Glaucoma

Part 2 – Intraocular pressure and tonometry

Dr Robert Harper and **Dr Paul Spry** continue their look at glaucoma with a discussion of intraocular pressure and its measurement. C8751, two general CET points, suitable for optometrists and dispensing opticians

In the first article in this series, the definition of glaucoma, epidemiological issues and the pathogenesis of glaucomatous optic neuropathy (GON) were considered, alongside the classification of glaucoma and the features of the more common glaucomas. This second article summarises the mechanisms responsible for elevated intraocular pressure (IOP) and the relationship between IOP and glaucoma. The normal variations in IOP and the factors which influence IOP are discussed, and, in view of the increasing recognition of the importance of a number of factors, in particular corneal characteristics, in obtaining accurate estimates of IOP, an updated review of tonometry is presented.

What causes elevated IOP in glaucoma?

The mechanisms for elevation of IOP in chronic glaucoma are not fully understood, although increased aqueous production rate or reduced aqueous outflow facility can be considered. Early work by Becker noted the outflow facility to be decreased significantly with age in normal individuals, a finding that appeared to be matched by compensatory reduced aqueous secretion, maintaining normal IOP. In cases of glaucoma, both variables were found to be further reduced. The majority of aqueous drainage from the eye is facilitated by the trabecular meshwork and Schlemm's canal, and some structures within this pathway appear to exhibit signs of accelerated ageing in glaucoma and have been the subject of investigation.

Cellularity

Endothelial cell populations lining connective tissue fibres (trabeculae) have been shown to decrease in number with age, and are further reduced in glaucoma. An important role of endothelial cells is phagocytosis of inter-trabecular debris. Significantly increased endothelial cell populations in meshwork areas nearest the cornea have been found in those with early glaucoma compared with normal individuals, but significantly less in those with longer-term pathology. It has also been found that those with more advanced glaucoma show significantly more cytoplasmic pigment than normals. Knowing the adverse effect of pigment ingestion on endothelial cells, it has been suggested that melanosome phagocytosis caused either accelerated cell death or 'activation' of endothelial cells, facilitating their movement from the meshwork in cases of glaucoma. Although such endothelial 'wear and tear' also occurs normally, it is believed to be increased in glaucoma due to variable aqueous dynamics.

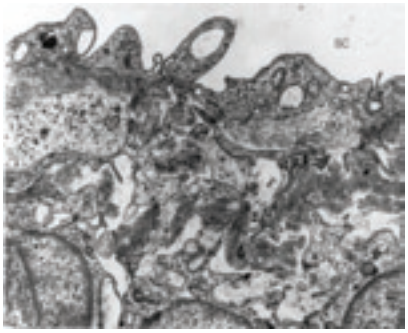

Figure 1 Cribriform layer changes in the drainage channels in glaucoma (courtesy of Professor John Lawrenson, City University London)

Trabecular beam structure

Depletion of the trabecular endothelial cell population has been found to cause progressive fibrotic thickening, fusion and compaction of trabeculae, producing apparent enlargement of the scleral spur. Without continuous cell cover, adhesions between denuded portions of adjacent trabecular beams can develop, serving to reduce trans-trabecular space, size, and frequency. The degree of thickening has been found to be ~40 per cent between birth and the eighth decade, although the focal nature of trabecular beam changes makes this change unlikely to be the cause of impaired outflow in glaucoma.

Cribriform layer

Lacking the structural regularity of the corneo-scleral and uveal meshwork, the sheet-like cribriform, or juxtacanalicular layer has been the subject of much investigation. The layer is continuous with the inner wall of Schlemm's canal and consists of fine fibrils, ground substance and an elastic-like fibre system, creating discrete pores. These pores indirectly connect the more widely spaced uveal and corneo-scleral meshwork and the inner wall of Schlemm's canal. In cases of COAG, this layer has been found to contain excessive amounts of extracellular material ('plaques'). Both treated and untreated cases of glaucoma have significantly higher amounts of plaque material than controls of a similar age range, and plaque formation shows no correlation with IOP, suggesting that such changes are part of the natural history of the glaucomatous process, rather than being secondary to it.

Schlemm's canal

It is thought unlikely that Schlemm's canal abnormality is the cause of elevated IOP. Indeed, it has been suggested that elevated IOP produced by a meshwork resistant to aqueous outflow causes Schlemm's canal collapse. Age-related changes in Schlemm's canal have been found to include a significantly reduced population of endothelial cells, equivalent to a cell drop out of up to 30 per cent between birth and the eighth decade, and reduced capacity to produce giant vacuoles from the fifth decade of life onwards. Significantly reduced Schlemm's canal cross-sectional area, perimeter and inner wall length have been found in COAG compared with normal eyes, which may account for up to 50 per cent of reduced outflow facility in glaucomatous eyes. Whether this finding is the result of primary glaucomatous pathology or secondary to trabecular meshwork change remains unclear.

IOP and glaucoma

Whatever the specific mechanisms for elevated IOP, the role of IOP in causing GON remains the subject of considerable debate. While there is no doubt that raised IOP is an important risk factor for the development of COAG, the concepts of 'normal tension glaucoma' ▶

TABLE 1
Prevalence of POAG at different levels of screening IOP and the relative risk at specific levels of IOP, from the population-based Baltimore Eye Study (Sommer *et al*, 1991)[9]

IOP (mmHg)	Cumulative % with POAG	Prevalence of eyes with POAG (%)	Relative risk
<15	13	0.65	1.0
16-18	37	1.31	2.0
19-21	59	1.82	2.8
22-24	78	8.30	12.8
25-29	88	8.33	12.8
30-34	97	25.37	39.0
>35	100	26.09	40.1

(NTG) and 'ocular hypertension' (OHT) challenged the traditional belief that a raised IOP was necessary for the development of COAG or that a raised IOP alone is sufficient for the development of COAG. While on the one hand there is evidence linking glaucoma to vascular dysregulation, summarised in Part 1, there is also an accumulating body of evidence from randomised trials that reducing IOP exerts a favourable influence on the course of the disease. The Ocular Hypertension Treatment Study (OHTS), the Early Manifest Glaucoma Trial, the Advanced Glaucoma Intervention Study (AGIS) and the Collaborative Normal Tension Glaucoma Study, for example, provide evidence that IOP reduction is beneficial in OHT and glaucoma, although these trials also serve as a reminder that IOP lowering does not inevitably arrest the development or progression of glaucoma.

Raised IOP is considered to be the most significant risk factor for COAG. Despite the fact that most epidemiological studies have found between one to two thirds of all cases of COAG have 'normal' IOP at presentation, the probability of GON increases with higher pressures and this 'dose-response' relationship between IOP and COAG provides supporting evidence for the role of IOP as a risk factor for COAG. Table 1 summarises data from the Baltimore Eye Study, showing how the prevalence of POAG and the relative risk for POAG increase the higher the screening IOP, particularly at levels of 22-29mmHg and >30mmHg. Early studies that examined the rate of conversion to glaucoma from OHT observed an incidence of visual field loss in approximately 1 per cent of patients per year. More recently, in OHTS, approximately 10 per cent of subjects with OHT converted to GON and/or glaucomatous visual field loss during the course of the five-year study (ie 2 per cent per year although approximately 90 per cent of subjects with OHT did not convert). Although the conversion rate from OHT to glaucoma is relatively low, several studies have demonstrated that the higher the IOP, the greater the risk of developing COAG. Also, research has demonstrated a relationship between raised IOP and the extent of visual field loss at presentation. While in treated glaucoma cases, the relationship between the IOP and the progression of visual field loss has been more difficult to establish, the AGIS research indicates that a dose-response relationship between IOP and visual field progression is also likely. AGIS findings suggest that eyes with an IOP <18mmHg at all visits over a six-year period are unlikely to show progression of their visual field defect, whereas eyes with worse control (ie some follow up visits with an IOP >18mmHg) are more likely to show progression. There is, therefore, a clear link between raised IOP and the development and progression of chronic glaucoma sub-types, although as was noted in the first article, the occurrence of glaucoma is very likely to depend on a balance between the number and degree of potential causal factors and individual susceptibility, thus explaining the variety of clinical presentations and differences in research findings.

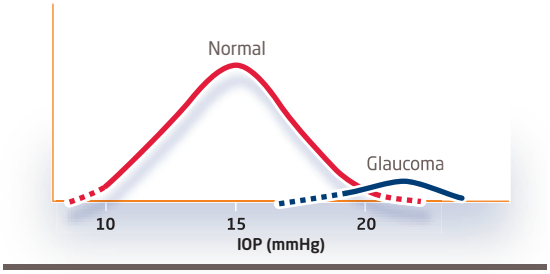

Figure 2 Theoretical distributions of IOP in a non-glaucomatous and glaucomatous population. Note the considerable overlap in IOP values between these populations, demonstrating that no single IOP value will discriminate perfectly between patients with and without glaucoma (From Edgar D and Rudnicka A. *Glaucoma identification and co-management*, Butterworth-Heinemann)

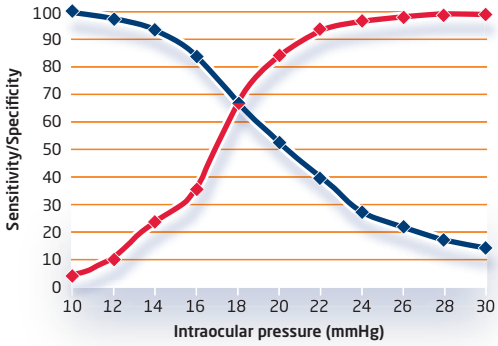

Figure 3 Sensitivity and specificity curves for IOP as a screening test for glaucoma (re-drawn from the data of the Baltimore Eye Survey, Tielsch *et al*, 1991).[2] The curves illustrate the trade-off in test sensitivity and test specificity as the criterion for classifying a subject as glaucomatous/non-glaucomatous is systematically varied. Note that no single cut-off criterion has both high sensitivity and high specificity

IOP and 'screening' for glaucoma

Normal IOP is usually considered in relation to the distribution of IOP in the general population and this distribution is positively skewed (ie towards higher levels of IOP). Figure 2 illustrates theoretical and overlapping distributions of IOP in normal and glaucomatous eyes. Epidemiological studies estimate the mean IOP in normal eyes to be ~15-16mmHg, with a standard deviation of ~2.5mmHg. The statistical upper limit of 'normal' is usually stated to be 21mmHg (a figure which is approximately the mean IOP plus 2 X SD, standard deviations). While it would be convenient if this cut-off value could be used as a simple screening criterion for detecting cases of glaucoma, the sensitivity at this level of IOP is very modest indeed, and of course with the specificity being set at the upper limit of 'normal' IOP from the statistical distribution, the proportion of false positives would always be higher than the proportion of true positives, because the prevalence of glaucoma is less than the prevalence of normal individuals with IOP >21mmHg. Several studies have evaluated the discriminatory power of glaucoma screening tests, including tonometry. Data from the population-based Baltimore Eye Study indicates that no single IOP cut-off criterion has both high sensitivity and high specificity (Figure 3). While specificity is relatively good for an IOP cut-off criterion of >21mmHg, the sensitivity is very limited at ~50 per cent. Tonometry is, therefore, a poor detection test when used in isolation, but fortunately sufficiently high sensitivity and specificity

for effective detection can be achieved by using the combination of tonometry, visual field assessment and optic disc evaluation.[3]

IOP and 'case-finding'

As discussed above, IOP is a key risk factor for COAG, and the higher the IOP, the greater the risk. However, referrals by optometrists need to be made following assessment of other test data and with full knowledge of the patient's risk factors. In view of the variability of IOP measures (considered in more detail below), it is better to repeat tests in borderline cases. While there are no national referral criteria applicable to primary care optometry, and clinical decision making involves the full complement of patient data, the following are important considerations:

- When repeated measures of the IOP are >28-30 mmHg, referral to a glaucoma specialist is justified even if the optic nerve head and visual field are normal, because at this level of IOP commencement of treatment is very likely on the basis of risk of glaucoma development
- Consider the risk of a central retinal vein occlusion. IOPs >35mmHg require an urgent referral (ie within days). In rare cases of exceptionally raised IOP, a same day emergency referral is indicated, regardless of the state of the AC angle
- Consider the inter-eye difference in IOP. An inter-eye difference of <4 mmHg is regarded as normal, 5-7 mmHg is suspect and >8 mmHg is usually abnormal without an explanatory factor (eg a deeper AC in one eye on account of pseudophakia reducing the IOP unilaterally)
- The diurnal variation of IOP means that recording the time of day for the IOP measure (both on the record card and also in any referral letter) is informative.

TABLE 2
Summary of the demographic, clinical and other patient characteristics that might influence IOP in the longer term

Source of variation	Impact on IOP
Age	IOP rises with advancing age, with a rise of 1-2mmHg between the 3rd and 7th decades
Sex	In older age groups, females have marginally higher IOP (1-2mmHg) than men
Race	A higher mean IOP has been reported in those of African or Asian descent compared to those born in America or Europe
Inheritance	IOP appears to be genetically determined, with those with a first degree relative with POAG tending to have higher IOP
Myopia	A reported association exists between myopia and raised IOP
Corneal characteristics	Corneal thickness, curvature, elasticity and hydration all influence IOP (see text)
Systemic disease	An association between systemic hypertension and raised IOP has been reported and some studies have shown a link between diabetes and raised IOP
Ocular disease	Ocular disease can cause OHT and secondary glaucoma (eg PDS), although some conditions can lower IOP (eg acute anterior uveitis or rhegmatogenous retinal detachment)

IOP variation

IOP is determined by the relationship between the rate of aqueous secretion and outflow. The level of IOP is, however, not constant in an individual, being influenced by a number of factors. Demographic, patient attributes and genetic factors influencing IOP in the longer term are summarised in Table 2, while factors that can cause short-term fluctuations in IOP are summarised in Table 3. Awareness of these issues can alert clinicians to the possibility of confounding factors, which in turn will assist in making clinical decisions about individuals. If there is concern that these factors might have influenced IOP, repeat readings should be taken.

Corneal characteristics

An understanding that the properties of the cornea influence an estimate of IOP has been recognised for many years, yet it is only in recent years that this matter has received more widespread clinical attention. For example, an IOP reading higher than the true IOP will be recorded in an individual with a thicker than average central cornea because such a cornea offers more resistance to flatten-

ing in comparison to an average cornea, whereas an IOP reading lower than the true IOP will be recorded in an individual with a thinner than average cornea. Similarly, a steeper than average cornea, also having greater resistance to applanation, results in a relative over-estimation of the true IOP (~1mmHg/3 dioptres), with the opposite being true for a flatter than average cornea. In relation to corneal thickness, pachymetry is an important adjunct to the assessment of OHT and glaucoma, not only in order that estimates of IOP can be 'corrected' for central corneal thickness (CCT), but also because it is recognised from OHTS that a knowledge of CCT provides information about an individual's risk of developing glaucoma. No single correction factor is universally agreed upon, although clinically an error range of ~0.2-0.7mmHg per 10µm difference from an average central corneal thickness has been suggested.[4] However, CCT alone does not completely explain the measurement errors that occur in applanation tonometry, because it only represents one aspect of corneal biomechanics. An example of why accounting for CCT alone would sometimes be misleading is in the case of corneal oedema, whereby the cornea is thicker, yet likely to be softer. Such corneae offer low resistance to flattening and result in an underestimate in IOP, rather than the over-estimate expected on the basis of CCT alone. Alternative forms of tonometry (see below) that might obviate the need to undertake pachymetry are in their relative infancy, and it may take several more years of research to determine their potential clinical role.

Diurnal variation

The diurnal variation is typically 5mmHg in normal eyes, but is higher in patients with OHT or glaucoma, with a diurnal variation of >10mmHg being usually considered to be pathological. The characteristic mid-to-late afternoon depression in IOP might be more significant in males (Figure 4). In clinical practice, IOPs are usually recorded at a single point in time, and a large diurnal variation may result in a failure to detect raised IOP. While there are no simple solutions to this problem, patients with borderline IOP should have repeat tonometry at an alternative time of day (preferably in the morning), in order to better inform the clinical decision. In theory, because the majority of patients' IOP peak is in the morning, carrying out tonometry early in the morning, especially in males, would reduce the number of false negatives. However, the feasibility of such a policy for population glaucoma screening or case finding might be problematic. For diagnosis, phasing (where IOP is monitored at different times during the course of the day, usually within the hospital) is considered important in the assessment of some 'at-risk' patients. Indeed, phasing is essential in order that some cases of COAG, initially appearing as the NTG sub-type, are not erroneously classified as such by the failure to detect raised IOP. Phasing is also needed to assess the level of IOP control in some previously diagnosed and treated patients (eg patients with progressive GON or visual field loss in the presence of apparently 'satisfactory' IOP).

TABLE 3
Summary of factors causing short-term fluctuations in IOP

Source of variation	Impact on IOP
Arterial pulse	Causes a 2-3mmHg oscillation in IOP ('ocular-pulse') due to the cyclic variation in IOP with the heartbeat
Time of day (diurnal variation)	IOP usually higher in the morning and lower in the afternoon and evening. A variation (which may be due to diurnal changes in plasma cortisol) of ~5mmHg is typical in normals.
Contraction of intra-/extra-ocular muscles	Contraction of ocular muscles can increase IOP, although sustained accommodation can increase outflow and lower IOP. Blinking/hard lid-squeezing can increase IOP, although repeated squeezing would potentially lower IOP
Fluid intake	Alcohol has been shown to lower IOP Caffeine can cause a small rise in IOP Drinking a large quantity of water (~1litre) increases IOP
Blood pressure, posture and exertion	IOP is increased (by 0.3-6mmHg) when changing from a sitting to a supine position, with this postural change being greater in glaucoma. An inverted position is even more likely to increase IOP due to elevated episcleral venous pressure Exertion can increase or decrease IOP. Aerobic exercise is usually followed by a drop in IOP and non-aerobic exercise, such as weightlifting is also followed by a drop in IOP, although during the act of heavy weightlifting IOP is increased When air is forced against a closed windpipe (Valsalva manoeuvre) IOP tends to rise. Valsalva manoeuvre commonly occurs when a person coughs, vomits, plays a resistive wind instrument or does heavy weightlifting A tight collar or tie can increase IOP by up to 4mmHg

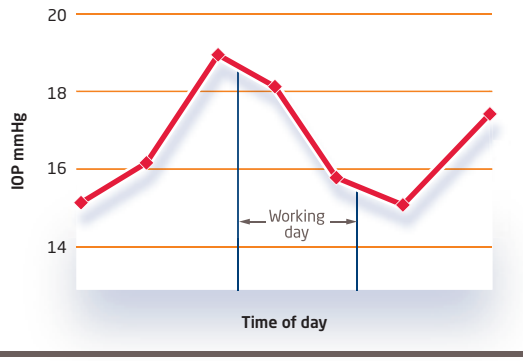

Figure 4 Schematic representation of diurnal variation in IOP. In this example, IOP varies between an approximate 'low' of 15mmHg and an approximate high of 19mmHg, with the peak pressure being recorded in the morning

Figure 5 Goldmann applanation tonometer

Tonometry

Tonometers are classified in simple terms into those that applanate the cornea and those that indent the cornea, while some instruments are more difficult to classify (eg Tonopen). Indentation tonometry is mainly of historical interest, and is not given further consideration here. Applanation tonometry is based on the Imbert-Fick law, which, when applied to the eye, states that IOP is equal to the weight applied to the cornea (in grams) divided by the applanated area (in mm^2). In strict terms, this law is true only for a spherical container with an infinitely thin, elastic, flexible, and dry limiting membrane (ie a container ▶

without resistance to flattening and allowing expansion elsewhere so that container pressure does not alter). The two most widely used applanation tonometers are the Goldmann applanation tonometer (GAT) and non-contact tonometers (NCTs).

Goldmann applanation tonometry

GAT measures the force required to applanate a circular area of 3.06mm diameter. This particular diameter was chosen for three reasons:

- The amount of fluid displaced with such a small area applanated is minimal, and while the eye has some rigidity, the IOP measured is almost identical to the true IOP
- Goldmann's studies showed that for applanated areas of 3-4mm diameter, the surface tension force from the corneal film (ie which tends to pull the tonometer cone towards the eye) is equal but opposite to the force of corneal resistance, and thus the tonometer force is equal to the IOP
- When a diameter of 3.06mm is used, the conversion between tonometer force and IOP is simple, ie force (in grams) x 10 = IOP (in mmHg).

GAT has been regarded as the instrument of choice for many years, since historically the instrument has been widely regarded as the first-choice tonometer for clinical decision making in glaucoma, but GAT has also become the 'gold-standard' or validating criterion against which all other tonometers are compared. The accuracy of GAT in manometric studies is good, provided CCT is close to average, although an early study found repeated GAT measures differed by 2mmHg or more in 35 per cent of cases. Later studies on repeat readings found good within observer repeatability (the standard deviation, SD, of test-retest differences was <1mmHg) although, as expected, poorer between-observer repeatability (the SD of differences was ~1.6mmHg). