Ophthalmic Surgery

Fourth Edition

Edited by
Thomas A. Rice
Ronald G. Michels
Walter J. Stark

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The C V Mosby Company St Louis Toronto

Butterworths London Boston Durban Singapore Sydney Toronto Wellington

ISBN 0-407-00657-5

First edition published in eight volumes 1956–1958
Second edition published in fourteen volumes 1968–1971
Third edition published in nineteen volumes 1976–1981
Fourth edition published 1984

Library of Congress Cataloging in Publication Data

Main entry under title:
Rob & Smith's operative surgery.
Rev. ed. of: Operative surgery / under the general editorship of Charles Rob and Rodney Smith; associate editor, Hugh Dudley. 3rd ed. 1976–
Includes bibliographies and indexes.
Contents: [1] Alimentary tract and abdominal wall / edited by Hugh Dudley--[2] The hand / edited by Rolfe Birch, Donal Brooks--[3] Ophthalmic surgery / edited by Thomas A. Rice, Ronald G. Michels, Walter J. Stark.
1. Surgery, Operative, I. Dudley, Hugh Arnold Freeman. II. Pories, Walter J. III. Carter, David C. (David Craig) IV. Rob, Charles. V. Smith of Marlow, Rodney Smith, Baron, 1914–
VI. Operative surgery. [DNLM: 1. Hand--Surgery. W0 500 061 1982 v.7]
RD32.R6 1983 617'.91 83-14465
ISBN 0-407-00651-6 (v. 1)

Photoset by Butterworths Litho Preparation Department
Printed by Blantyre Printing & Binding Co, Ltd, Glasgow, Scotland

Ophthalmic Surgery

Fourth Edition

Ophthalmic Surgery is a volume from

Rob & Smith's

Operative Surgery

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Preface

Ophthalmic surgery is one of the most interesting and technically demanding surgical disciplines, and there have been important advances during the past two decades because of expanding scientific knowledge, new operative techniques and improved instrumentation. Surgical methods have grown from a limited number of operations for conditions such as cataract, glaucoma and strabismus to a variety of refined techniques for improved treatment of these conditions and also effective management of other disorders for which no treatment methods were available before.

The purpose of this text is to describe current surgical methods and their role in treatment of various ocular disorders. We present the general principles of ophthalmic surgery and also the details of technique. In addition, each subject is organized to include information on the preoperative evaluation, indications for surgery, results, complications and postoperative management.

Each chapter deals with a different type of ophthalmic surgery and is written by a surgeon or surgeons specializing in that area. Although little of the material is original, the techniques described do reflect recent advances and the personal preferences of the authors, and they are illustrated with detailed drawings. Space limitations often prevent thorough presentation of alternative techniques, although these are frequently mentioned.

At a time when subspecialization in ophthalmology has resulted in a fragmentation of the ophthalmic literature, our intent is to provide a single, reasonably comprehensive source reflecting the current philosophies and methods of ophthalmic surgery. We believe now is an opportune time to compile this work because, in many areas, the rapid advances in surgical techniques of the past two decades have become more established. Some of the new procedures require expensive, specialized equipment or advanced surgical training, but often there are no equally effective, simpler alternatives.

We realize that some of the current methods presented will become outmoded. Even so, we present those approaches we now believe must have merit, and we hope that awareness of our present limitations in ophthalmic surgery will stimulate development of more effective methods to investigate and treat ocular diseases.

The editors gratefully acknowledge: (*1*) the capable and steadfast assistance of David Andrews of the Wilmer Institute in editing each of the chapters; (*2*) the exceptional skill and dedicated cooperation of the artists Timothy Hengst and Nadine Sokol in the preparation of the beautiful illustrations; and (*3*) the special assistance of Isabel Deleon, MD, Assistant Professor of Anesthesia, The Johns Hopkins University School of Medicine, for her scholarly review of the chapter dealing with anesthesia.

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Ocular anesthesia

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Introduction

The special needs of intraocular surgery are naturally the major consideration in any discussion of ocular anesthesia. This is because the eye is particularly vulnerable to damage, and the outcome of surgery may be influenced by anesthetic events. This chapter discusses general and local anesthesia for eye surgery, and the special requirements for intraocular and some extraocular procedures. The requirements for intraocular procedures are best understood by a consideration of the anatomy and physiology of the eye.

General anatomy and physiology

The eye may be considered as a fluid-filled semirigid sphere created by the corneoscleral envelope. Internally it is divided into two major compartments. The anterior chamber, containing aqueous humor, lies between the cornea anteriorly and the iris posteriorly. The iris and lens form a diaphragm between the anterior chamber and the vitreous, the largest compartment of the eye. The iris together with the ciliary body and choroid make up the uveal tract, the major vascular compartment of the globe. Choroidal thickness has been estimated to be 500–1000 μm[1,2]. Internal to the choroid lies the retina, whose axons leave the globe as the optic nerve.

Blood supply of the globe

Arterial supply

The blood supply to the globe is entirely from the ophthalmic artery, which supplies the central retinal artery and the long and short posterior ciliary arteries of the uveal tract (*Figure 1.1*). Ninety per cent of the blood flow of the eye passes through the uveal tract[3]. Blood flow through the choroid is very rapid; and despite the fact that the

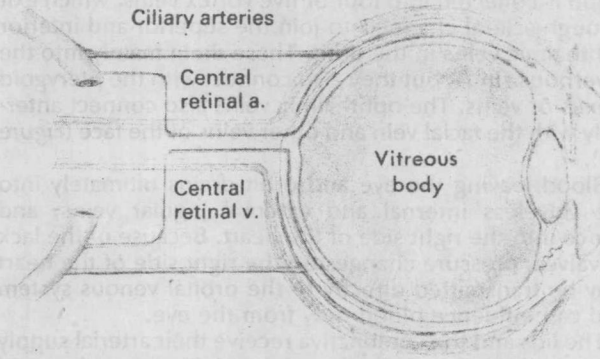

Figure 1.1 *Ocular anatomy and blood supply. Central portion of the posterior segment is occupied by the vitreous body. Arterial blood is provided by ciliary arteries supplying the choroid and the central retinal artery supplying the retinal circulation. Venous drainage from the retina is through the central retinal vein and choroidal drainage is through the vortex veins*

