ASPECTS OF MEASUREMENT IN OPHTHALMOLOGY

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Blood supply

The blood supply to the eye differs in a number of respects from that to other organs in the body. The retina is a part of the brain and is connected to the brain by the optic nerves. The term “optic nerve” is incorrect, as it is in reality a cerebral tract connecting one part of the brain to another. The retinal blood supply is therefore similar to the cerebral blood supply and nutrition of the retina is accomplished from the retinal capillary beds by glial cells, which transfer nutrients directly from the vascular lumen. In the systemic circulation nutrition of body cells is accomplished by the extracellular fluid and removal of this fluid is effected by veins and lymphatics. The retina, in common with brain tissue, has no lymphatic supply. The blood supply to the inner two-thirds of the retina is by the central retinal artery, an end artery derived from the internal carotid via the ophthalmic artery. The retina contains two major capillary beds at different levels, supplied by the branches of the central retinal artery. The outer one-third of the retina is supplied by diffusion from the choroid, or middle coat of the eye. The choroid derives its blood supply from multiple small vessels which pierce the sclera posteriorly and provide a segmental blood supply to the choroidal capillary bed. There are between 10 and 20 short posterior ciliary arteries supplying the posterior choroid and prelaminar optic nerve head. These are derived from two or three main posterior ciliary arteries, which in turn are branches of the ophthalmic artery. Two of the posterior ciliary arteries derived from the main posterior ciliary arteries run forward in the suprachoroidal space between the choroid and the sclera. These may supply small segments of the choroid laterally and medially (Hayreh, 1976), but mainly terminate by forming the major arterial circle of the iris in the anterior ciliary body.

The ophthalmic artery gives branches to the extraocular muscles in the posterior orbit and anteriorly these vessels enter the globe at the attachments of the muscles. At this point, they are known as the anterior ciliary arteries. The anterior ciliary arteries also terminate at the major circle of the iris. The blood supply to the anterior segment and ciliary body is derived from the conjunctival, anterior ciliary and long posterior ciliary arteries.

The venous drainage of the eye mirrors the arterial supply, but there is no major venous circle within the iris. The venous anastomosis is less well defined and its situation closer to the pupil is responsible for its having been named the “minor vascular circle of the iris”. The inner retina drains into the central retinal vein, which crosses the dural spaces before leaving the optic nerve sheath posterior to the globe. At this point, the vein can be compressed by increased cerebro-spinal fluid pressure and swelling of the optic nerve head may result. The choroidal venous drainage is through the vortex veins, situated in the four quadrants of the eye, just behind the equator. The fact that there are up to 20 arteries supplying the choroid and only four main veins means that ischaemic areas of the choroid may fill by retrograde flow through the venous channels (Hayreh, 1976).

Formation and drainage of aqueous

The aqueous humour is produced in the posterior chamber of the eye and flows through the pupil into the anterior chamber where it passes out of the eye through the trabecular meshwork. The trabecular meshwork is situated in the angle of the anterior chamber between the peripheral cornea and iris. The resistance to flow in the trabecular tissue probably maintains the intraocular pressure (i.o.p.) within physiological values, but the mechanism of homeostasis is unknown. The aqueous drains into the venous system via the canal of Schlemm, then into aqueous veins in the episcleral tissue. The episcleral veins drain into the orbital venous system and eventually to the cavernous sinus via the superior and inferior ophthalmic veins. A gradient normally exists between i.o.p. and the pressure within the aqueous veins; intraocular pressure may increase if episcleral venous pressure is increased.


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In some disease states, an increase in intraocular pressure may occur in a number of situations (table I). The term "glaucoma" is applied to any condition causing increased i.o.p. Any chronic increase in pressure in the eye will in turn damage the trabecular meshwork and may exacerbate the situation (Lee and Grierson, 1974). Chronic forms of glaucoma may therefore develop as a result of degeneration in the trabecular meshwork or because of meshwork degeneration following prolonged increase of intraocular pressure for other reasons.

CLINICAL MEASUREMENT

Measurement of intraocular pressure

The accurate regulation of intraocular pressure is necessary to maintain the shape of the eye for optical purposes. Too small a pressure will enable external forces to compress and distort the eye and may permit the development of unwanted oedema; too great a pressure will prevent perfusion of the eye with blood. The eye is a closed organ and the analogy of intracranial pressure is often used to assist understanding of ocular fluid dynamics. The aqueous humour, in addition to its supportive role, is also nutritive to the cornea and to the lens. There is therefore an active turnover of aqueous in order that the nutritive functions can be maintained. Like cerebrospinal fluid pressure, i.o.p. is the result of the balance between inflow and outflow and increase or decrease in i.o.p. may reflect change in either. Aqueous is produced by the ciliary epithelium as a result of ultrafiltration of plasma by hydrostatic pressure and by the process of active transport of sodium ions into the posterior chamber. This active transport is responsible for the subsequent inflow of water into the posterior chamber along the resultant osmotic gradient. Assuming a constant rate of aqueous inflow, the intraocular pressure is therefore dependent on the outflow resistance offered by the trabecular tissue, and outflow of aqueous assumes a pressure gradient between i.o.p. and the episcleral venous pressure. External pressure on the eye will obviously increase i.o.p. initially, but even if the compression is maintained, aqueous outflow will cause a decrease in i.o.p. and regulation at the previous pressure should ensue. Measurement of i.o.p. should therefore give information relating to the relative amounts of aqueous inflow and aqueous outflow and should enable some conclusions regarding the normality of aqueous production and aqueous drainage. Like arterial pressure, i.o.p. can be measured manometrically (directly) or tonometrically (indirectly, by inference). Manometric measurement of i.o.p., when the anterior chamber is cannulated, is not used clinically and the method is restricted to experimental work. Indirect methods of measurement, therefore, are important. Historically, indentation of the eye was practised through the eyelid and on the
sclera, but more recently instruments have been applied to the cornea, usually requiring corneal anaesthesia. There are two basic methods of measuring i.o.p. indirectly. Both depend on the application of a measured force to the eye and the resulting deformation. In one, the amount of indentation produced by a given force is measured, and in another the area of flattening is noted (applanation). All tonometers increase i.o.p. and the measured pressure is greater than the pressure before tonometry. The rigidity of the wall of the eye is an additional complicating factor to oppose the applied weight and may cause inaccuracy.

The ophthalmic literature contains many references to new tonometers, but several have remained in common use. The Schiotz tonometer (Schiotz, 1920, 1925) is the most widely used indentation tonometer, but is inaccurate because of wide variations in scleral rigidity (Friedenwald, 1957). Repeated measurements with different loading weights are required to calculate correcting coefficients and improve the accuracy. The Schiotz method remains in common use despite its inaccuracy, and it must be said in its favour that it is easy to use and is therefore a useful instrument for semi-skilled personnel (fig. 1). The most widely used instrument is the applanation tonometer in one of its many forms. Significant improvement in accuracy can be achieved by the applanation of a standard area of cornea. The tonometer is in contact with the tear film and the area of flattening is usually visualized by the addition of a dye to the tear film and observation of the flattened and therefore dye-free area. With small areas of applanation, the surface tension of the tears makes the area of flattening greater than expected and with large areas of applanation, corneal rigidity makes the

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**FIG. 1.** The Schiotz Tonometer (left). This is an example of an indentation tonometer. The scale does not give a direct reading of intraocular pressure, which is found by consulting a separate table. The Perkins Tonometer (right) is a hand-held applanation tonometer and gives an accurate reading directly from the scale.
area of flattening less. An intermediate area of flattening is adopted therefore, and in the Goldmann Tonometer, an area 3.06 mm in diameter is chosen. This instrument only displaces a small volume of aqueous and the small increase in i.o.p. produced makes the Goldmann tonometer one of the easiest to calibrate and the most accurate in use. A portable tonometer has been devised by Perkins (Perkins, 1965), utilizing the Goldmann tonometer head. By the use of a split-prism doubling device in the optics of the tonometer (Perkins, 1965; Draeger, 1967), the end-point of the tonometer is easily visualized and the device is quick and easy to use. Because of its accuracy and ease of use the Perkins tonometer has been popular for assessment of intraocular pressure during anaesthesia (Al-Abrak and Samuel, 1974; Samuel and Beaugié, 1974). The Mackay-Marg electronic tonometer has attempted to eliminate the problems of the forces of rigidity and surface tension and has the advantage of producing a permanent graphic record (Mackay and Marg, 1959). This instrument contains a small plunger in the tonometer head the movements of which are recorded graphically during a rapid application to the cornea. The equipment is cumbersome and requires experience to obtain satisfactory readings.

Finally, the most recent development is the no-contact or air tonometer (Langham and McArthy, 1968). Corneal flattening is produced by a strong jet of air of constant known force. This jet is directed at the central cornea from a fixed distance. The optics of the system ensure that the air jet is normal to the cornea. A photocell measures the reflection of an incident beam of light from the cornea. At the moment of corneal flattening the reflected light increases; the amount of the increase is inversely proportional to the intraocular pressure, and the value for i.o.p. is calculated electronically. The air tonometer was developed for the American market where opticians and other non-medical personnel are not permitted to apply instruments to the cornea or to instill topical anaesthetics. This tonometer is accurate, but can be used only on a mobile patient and in the erect position. The cornea must be smooth and normally reflective.

The air tonometer is favoured by some opticians in the United Kingdom, but it is expensive and the lack of legal restraints on the use of alternative instruments have limited wider adoption.

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**Fig. 2.** Demonstration of rapid changes in ocular fluid dynamics with variation in end-expired $P_{\text{CO}_2}$. The tonography gradient is steeper with decreasing carbon dioxide and reverses with large increases.
Tonography

Tonography is a technique which was introduced for the clinical assessment of patients with glaucoma. If serial readings are taken with the Schiotz indentation tonometer, successive values for i.o.p. are smaller because of the displacement of aqueous through the normal drainage channels. By utilizing electronic versions of the Schiotz tonometer, a continuous reading of the resultant decreasing intraocular pressure can be obtained. If the readings are taken over the course of a few minutes a graph is obtained showing a uniform descending slope. The gradient of the slope (tangent) is a measure of the ease with which aqueous can be driven out of the eye through the normal drainage channels, and thus the patient with reduced aqueous outflow should show a decrease in the gradient of this slope. Readings can also be made to demonstrate increased facility of outflow after surgery or after the introduction of medical therapy. This technique has largely been abandoned clinically because of the poor repeatability of results on individual patients. Facility of outflow (or its reciprocal, outflow resistance), expressed numerically, is reduced in groups of patients with glaucoma when they are paired with a similar group of normal patients, but a knowledge of the facility of outflow is often of little value in the individual patient. The technique of tonography is not now widely used, because of the difficulty in interpretation of results.

Experimentally, tonography gradients have been shown to reverse rapidly with rapid variations in $P_{CO_2}$ (fig. 2). This is presumably the result of a rapid vascular volume change within the eye, almost certainly occurring in the choroid. These changes have been shown to be independent of central venous pressure.

Measurement of ocular blood flow

Ocular blood flow is the most difficult parameter to measure when considering ocular fluid dynamics. Ophthalmic artery pressure can be estimated by using the eye itself as a sphygmomanometer. The central retinal artery can be observed ophthalmoscopically and its behaviour on changes in intraocular pressure observed. Again, manometric pressure changes are the most accurate, but some idea of systolic and diastolic pressures can be obtained by digital compression of the globe or by the application of a calibrated spring plunger (ophthalmodynamometry). The technique of ophthalmodynamometry is not widely used, in spite of the great number of instruments which have been designed to give a numerical value to the pressures within the ophthalmic artery. The technique is mainly used for comparison of one eye with the other.

Qualitative estimation of ocular blood flow may be obtained utilizing the technique of fluorescein angiography. Following an i.v. injection of 15–25% sodium fluorescein 3 ml, serial ocular fundus photography is performed. The illuminating light is a blue light produced by filtering the emission from a xenon photographic strobe through an interference filter passing a narrow band within the blue range. The resultant blue light excites the i.v. fluorescein and causes a yellow fluorescence. The retinal vessels are photographed through a second narrow band interference filter, this time selected to absorb the blue light which may be reflected from the retina and to pass only yellow fluorescent light. The fluorescein is largely protein-bound in the circulation and since retinal vessels are normally impermeable to protein, the passage of dye through the retinal vessels is visualized clearly. The choroidal circulation can often be visualized at the same time, especially in lightly pigmented races where the mass of fundus pigment does not obscure the choroid (fig. 3). Choroidal capillaries are permeable to protein and the resultant leakage of fluorescein can be visualized...
under some circumstances. Some workers have used a technique similar to fluorescein angiography to visualize the choroidal circulation, but have used the dye indocyanine green and infra-red light in an attempt to remove the problems of obscuration by the retinal pigment epithelium.

Quantitative estimation of choroidal blood flow has been obtained by measuring the density of injected dye by the technique of reflective densitometry, by radioactive methods using injected microspheres or by inert gas clearance. The Fick principle applied to the ocular circulation can measure the clearance of radioactive xenon-133 or krypton-85 (Strang, 1976). Since there is also loss of radioactivity as a result of diffusion, the calculation of proportional loss resulting from circulation is complex. Using these methods the response of the ocular circulation under various physiological stresses has been studied and the behaviour of the normal eye can be predicted.

FACTORS CAUSING VARIATION IN INTRAOCULAR PRESSURE

In a closed elastic organ such as the eye it becomes difficult to separate the terms used to express pressure and volume. An increase in intraocular pressure is usually produced by an increase in the volume of the ocular contents and the resulting stretching of the connective tissue coat. The sclera reacts to stretching in a non-linear fashion (Gloster, Perkins and Pommier, 1957; Woo et al., 1972). Because of the presence of both collagen and elastic fibres in the sclera, the behaviour of the eye is analogous to a balloon contained within a relatively inelastic string bag. A moderate increase in volume will produce only a small increase in intraocular pressure initially, and thus a change in i.o.p. from 20 mm Hg to 30 mm Hg during an attack of acute glaucoma probably represents a moderate increase in intraocular volume. At greater pressure (70-80 mm Hg) rapid fluctuation in i.o.p. can be obtained by small changes in fluid volume because of the relative inelasticity of the now stretched sclera (Perkins and Gloster, 1957). For this reason it is easier to produce a decrease in i.o.p. at high pressures by small alterations in relative aqueous inflow or outflow.

Expression of intraocular pressure becomes meaningless once the eye has been opened surgically. The intraocular pressure is now equal to ambient pressure and has decreased because of the release of a small quantity of aqueous. During surgery, concern is not for intraocular pressure, but for the relative volume within the eye of choroid and vitreous. Loss of vitreous humour during a surgical operation was once considered disastrous. Vitreous within the anterior chamber led to rapid opacification of the cornea and blockage of the drainage angle, and vitreous within the wound led to chronic fistula formation. Contraction of vitreous subsequently led to a high frequency of traction retinal detachment. Modern surgical techniques of removal of prolapsed vitreous have greatly reduced the morbidity of vitreous loss. Prolapse of vitreous was once associated with attempted cataract extraction under general anaesthesia and a high frequency of this complication has led to a continuation of cataract surgery with retrobulbar local anaesthesia. Local anaesthesia (1% lignocaine 0.5 ml and adrenaline 1 : 200 000) to the ciliary ganglion produces a significant and measurable decrease of i.o.p. in the intact eye. Vitreous loss is caused by a relative increase in intraocular volume, when the anterior chamber is open. More accurately, it is an increase in volume posterior to the iris that causes concern. An apparent increase in volume may occur as a result of deformation of the eye by fixation sutures or the eyelid speculum and this must be guarded against by the surgeon. Occasionally, retrobulbar injection causes bleeding within the orbit and this may have a similar effect.

At the beginning of an intraocular procedure, the eye should ideally be soft before the anterior chamber is open. A sudden decompression by release of aqueous from a tense eye may produce shearing stresses on posterior ocular blood vessels and disastrous haemorrhage may occur, particularly in the atherosclerotic subject. A severe expulsive haemorrhage is rare, but may rapidly expel vitreous, retina and choroid, causing permanent blindness. Even when bleeding is not profuse, small amounts of blood or transudate may produce bloody or serous detachments of the choroid, increasing posterior intraocular volume. Anaesthesia should therefore be directed to reducing choroidal vascular congestion, as the ocular blood content is probably the easiest part of the intraocular volume to influence. The administration of acetazolamide is a traditional precursor to ophthalmic surgery, but in the subject with an initially normal i.o.p., it produces only a minimal reduction in pressure and may even increase ocular blood flow (Wilson, MacKenzie and Strang, 1976). Acetazolamide also reduces aqueous outflow (Wistrand, 1964). The reduction of choroidal congestion can be accomplished by any of the factors known to reduce vascular engorgement. Thus,
reduction of $P_{\text{aCO}_2}$, prevention of anoxia and reduction of systemic arterial pressure and central venous pressure will reduce the choroidal blood volume. The choroid in the cadaver is usually stated as being about 200 $\mu$m thick, but evidence from ultrasound examination in the living eye suggests that it is in fact much thicker, perhaps 500 $\mu$m at physiological values for $P_{\text{aCO}_2}$ and c.v.p. (Coleman and Lizzi, 1979). Coleman and colleagues (1974) suggested from ultrasonic evidence that the choroid may be up to 1 mm thick at times. Figure 4 shows an ultrasonogram of the posterior ocular wall at physiological values in a conscious patient. Using simple mathematics, we can consider the volume changes which would occur in the eye with a change in choroidal thickness; a 500-$\mu$m alteration (from say 200 $\mu$m to 700 $\mu$m) would produce an increased posterior volume of more than 0.5 ml. (This is with a normal vitreous volume of about 5 ml and assuming that the choroid covers only two-thirds of the interior of the eye.) Wide variations in intraocular volume have been shown to occur instantaneously with changes in carbon dioxide tension (Wilson and LeMay, 1974; MacDiarmid and Holloway, 1976) and some observers have recorded rapid changes in intraocular pressure occurring during anaesthesia (Dominguez et al., 1974; Dominguez, Gimenez Alvarez and Sanchez Baños, 1975). These changes are so rapid that it seems definite that some sort of vascular response is involved. There seems little doubt that rapid changes in intraocular volume are the result of rapid changes in the state of engorgement of the choroid, although these changes have not been observed directly.

During the early stages of cataract surgery when the anterior chamber is open an increase in the volume of the ocular contents is suspected when the iris bulges or the lens spontaneously dislocates anteriorly. At this stage in the operation if time is spent attempting to reduce intraocular volume, the complication of vitreous presentation can be averted. All external deforming forces are removed. Anaesthesia should have been appropriate to the type of surgery from the initial stages of the operation, but further steps can be taken at this time to reduce ocular volume if a disaster appears imminent. The appropriate measures are discussed fully in later papers. The decreasing of systemic arterial pressure alone or the decreasing of arterial pressure and central venous pressure by an increase in the tilt of the operating table may produce a rapid decrease in ocular blood volume. I.v. sucrose (25 g of 50% solution) reduces vitreous volume, but takes 20 min to be fully effective and the increased anaesthetic time may be considered undesirable. Samuel and Beauquié (1974) remarked on the changes in i.o.p. occurring with changes in arterial $P_{\text{CO}_2}$. Choroidal blood flow increases with increasing $P_{\text{aCO}_2}$ (Strang, Wilson and Johnston, 1974; Wilson, MacKenzie and Strang, 1976). In animals, increases in choroidal blood volume accompany increases in choroidal blood flow (Bettman and Fellows, 1956). The volume of the choroid can be reduced if $P_{\text{aCO}_2}$ is kept small and if hypoxia is avoided. Control of these parameters, more than any others, prevents vitreous prolapse. Reduced congestion also minimizes bleeding from the wound, and reduces the risk of posterior bleeding with consequent choroidal detachment or expulsive haemorrhage. The retinal and orbital vessels respond in a similar fashion to choroidal vessels, but the contribution of engorgement of these vessels to vitreous presentation is probably slight. Certainly, haemorrhage within the orbit has to be gross before compression of vessels or an increase in i.o.p. is produced, and similar volume changes in the orbit are not likely to be produced by simple engorgement of vessels. If immediate measures have been successful the surgeon observes recession of the lens–iris diaphragm. Subsequent surgery is likely to be uncomplicated.

In the present short review, the volume of the ocular contents has been given great prominence. Little mention has been made of the additional complication of bleeding occurring during surgery.
There is no doubt that because of a reduction in bleeding generally, hypotensive anaesthesia has made developments in ophthalmic surgery possible and technical advances in surgery have paralleled advances in anaesthesia. Perhaps it is more honest to say that advances in surgery have only been possible after the appropriate improvements in anaesthesia. It is only with a full knowledge and understanding of the ocular fluid and vascular physiology during anaesthesia that we can contemplate embarking on difficult and otherwise hazardous procedures such as partial choroidal resection or dissection within the orbit. These topics are discussed by Foulds elsewhere in this issue (Foulds, 1980).

REFERENCES


