, this author has seen brain stem anesthesia from central spread from retrobulbar injection of local anesthetic drugs, presumably by accidental injection beneath the sheath of the optic nerve, on three occasions in 1,500 consecutive cases. The reported findings in the published cases and those in this author's three cases are discussed and management options suggested as to how to avoid this problem.

REPORT OF THREE CASES

Case 1. A 54-yr-old, 65-kg man was scheduled for a right cataract extraction with intraocular lens implantation. He had mild arterial hypertension and stable angina and was receiving no regular medications. Without sedation and while reclining in a dental-type chair raised 20 degrees from the horizontal position, a right retrobulbar block was performed with 4 ml of a solution with after-mixing concentrations of 2.4% lidocaine, 0.15% bupivacaine, hyaluronidase 20 units/ml, and epinephrine 1:200,000 (3:1 mixture of 4% lidocaine and 0.75% bupivacaine with added hyaluronidase and epinephrine), followed by an Atkinson[8] block of the periorbital musculature using 8 ml lidocaine 2% with 1:200,000 epinephrine. The time sequence of changes is shown in figure 1. Five minutes after the block he complained of difficulty with swallowing and of having a "numb" throat. The patient remained calm and cooperative; his recumbent position was maintained. Ten minutes after the block he complained of difficulty in breathing and a decline of hearing ability. Oxygen was given via a mask. Communication with the patient was easily maintained in spite of his drowsiness and hearing impairment. After 1 h he had fully recovered. A repeat retrobulbar block using 3.0 ml of the original mixture was by this time necessary because some activity had returned to the extraocular muscles. The proposed surgery then was completed without further incident.

Case 2. An 84-yr-old, 60-kg woman was scheduled for a right cataract extraction with intraocular lens implantation. She had moderate arterial hypertension controlled with methyldopa and hydrochlorothiazide. Without sedation her regional anesthesia was administered identically to that in case 1. Two minutes after the retrobulbar block the patient lapsed into unconsciousness over a period of about 20 s and became apneic. The events and drug management are shown in figure 2. Respiration was controlled using oxygen and face mask for 20 min. After her recovery from this complication, surgery was completed without further incident.

Case 3. An 81-yr-old, 55-kg woman was scheduled for a right cataract extraction with intraocular lens implantation. She had mild arterial hypertension for which she was receiving no medication. Her regional anesthesia was managed identically to the above two cases. Two minutes after the retrobulbar block she complained of sleepiness and lapsed into unconsciousness and apnea 5 min later. The events and drug management are shown in figure 3. Respiration was controlled using oxygen and face mask for 20 min. The proposed surgery was carried out after repeat retrobulbar blockade.

DISCUSSION

The rationale for the relatively high concentration of local anesthetic drugs used by the author is to achieve total akinesia with a low volume of injectate. Higher volumes may result in less desirable conditions for the surgeon. Bupivacaine is included in the retrobulbar block to allow a slower return of sensation and muscle movement than if lidocaine alone were used, thus promoting a more restful state of the eye for the immediate postoperative period. Hyaluronidase, in the concentration used, aids the even spread[9] of anesthesia within the orbit. Epinephrine in 1:200,000 dilution delays systemic release of the local anesthetic agents[9] and reduces the tendency for retrobulbar hemorrhage.

The only supportable explanation of the phenomena in the above reported cases is the injection of local anesthetic drug beneath the sheath of the optic nerve. The eye, being developmentally part of the brain, has extensions of the meninges covering the optic nerve, and it is logical that solution injected beneath the optic nerve sheath can track centrally into the subarachnoid or subdural space. This has been demonstrated radiologically by Lombardi.[10]

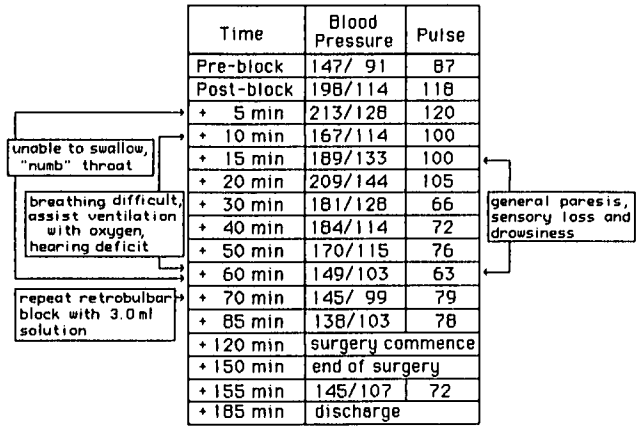
In the more common circumstances of high cerebrospinal fluid levels resulting from unplanned subarachnoid injection of large doses of local anesthetic drug in the lumbar spine area, invariably there are signs of sympa-

* Clinical Associate Professor of Anaesthesia, University of Calgary, Specialist Anaesthetist, Gimbel Eye Centre.

Received from the Department of Anaesthesia, University of Calgary, Calgary, Alberta, Canada, and Gimbel Eye Centre, Suite 450, 4935, 40th Avenue, N. W., Calgary Alberta, Canada. Accepted for publication July 1, 1985.

Address reprint to Dr. Hamilton: c/o Gimbel Eye Centre, Suite 450, 4935 40th Avenue, N.W., Calgary, Alberta, Canada T3A 2N1.

Key words: Anesthetic techniques: regional; retrobulbar. Complications: brain stem anesthesia.



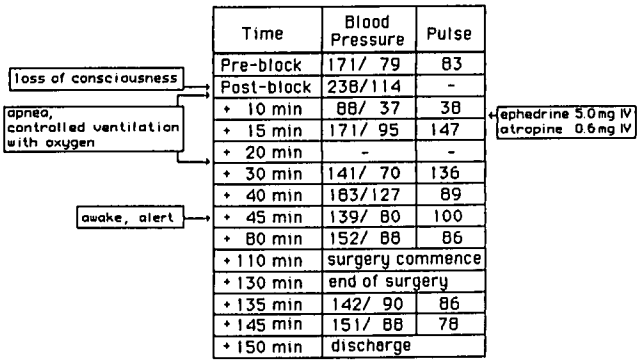
Time	Blood Pressure	Pulse
Pre-block	147/ 91	87
Post-block	198/114	118
+ 5 min	213/128	120
+ 10 min	167/114	100
+ 15 min	189/133	100
+ 20 min	209/144	105
+ 30 min	181/128	66
+ 40 min	184/114	72
+ 50 min	170/115	76
+ 60 min	149/103	63
+ 70 min	145/ 99	79
+ 85 min	138/103	78
+ 120 min	surgery commence	
+ 150 min	end of surgery	
+ 155 min	145/107	72
+ 185 min	discharge	

FIG. 1. Changes in vital signs following the retrobulbar nerve block in Case 1.

thetic block as evidenced by arterial hypotension and bradycardia, which may be followed by apnea (intercostal paralysis preceding diaphragmatic paralysis) and loss of consciousness and cranial nerve palsy from cephalad spread, if critical levels of drug are reached. In an earlier era,[11] low-concentration local anesthetic drug levels were deliberately induced in cerebrospinal fluid with retention of spontaneous respiration.

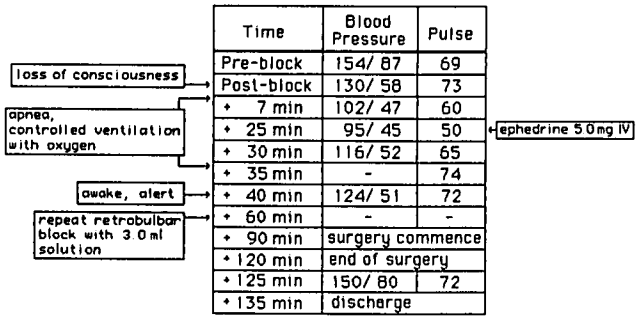
In the more unusual circumstances under consideration in this report, in which the local anesthetic drugs enter the cerebrospinal fluid at the cranial end of the neuraxis, parasympathetic blockade may be more in evidence initially; sympathetic blockade, along with loss of consciousness and apnea may follow. As in the first case reported above, diaphragmatic respiration will be affected before intercostal respiration.

Sustained hypertension and tachycardia were common in the series[2-7] (table 1) (including the report of Rosenblatt *et al.*[4] in which these signs were present after a short period of cardiac asystole) and did not bear any relationship to the presence or absence of epinephrine in the injectate. Early physiologists were aware of the profound influence



Time	Blood Pressure	Pulse
Pre-block	171/ 79	83
Post-block	238/114	-
+ 10 min	88/ 37	38
+ 15 min	171/ 95	147
+ 20 min	-	-
+ 30 min	141/ 70	136
+ 40 min	183/127	89
+ 45 min	139/ 80	100
+ 80 min	152/ 88	86
+ 110 min	surgery commence	
+ 130 min	end of surgery	
+ 135 min	142/ 90	86
+ 145 min	151/ 88	78
+ 150 min	discharge	

FIG. 2. Changes in vital signs and response to therapy in Case 2.



Time	Blood Pressure	Pulse
Pre-block	154/ 87	69
Post-block	130/ 58	73
+ 7 min	102/ 47	60
+ 25 min	95/ 45	50
+ 30 min	116/ 52	65
+ 35 min	-	74
+ 40 min	124/ 51	72
+ 60 min	-	-
+ 90 min	surgery commence	
+ 120 min	end of surgery	
+ 125 min	150/ 80	72
+ 135 min	discharge	

FIG. 3. Changes in vital signs and response to therapy in Case 3.

of the vagus nerve on the heart and first called the nerve the depressor nerve because the bilateral section of the vagi in ex-perimental animals resulted in hypertension and tachycardia. Nique and Bennett[5] explained the hypertension and tachycardia in their patient by implicating vagal blockade at the brain stem. This is logical but does not fully account for the sustained nature of the increase in arterial blood pressure that would normally settle with carotid baroreceptor reflex adjustment. However, in the situation under consideration, the glossopharyngeal nerve rootlets were presumably affected, resulting in abolition of the carotid sinus reflex. In the first case reported above the initial symptom was swallowing difficulty, indicating early glossopharyngeal involvement.

In all of the reported cases there was a successful outcome, which followed appropriate life support measures. It behooves all personnel involved in nerve blocks with this potential to be fully conversant with techniques and equipment used in resuscitation of patients caught in this life-threatening situation. Precise knowledge of anatomy is desirable in achieving a low incidence of this type of

TABLE 1. Responses to Retrobulbar Block

	Apnea and Loss of Consciousness	Blood Pressure	Pulse Rate
Kepes and Foldes[2]	No	↑	
Whitehurst and Harrelson[3]	Yes	↓	
Rosenblatt *et al.*[4]	Yes	See text	See text
Nique and Bennett[5]	Yes	↑	↑
Smith (>8 cases)[6]	Yes		
Chang *et al.*[7]	Yes	↑	↑
Hamilton (case 1)	Dyspnea and drowsiness	↑	↑
Hamilton (case 2)	Yes	↑ then ↓	↓
Hamilton (case 3)	Yes	↓	↓

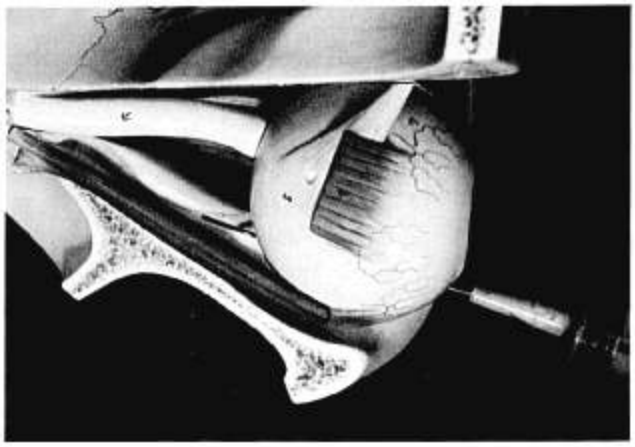


FIG. 4. Photograph of model of right orbit as seen from above. The roof of the orbit and the belly of the superior rectus muscle have been removed. *1.* Insertion of superior rectus muscle. *5.* Insertion of superior oblique muscle. *16.* Optic nerve. The needle is seen lying inferior to the globe, its tip within the extraocular muscle cone, medial to the lateral rectus muscle and well away from contact with the optic nerve.

complication. In 1,000 subsequent cases of retrobulbar blockade, this author has used a modified technique and has not had the complication of brain stem anesthesia. Figure 4 illustrates the modified technique; the needle enters the inferior orbit in the saggital plane of the lateral limbus and remains at all times in that plane. The final position of the needle tip is within the extraocular muscle cone, medial to the lateral rectus muscle and well away from the optic nerve.

The author acknowledges the assistance of Professors Thomas J. Harrison and Ian C. Roddie.

REFERENCES

1. Backer CL, Tinker JH, Robertson DM, Vlietstra RE: Myocardial reinfarction following local anesthesia for ophthalmic surgery. Anesth Analg 59:257–262, 1980
2. Kepes ER, Foldes FF: Transient abducens paralysis following therapeutic nerve blocks of head and neck. ANESTHESIOLOGY 38:393–394, 1973
3. Whitehurst L, Harrelson J: Brain stem anesthesia: An unusual complication of stellate ganglion block. J Bone Joint Surg 59A: 541–542, 1977
4. Rosenblatt RM, May DR, Barsoumian K: Cardiopulmonary arrest after retrobulbar block. Am J Ophthalmol 90:425–427, 1980
5. Nique TA, Bennett CR: Inadvertent brain stem anesthesia following extra-oral trigeminal V_2–V_3 blocks. Oral Surg 51:468–470, 1981
6. Smith JL: Retrobulbar marcaine can cause respiratory arrest. J Clin Neuroophthalmology 1:171–172, 1981
7. Chang J-L, Gonzalea-Abola E, Larson CE, Lobes L: Brain stem anesthesia following retrobulbar block. ANESTHESIOLOGY 61: 789–790, 1984
8. Atkinson WS: Observations on anesthesia for ocular surgery. Trans Am Acad Ophthal Otolaryngol 60:376–378, 1956
9. Wood-Smith FG, Vickers MD, Stewart HC: Drugs in anaesthetic practice, fourth edition. London, Butterworths, 1973, p 465
10. Lombardi G: Radiology in neuroophthalmology. Baltimore, Williams and Wilkins, 1967, pp 6–8
11. Griffiths HWC, Gillies J: Total spinal analgesia. Anaesthesia 3: 134–145, 1948

Anesthesiology
63:690–692, 1985

Nebulized Anesthesia for Awake Endotracheal Intubation

DENIS L. BOURKE, M.D.,* JEFFREY KATZ, M.B., Ch.B.,† ALAN TONNESON, M.D.‡

Awake endotracheal intubation is frequently the safest approach for patients with problems such as unusual airway anatomy, facial or neck trauma, or cardiovascular instability. Several authors have described methods to anesthetize the airway for awake intubation.[1–3] Most of these methods require several steps and can be uncomfortable for the patient. We describe a single technique that requires the use of an aerosol of local anesthetic for anesthetizing the entire nasotracheal airway and illustrate its usefulness with a case presentation. Our technique provides complete anesthesia for nasotracheal intubation and is painless, reliable, and simple to perform.

* Associate Professor of Anesthesiology, Boston University Medical School.

† Assistant Professor of Anesthesiology, University of Texas Medical School at Houston.

‡ Associate Professor of Anesthesiology, University of Texas Medical School at Houston.

Received from the Department of Anesthesiology, Boston University Medical School, Boston, Massachusetts, and the Department of Anesthesiology, University of Texas Medical School at Houston, Houston, Texas. Accepted for publication July 1, 1985.

Address reprint requests to Dr. Bourke: Department of Anesthesiology, University Hospital, 75 East Newton Street, Boston, Massachusetts 02118.

Key words: Anesthetics, local: lidocaine. Intubation, endotracheal: technique.

REPORT OF A CASE

A 32-year-old woman was involved in a motor vehicle accident that resulted in an open laceration of the right eye, a right zygomatic fracture, and a left femoral fracture. Cervical spine roentgenograms could not rule out a C-5 fracture. A cervical collar was applied, and the patient was scheduled for emergency surgery. Our preoperative ex-