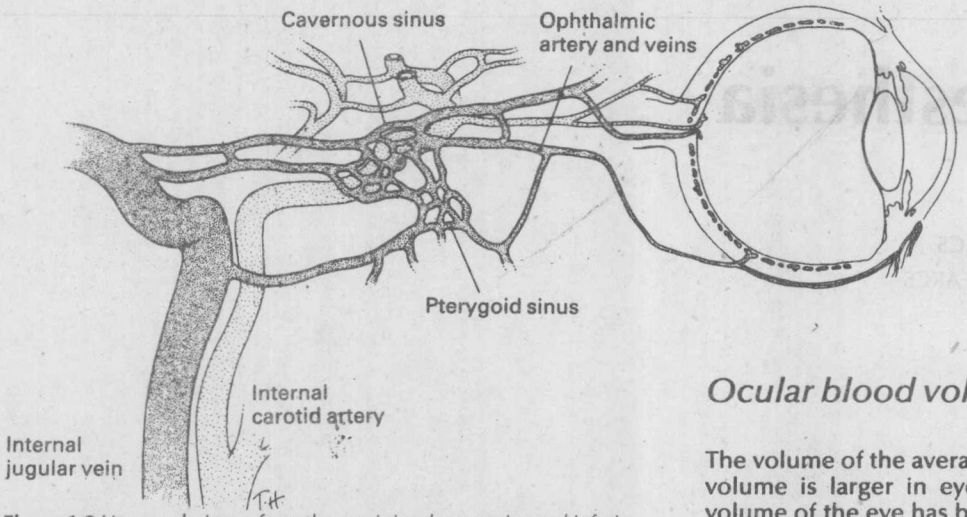

Figure 1.2 *Venous drainage from the eye joins the superior and inferior ophthalmic veins and then flows into the cavernous sinus and the pterygoid plexus. Blood leaving the eye and orbit drains into the valveless jugular veins so that pressure changes in the right side of the heart are transmitted directly to the eye*

choroid supplies the outer 60 per cent of the retina[4], the choroidal venous blood maintains a high level of oxygen saturation[5]. The responsiveness of the choroidal and retinal vasculatures to physiological changes in arterial oxygen tension (Pa_{O_2}) are different. Hypoxia leads to vasodilatation in the retinal vessels[6] but not the choroid[7]. Elevated Pa_{O_2} leads to slight retinal arteriolar vasoconstriction with reduced blood flow[8], but has little effect on the choroidal circulation[7].

Venous drainage

Venous drainage of the retina and choroid is ultimately into the ophthalmic veins of the orbit, but within the eye the venous drainage is separate. The retinal tributary veins combine to form the central retinal vein, which crosses the subarachnoid space within the sheath of the optic nerve before exiting into the orbit. Choroidal venous blood is collected into four or five vortex veins, which exit through scleral channels to join the superior and inferior ophthalmic veins in the orbit. These drain mainly into the cavernous sinus, but they also connect with the pterygoid plexus of veins. The ophthalmic veins also connect anteriorly with the facial vein and other veins of the face (*Figure 1.2*).

Blood leaving the eye and orbit drains ultimately into the valveless internal and external jugular veins, and hence into the right side of the heart. Because of the lack of valves, pressure changes on the right side of the heart may be transmitted directly to the orbital venous system and can influence blood flow from the eye.

The lids and the conjunctiva receive their arterial supply from the branches of the internal and the external carotid artery. Venous drainage is mainly into the veins of the face, although the perilimbal bulbar veins drain partly into the episcleral venous plexus and then posteriorly into the ophthalmic veins of the orbit. This plexus also receives the aqueous humor outflow from the eye.

Ocular blood volume and flow

The volume of the average adult eye is about 6 ml, and the volume is larger in eyes with axial myopia. The blood volume of the eye has been estimated at between 200 and 300 μl[9]. Total ocular blood flow in man has not been measured, but in the dog a value of 680 μl/min has been found[10]. This represents a high blood flow perfusing the retina, equivalent to that perfusing the brain[5,11]. Because the uveal and retinal vascular systems are contained within the corneoscleral envelope, acute fluctuations in blood flow rapidly influence vascular volume and indirectly affect the intraocular pressure. However, the long-term homeostasis of intraocular pressure is determined by another fluid system, concerned with production and drainage of aqueous humor.

Ocular pressure and aqueous humor dynamics

Aqueous humor is formed by the ciliary body and circulates from the posterior chamber through the pupil into the anterior chamber. It leaves the anterior chamber through the filtering meshwork of the trabeculum and then Schlemm's canal, passing thence into the episcleral venous system. These veins connect with the ophthalmic veins of the orbit (*Figure 1.3*).

The aqueous humor volume is about 200 μl. Aqueous flow in man is about 1 per cent of the aqueous volume per minute, and thus about 2 μl/min[12]. By comparison, the blood flow of the eye is probably 400 times as great. This

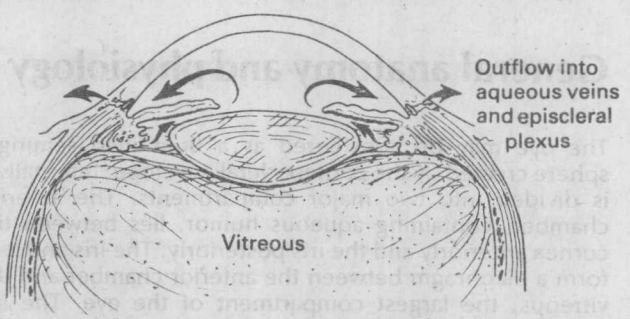

Figure 1.3 *Formation and drainage of aqueous humor. Aqueous humor is produced by the non-pigmented epithelium of the ciliary processes and passes from the posterior chamber, through the pupillary space, into the anterior chamber. It then drains out through the trabecular meshwork and canal of Schlemm into the episcleral venous plexus*

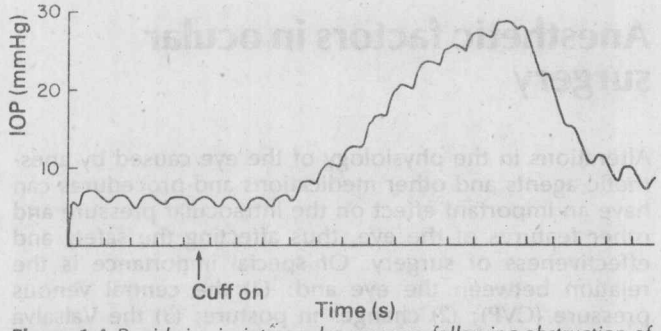

Figure 1.4 *Rapid rise in intraocular pressure following obstruction of venous drainage by inflation of a cuff around the neck*

difference in flow of the two systems results in part from the very different perfusion pressures acting in the aqueous and the vascular compartments; and it implies that obstruction of flow – for example, by totally obstructing either the aqueous or venous drainage – will produce very different effects. Obstruction of aqueous outflow will produce a comparatively slow rise in intraocular pressure, and obstruction of venous return will produce a rapid rise (*Figure 1.4*).

Perfusion pressures in the eye

Ocular fluid flow is determined by pressure gradients. In the case of aqueous flow, the 'upstream' pressure is the mean ocular pressure (e.g. 15 mmHg), and the 'downstream' pressure is that in the episcleral veins into which the aqueous drains (e.g. 9 mmHg). The usual pressure gradient for aqueous flow is therefore about 15 minus 9 = 6 mmHg (*Figure 1.5*).

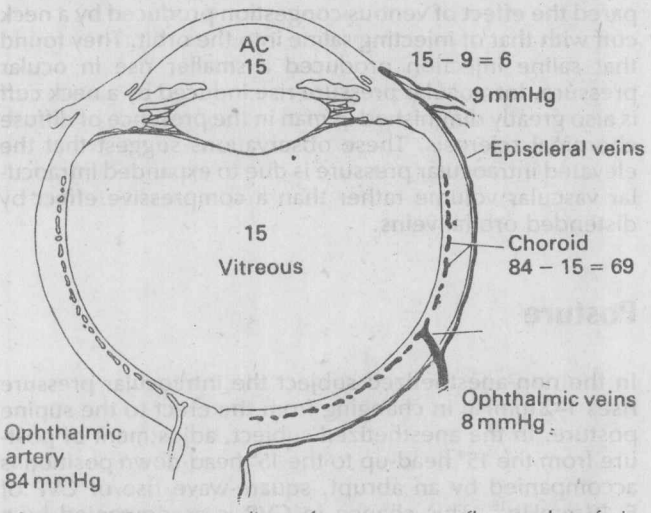

Figure 1.5 *Pressure gradients for aqueous outflow and perfusion pressure of the vascular compartment. Pressure gradient for aqueous outflow is low and is the difference between the intraocular pressure (15 mmHg) and the episcleral venous pressure (9 mmHg). The perfusion pressure of the vascular compartment is high and is the difference between the mean arterial pressure (84 mmHg) and the intraocular pressure (15 mmHg)*

The upstream pressure for vascular flow in the eye is probably the mean arterial pressure in the ophthalmic artery. This varies with posture, but it is about 84 mmHg in the supine posture in the awake subject[13,14]. The downstream pressure is mainly determined by the intraocular pressure, since the intraocular venous pressure (whether retinal or uveal) must be higher than the intraocular pressure to maintain patency of the veins. Therefore, the downstream pressure is about 15 mmHg, and the perfusion pressure in the vascular compartment is about 84 minus 15 = 69 mmHg.

The difference in perfusion pressure is the major factor determining the flow difference between the aqueous and vascular compartments. Changes in aqueous production or drainage are likely to involve such small changes in intraocular pressure, in a short period of time, that they would not be expected to cause significant clinical effects during anesthesia. However, vascular changes may produce rapid and wide fluctuations in intraocular pressure because of changes in ocular vascular volume within the corneoscleral envelope.

Another result of the difference in arterial and venous pressures is that changes in arterial and venous vascular volume do not affect the intraocular pressure in the same way. The systemic arterial pulse pressure is directly related to the ocular pulse pressure (*Figure 1.6*), but there is no relationship between mean systemic arterial pressure and the mean intraocular pressure. Although sudden changes in ocular arterial pressure will lead to changes in intraocular pressure of the same sign, a rise in arterial vascular volume, such as that due to fluctuations in arterial pressure during anesthesia, will be buffered acutely by expression of venous blood from the vascular compartment of the eye. Therefore, the ocular pressure fluctuation will be minimized. A rise in venous vascular volume of the eye, however, will not be buffered significantly by compression of the arterial compartment. Although the rise in intraocular pressure will be buffered eventually by increased aqueous drainage from the normal eye, this will

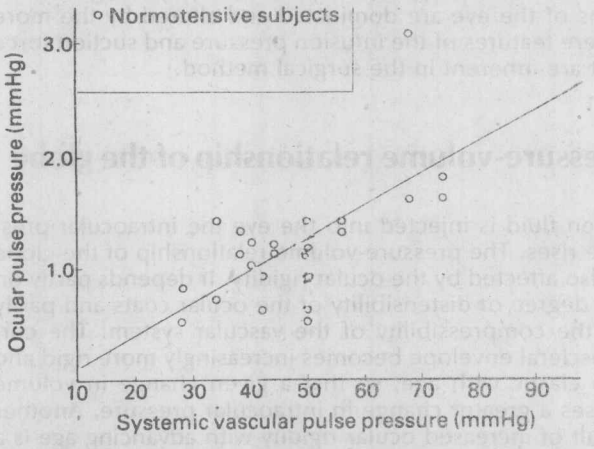

Figure 1.6 *Relationship between systemic arterial pulse pressure and the ocular pulse pressure. In normotensive patients, as the systemic vascular pulse pressure rises there is a linear rise of ocular pulse pressure*

be reduced if there is also a rise in episcleral venous pressure, thereby reducing the pressure gradient driving aqueous humor from the eye. Thus, a sustained rise in the central venous pressure would be expected to produce a sustained rise in intraocular pressure.

In the same way, compression of the globe will raise intraocular pressure, and the rise in pressure will be buffered acutely by expression of blood from the ocular vascular compartments and more slowly by expression of aqueous humor from the eye. With sustained external compression, further fluid is lost from the eye, so that when compression is released the intraocular pressure and volume will be below its usual level. Compression of the eye is of extreme importance in achieving low preoperative intraocular pressure and volume prior to cataract extraction, especially if intraocular lens (IOL) implantation is planned.

The closed eye and the open eye

The physiological relations discussed previously relate to the intact, or 'closed' eye. Once the eye is entered during surgery – as in cataract, corneal graft or glaucoma surgery – or after a penetrating injury, the intraocular pressure falls toward atmospheric pressure. With the eye thus opened, compression of the globe or a rise in the vascular volume will cause only a small rise in intraocular pressure, but it will result in displacement of the ocular contents toward the opening in the eye. This will also occur with expansion of the vascular compartment associated with a rise in the arterial and/or venous pressure within the eye and may be caused by a rise in the systemic arterial or central venous pressure. In still another situation, such as in pars plana vitrectomy, surgery may be performed in a 'closed-eye' system in which nearly watertight closure of the scleral incision(s) is maintained and there is an infusion system under hydrostatic control maintaining intraocular volume and pressure. The intraocular pressure is likely to be elevated during such surgery, thereby reducing the vascular perfusion pressure and counteracting the normal process of both aqueous humor production and outflow. In this special situation the physiological relations of the eye are dominated and altered by the more severe features of the infusion pressure and suction force that are inherent in the surgical method.

Pressure-volume relationship of the globe

When fluid is injected into the eye the intraocular pressure rises. The pressure-volume relationship of the globe is also affected by the ocular rigidity. It depends partly on the degree of distensibility of the ocular coats and partly on the compressibility of the vascular system. The corneoscleral envelope becomes increasingly more rigid and less elastic with age, so that a given change in volume causes a greater change in intraocular pressure. Another result of increased ocular rigidity with advancing age is a reduced tendency toward collapse of the globe when the eye is opened. Collapse of the globe is another factor tending to express ocular contents when the eye is opened.

Anesthetic factors in ocular surgery

Alterations in the physiology of the eye caused by anesthetic agents and other medications and procedures can have an important effect on the intraocular pressure and other features of the eye, thus affecting the safety and effectiveness of surgery. Of special importance is the relation between the eye and: (*1*) the central venous pressure (CVP); (*2*) changes in posture; (*3*) the Valsalva response; (*4*) blood-gas concentrations; and (*5*) direct ocular effects from anesthetic agents.

Central venous pressure

Recent studies suggest that, in the anesthetized supine subject, the CVP is about 5 mmHg lower than the intraocular pressure[15]. This implies that the pressure in veins immediately outside the eye (e.g. the episcleral venous plexus and ophthalmic veins of the orbit) are at a similar level, or slightly higher. If there is no major drop between the venous pressure within the eye and that outside the eye, then a rise in central venous pressure should retard venous outflow from the eye and raise intraocular vascular volume and intraocular pressure[15,16]. It would also be expected that a rise in venous pressure outside the globe would be associated with congestion of the conjunctival vasculature and perhaps increased bleeding in the operative field.

Hvidberg, Kessing and Fernandes[15] have shown that intraocular pressure closely follows changes in central venous pressure. This probably reflects the direct effect of elevated ophthalmic vein pressure on outflow from the vortex veins, rather than compression of the globe by expanded orbital vasculature. Comberg and Pilz[17] compared the effect of venous congestion produced by a neck cuff with that of injecting saline into the orbit. They found that saline injection produced a smaller rise in ocular pressure. Intraocular pressure rise induced by a neck cuff is also greatly diminished in man in the presence of diffuse choroidal sclerosis. These observations suggest that the elevated intraocular pressure is due to expanded intraocular vascular volume rather than a compressive effect by distended orbital veins.

Posture

In the non-anesthetized subject the intraocular pressure rises 1–2 mmHg in changing from the erect to the supine posture. In the anesthetized subject, adjustment of posture from the 15° head-up to the 15° head-down position is accompanied by an abrupt, square-wave rise of CVP of 5–10 mmHg[15]. This change in CVP is accompanied by a corresponding rise in intraocular pressure in the same range (*Figure 1.7*). The exact relationship between the CVP and intraocular pressure rise varies from subject to subject.

The smaller change in pressure with postural change in the awake subject may reflect a greater differential be-

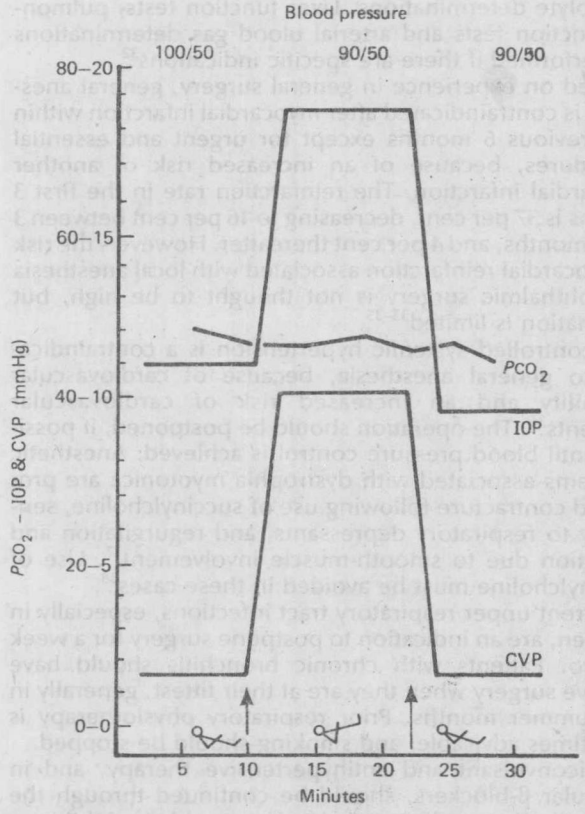

Figure 1.7 *Relationship between central venous pressure (CVP) and intraocular pressure (IOP). A rise in the CVP is accompanied by a corresponding rise in intraocular pressure in the same range (after Hvidberg, Kessing and Fernandes[15])*

tween the intraocular pressure and the ophthalmic vein pressure in the awake, erect subject. If there is a large drop in pressure between the intrascleral vortex veins and the ophthalmic veins in the orbit, then a moderate rise in CVP may not bring the ophthalmic vein pressure sufficiently close to the vortex vein pressure to retard outflow, expand the uveal vascular volume, and raise intraocular pressure. Langham and To'mey[14] suggested that cephalic venous pressure in the awake subject is about −25 mmHg in the erect posture.

Valsalva response

Such effects as laryngeal stimulation, airway obstruction, coughing, and 'straining' are all capable of stimulating a Valsalva response, involving a rise in intrathoracic pressure. This is transmitted to the cephalic arterial and venous systems, producing a square-wave rise in vascular pressures[18,19]. This vascular pressure rise is reflected precisely in the ocular vasculature, and it is associated with a rise in ocular pressure in the intact eye (*Figure 1.8*)[20,21]. A sustained Valsalva response is followed by further changes in cephalic arterial blood pressure and pulse, which are also reflected in the intraocular pressure response.

In the open eye, elevated intrathoracic pressure can be a major factor leading to prolapse of ocular contents. During cataract surgery this may lead to premature expulsion of the lens or prolapse of vitreous. In less severe cases there may be shallowing of the anterior chamber with iris prolapse and other conditions that prevent insertion of a lens implant. Elevated venous pressure might also increase the likelihood of choroidal hemorrhage.

Blood gases

Hypoxia results in vasodilation in the retinal circulation but not the uveal tract. A rise in arterial carbon dioxide tension ($PaCO_2$) is a powerful stimulus to uveal vascular dilation[22] and a lesser stimulus to retinal vascular dilation[23,24]. Vasodilation will increase the volume of the vascular compartment of the eye and will result in a rise in intraocular pressure in the intact eye, as discussed before[25]. Conversely, positive pressure ventilation while maintaining a low $PaCO_2$ is effective in constricting the choroidal vessels, reducing choroidal volume, and maintaining a low intraocular pressure[26–28].

Another mechanism may also account for elevated intraocular pressure with increased arterial carbon dioxide

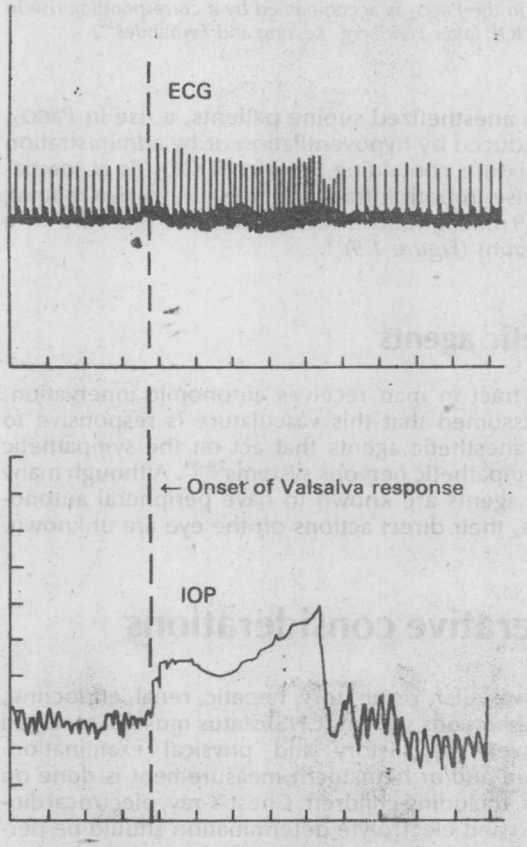

Figure 1.8 *Bottom graph: Valsalva response showing the pressure rise in IOP. The IOP shows a square-wave rise, with a later rise associated with tachycardia. Top record: ECG*

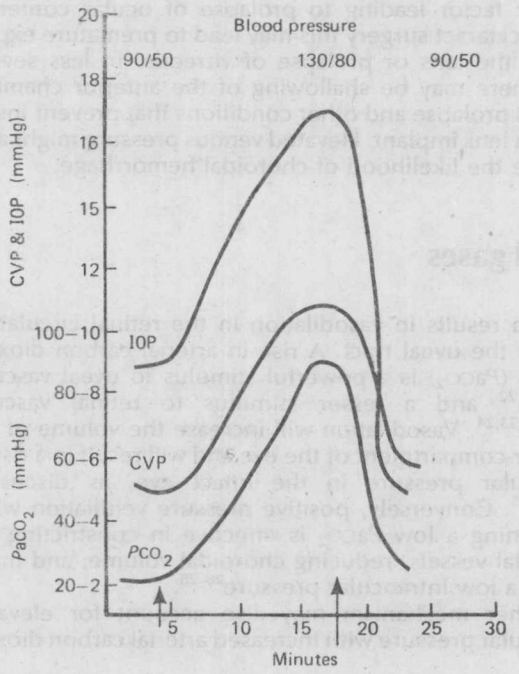

Figure 1.9 *Relationship between increased arterial carbon dioxide tension and the central venous pressure (CVP) and intraocular pressure (IOP). A rise in the $Paco_2$ is accompanied by a corresponding rise in the CVP and IOP (after Hvidberg, Kessing and Fernandes[15])*

tension. In anesthetized supine patients, a rise in $PaCO_2$, whether induced by hypoventilation or by administration of a gas mixture containing 5 per cent CO_2, is accompanied by a rise in both intraocular pressure (a rise ranging from 5 to 9 mmHg) and in central venous pressure of a similar amount (*Figure 1.9*)[15].

Anesthetic agents

The uveal tract in man receives autonomic innervation, and it is assumed that this vasculature is responsive to effects of anesthetic agents that act on the sympathetic and parasympathetic nervous systems[29–31]. Although many anesthetic agents are known to have peripheral autonomic effects, their direct actions on the eye are unknown.

Preoperative considerations

The cardiovascular, respiratory, hepatic, renal, endocrine, and central nervous system (CNS) status must be assessed preoperatively by history and physical examination. Hemoglobin and/or hematocrit measurement is done on all patients, including children. Chest X-ray, electrocardiogram (ECG) and electrolyte determination should be performed routinely on patients over 50 years of age and on younger patients if there is a specific indication. All patients on diuretic therapy, diabetics, and those on long-term steroid therapy should have serum urea and electrolyte determinations. Liver function tests, pulmonary function tests and arterial blood gas determinations are performed if there are specific indications[32].

Based on experience in general surgery, general anesthesia is contraindicated after myocardial infarction within the previous 6 months except for urgent and essential procedures, because of an increased risk of another myocardial infarction. The reinfarction rate in the first 3 months is 37 per cent, decreasing to 16 per cent between 3 and 6 months, and 4 per cent thereafter. However, the risk of myocardial reinfarction associated with local anesthesia for ophthalmic surgery is not thought to be high, but information is limited[33–35].

Uncontrolled systemic hypertension is a contraindication to general anesthesia, because of cardiovascular instability and an increased risk of cardiovascular accidents[36]. The operation should be postponed, if possible, until blood pressure control is achieved. Anesthetic problems associated with dystrophia myotonica are prolonged contracture following use of succinylcholine, sensitivity to respiratory depressants, and regurgitation and aspiration due to smooth-muscle involvement[37]. Use of succinylcholine must be avoided in these cases[38].

Current upper respiratory tract infections, especially in children, are an indication to postpone surgery for a week or two. Patients with chronic bronchitis should have elective surgery when they are at their fittest, generally in the summer months. Prior respiratory physiotherapy is sometimes advisable, and smoking should be stopped.

Anticonvulsant and antihypertensive therapy, and in particular β-blockers, should be continued through the perioperative period. Monoamine oxidase inhibitors should be stopped 10–14 days before elective surgery under general anesthesia, because of possible interactions with drugs used in anesthesia, as mentioned below. Tricyclic antidepressants potentiate catecholamines, and use of epinephrine (adrenaline) and norepinephrine (noradrenaline) should be avoided in patients on these medications[39,40].

Any adverse reaction to previous anesthetic agents or other medications, and other allergies, should be noted. Repeated use of general anesthesia with the inhalation anesthetic halothane should be avoided because of possible liver damage[41].

Some commonly used drugs have perianesthetic side-effects that are especially important. Knowledge of these side-effects and their underlying mechanisms make the anesthetic management easier and safer.

The following discussion of these drugs and their side-effects has been provided by the Editors.

Methyldopa (Aldomet)

The problems encountered with methyldopa are postural hypotension and sedation. Its metabolite, α-methylnorepinephrine, replaces norepinephrine both centrally and peripherally, leading to decreased adrenergic compensatory activity. Inhibition of dopa-decarboxylase resulting in decreased synthesis of norepinephrine is another mechanism causing hypotension. Decreased norepinephrine, dopamine and serotonin in the central nervous system account for the sedative effect[42].

Propranolol (Inderal)

Problems associated with use of propranolol are acute cardiac failure and bronchoconstriction. Cardiac failure is due to decreased inotropic and chronotropic responses to adrenergic cardiac stimulation. Bronchoconstriction is caused by blockade of β_2 receptors of smooth muscles in the bronchi and bronchioli[42].

Hydrochlorothiazide (Esidrex)

Hydrochlorothiazide causes an increased exchange of potassium for sodium in the distal tubules of the kidney. This causes chronic potassium loss that affects myocardial and peripheral nerve conduction and increases sensitivity to non-depolarizing muscle relaxants[43]. Loss of potassium also exaggerates the activity of digitalis, leading to brady-arrhythmias and other disturbances of cardiac conduction[42].

Quinidine sulfate (quinidine sulphate)

Quinidine sulfate is an antiarrhythmic drug that increases the refractory period and decreases response to repetitive stimulation in skeletal muscle, probably by interfering with transmembrane sodium-potassium movement. Prolongation and increased depression of respiration have been reported with use of depolarizing and non-depolarizing muscle relaxants[42].

Anticonvulsants

Anticonvulsants can cause anesthetic-related problems because of increased biotransformation. This is due to an increase in the activity of drug-metabolizing enzymes and increased tolerance to CNS drugs[42]. Also anticonvulsants are CNS depressants that potentiate the CNS depressant effect of anesthetic agents.

Monoamine oxidase inhibitors

Monoamine oxidase inhibitors can cause orthostatic hypotension secondary to decreased release of norepinephrine (noradrenaline) from the cytoplasmic storage pool and formation of a false weak transmitter, octopamine. Hypertension, hypotension, hypothermia, convulsion and coma may occur with use of meperidine (pethidine; Demerol). Drug-metabolizing enzymes are inhibited by monoamine oxidase inhibitors, leading to the production of different metabolites or toxic metabolites of opioids[42].

Tricyclic antidepressants

Tricyclic antidepressants can cause hypotension due to blockage of norepinephrine reuptake at adrenergic nerve endings. Urinary retention, tachycardia and increased intraocular pressure may occur secondary to its marked anticholinergic effects. The combination of tricyclic antidepressants and monoamine oxidase inhibitors can lead to hypertensive crisis[42].

Echothiophate iodide (echothiopate iodide; Phospholine Iodide)

Echothiophate iodide is a pseudocholinesterase inhibitor which may cause prolonged apnea after administration of succinylcholine. Pseudocholinesterase activity begins to decrease after several days of application, and 3–6 weeks may be required for pseudocholinesterase to return to normal activity after discontinuing echothiophate[44].

Phenylephrine (neosynephrine)

Phenylephrine is used topically in ophthalmic practice to produce capillary decongestion and pupillary dilation. Systemic effects include severe hypertension and possible subarachnoid hemorrhage.

Timolol (Timoptic; Timoptol)

Timolol is an ophthalmic solution used for treatment of glaucoma. It is a β-adrenergic blocker and may cause bradycardia and bronchospasm.

General anesthesia

Patients undergoing eye surgery are often in the extreme age groups, i.e. young children for strabismus surgery or examination under anesthesia, and elderly patients for cataract extraction and other intraocular procedures. There is also an increasing frequency of chronically ill patients, such as diabetics, undergoing vitreous surgery. Factors to be considered relating to general anesthesia include: (*1*) premedication; (*2*) induction; (*3*) intubation; (*4*) maintenance of anesthesia; (*5*) extubation; and (*6*) recovery from anesthesia.

Premedication

The objective of premedication is to produce relief of anxiety and sedation, with appropriate drying of secretions. Opiates are avoided because they are unnecessary and increase the incidence of nausea and vomiting. Opiate-induced respiratory depression may also be troublesome if an anesthetic technique involving spontaneous respiration is chosen.

In some circumstances oral premedication is satisfactory, although in some hospitals premedication is routinely given by injection. An antisialogogue is not essential in elderly patients and may be unpleasant for the patient. It

is, however, desirable in children, where secretions may provoke laryngeal spasm. If ketamine is to be used, an antisialogogue is probably essential. The antiemetic, sedative and antisialogogue actions of scopolamine (hyoscine) are useful. Scopolamine should not be used in elderly patients, because it may cause confusion and restlessness.

A satisfactory regimen is as follows. In adults under 65 years old, diazepam (Valium) 7.5–15 mg is given orally 2 hours before the operation, together with atropine 0.4–0.6 mg or scopolamine 0.4–0.6 mg intramuscularly. In adults over 65, diazepam 5–10 mg is given orally 2 hours before the operation, and this may be combined with atropine 0.4–0.6 mg intramuscularly. In the very frail or elderly patients, atropine 0.6 mg intramuscularly or no premedication whatsoever may be safer. Diazepam is a tranquilizing, centrally acting muscle relaxant and amnesic agent. Oral absorption is rapid and complete. Intramuscular injection is best avoided because it is painful and absorption is erratic.

In the United States, children over 2 years of age would commonly be given a combination selected from pentobarbital (pentobarbitone; Nembutal) 2–4 mg/kg intramuscularly 1 hour preoperatively; meperidine (pethidine; Demerol) 0.5–1 mg/kg intramuscularly; promethazine (Phenergan) 0.5 mg/kg intramuscularly; atropine 0.02 mg/kg intramuscularly. The usual combinations are meperidine-pentobarbital-atropine and meperidine-promethazine-atropine. If the exact time of surgery cannot be anticipated, it is recommended that intramuscular premedications be given 'on call'. In Great Britain a common approach would be trimeprazine syrup (Vallergan forte) 3–4 mg/kg body weight orally (to a maximum of 90 mg) and atropine 0.3–0.6 mg orally, both given 2 hours preoperatively. If desired, the atropine may be given 0.3–0.6 mg intramuscularly, 1 hour preoperatively. Infants less than 18 months of age are not given narcotics or sedatives because of the danger of respiratory depression and airway obstruction while they are unattended. Premedication is limited to atropine alone.

In patients undergoing outpatient surgery, premedication is omitted in most cases. Atropine is desirable for children, especially if ketamine is to be used or if intubation is planned.

Induction of anesthesia

Adults

Any of the currently available intravenous induction agents are satisfactory for use in adults, with the exception of ketamine, which often produces psychological disturbances in adults on awakening and cannot be used in 'open-eye' cases because it raises intraocular pressure. Thiopental sodium (thiopentone sodium; pentothal) produces reliably smooth induction, but it has a tendency to depress the cardiovascular system, especially in the debilitated patient. It produces a transient fall in intraocular pressure[45]. The long physiological half-life of thiopental causes a noticeable 'hangover' effect in the elderly but is now probably the most commonly used induction agent.

Etomidate, a new induction agent widely used outside the United States, is a derivative of imidazole, and it is now related to other agents used. It may have specific advantages in ophthalmic surgery. Etomidate produces an immediate and sustained fall in intraocular pressure of about 40 per cent lasting for at least 20 min[46]. Cardiovascular stability is excellent with etomidate, and no adverse allergic reactions have yet been reported. It is rapidly metabolized, and thus recovery is free from 'hangover'. However, troublesome side-effects include: (1) pain on injection, which can be reduced by use of a larger vein and mixing it with a local anesthetic agent; (2) myoclonic movements, which can be lessened by premedication. Furthermore etomidate abolishes the stress response after intravenous infusion and for this reason its more widespread use must await a fuller appraisal.

Children

Induction in children may be by inhalation or by the intravenous route. The preferences of the child and the anesthetist may be taken into account. If intubation is not required and where access is adequate with a mask, such as during probing of the nasolacrimal duct or examination under anesthesia, inhalational induction with halothane in nitrous oxide and oxygen is satisfactory.

If endotracheal intubation is planned, the intravenous route allows rapid induction and injection of a muscle relaxant to facilitate intubation. Alternatively, deep inhalational anesthesia with halothane, enflurane or isoflurane (Forane) will allow intubation.

Endotracheal intubation

Endotracheal intubation is necessary for intraocular surgery under general anesthesia to allow access to the eye and to ensure a clear airway. It also allows controlled ventilation, if desired, by using a cuffed endotracheal tube.

Spraying the trachea and larynx with 4 per cent lidocaine (lignocaine; Xylocaine) just before intubation reduces the likelihood of coughing or straining because of irritation caused by the tube and helps ensure smooth extubation. If local anesthetic spray is used, the patient should have nothing by mouth for about 3 hours after induction to avoid aspiration or thermal damage to the oropharynx. If intubation is used in young children, careful observation is needed after surgery because of the possibility of stridor from postintubation edema of the glottis or subglottic area.

Muscle relaxants are used to facilitate intubation without coughing or straining and to allow controlled intermittent positive pressure ventilation (IPPV) when necessary. Succinylcholine (suxamethonium; Scoline) is commonly used as the muscle relaxant. It is a short-acting 'depolarizing' drug that rapidly produces profound relaxation, and the effect lasts only about 5 minutes.

Succinylcholine causes a transient rise in intraocular pressure lasting less than 5 minutes[47]. The mechanism of this effect is probably a tonic contraction of the extraocular muscles[48–50]. However, it has also been suggested that the rise in intraocular pressure caused by succinylcholine is due to choroidal vasodilation[51], and pretreatment with a depolarizing muscle relaxant to abolish fasciculation does not reliably prevent the intraocular pressure rise[52].

Whatever the mechanism of action of succinylcholine that causes a rise in intraocular pressure, if this drug is used in cases with an 'open' eye, such as from a penetrating injury, contraction of the extraocular muscles and/or choroidal vasodilation could cause prolapse of the intraocular contents. Instead, a non-depolarizing relaxant such as pancuronium bromide (Pavulon) or curare can be used where it is safe to do so. Use of non-depolarizing relaxants may be hazardous in such cases if intubation proves difficult.

Succinylcholine is broken down by plasma pseudocholinesterase[53]. The topical antiglaucoma agent, echothiophate iodide (echothiopate; Phospholine Iodide), is a potent anticholinesterase agent and may cause prolongation of action of succinylcholine, leading to prolonged apnea. This event must be treated with IPPV until reversal occurs. Succinylcholine is avoided, therefore, in patients being treated with echothiophate, and echothiophate is discontinued preoperatively when possible.

Muscle relaxants of the non-depolarizing (curare-like) type tend to cause a fall in intraocular pressure by relaxing the extraocular muscles and by lowering the arterial pressure[54]. Alcuronium chloride (not yet available in the United States) can be used effectively, although it causes a small lowering of arterial blood pressure[55]. Pancuronium is an alternative if hypotension is undesirable[56].

Maintenance of anesthesia

The major choice in technique is between spontaneous or controlled ventilation. In both cases a volatile agent such as halothane (Fluothane), enflurane (Ethrane) or isoflurane (Forane) in nitrous oxide and oxygen is used to maintain anesthesia. The former agents lower intraocular pressure, perhaps by depression of midbrain and hypothalamic centers[57,58]. If an intraocular bubble will be injected during retinal or vitreous surgery, nitrous oxide is stopped at least 10 minutes before the bubble is injected and is withheld during the rest of the operation. This is done to permit clearing of the nitrous oxide through the lungs before intraocular gas injection, and thereby to avoid an equilibration between the soluble nitrous oxide and the injected gas that would cause rapid temporary expansion of the bubble size and possible marked elevation of the intraocular pressure.

Spontaneous ventilation

Spontaneous ventilation has the advantage of simplicity and ease of extubation without the need for reversal of muscle relaxation. Mean intrathoracic pressure is also negative, leading to low central venous pressure. However, the patient must be kept at a deep level of anesthesia to avoid coughing and straining due to irritation by the endotracheal tube. This causes respiratory depression and a raised $PaCO_2$.

The deep anesthesia will help to counteract the rise in intraocular pressure produced by the raised $PaCO_2$, but it is also desirable to position the patient in a 10–15° head-up tilt to lower venous pressure in the orbit. Problems of hypotension caused by these maneuvers may be troublesome, especially in the elderly. Deep anesthesia is also associated with a prolonged recovery time. The anesthetic system used to administer the gases must offer minimal expiratory resistance to prevent rises in intrathoracic pressure, and should not result in rises in $PaCO_2$.

Controlled ventilation

Controlled ventilation with N_2O and a small (0.5–1 per cent) concentration of halothane has certain advantages for intraocular surgery. $PaCO_2$ may be maintained at a normal level or lowered, if desired, to reduce intraocular pressure. Non-depolarizing muscle relaxants relax the extraocular muscles and reduce the risk of coughing or straining during the procedure, without the need for deep anesthesia. The synthetic opiate, fentanyl (phenoperidine; Sublimaze) (50–100 μg), may be given before induction to reduce the dose needed of induction and maintenance agents, and may reduce coughing on extubation.

It has been suggested that positive pressure ventilation may increase intraocular pressure because of increased intrathoracic pressure. However, this effect appears to be small and easily counteracted by control of $PaCO_2$. Positive pressure ventilation with low $PaCO_2$ causes choroidal vasoconstriction and therefore may discourage forward displacement of ocular contents when the eye is opened.

Controlled ventilation, therefore, may be of special value in certain cases in which: (*1*) spontaneous ventilation is likely to be inadequate or depressed; (*2*) deep anesthesia may not be well tolerated, as in elderly patients or those with cardiac disease; (*3*) there is an increased risk of vitreous loss as in cases of high myopia, young patients, and aphakic corneal grafts; and (*4*) forward displacement of the vitreous would cause special problems, as in intraocular lens implant surgery. Lowering of intraocular pressure may also be helpful in intraocular retinal detachment surgery, to allow easier indentation of sclera during examination and during placement of a scleral buckle.

Antiemetic

Administration of an antiemetic before the end of the operation reduces the incidence of vomiting and retching in the postoperative period. Perphenazine (Trilafon; Fentazin) 2–3 mg or prochlorperazine (Compazine; Stemetil) 5–12.5 mg intramuscularly about 15–20 minutes before the end of surgery is suitable for most adults. It also has a sedative effect. Care is taken in patients with cardiovascular disease, because both drugs can cause hypotension. Metoclopramide (not available in the United States) 10 mg intramuscularly may be preferable in these cases. A small dose of droperidol (Inapsine; Droleptan) 2.5 mg intravenously is also extremely effective and does not interfere with emergence from anesthesia. For children a small dose of cyclizine (Marezine; Valoid) intramuscularly is suitable. However, trimeprazine (Temaril; Vallergan), which is often used in premedication of children, is also an effective antiemetic.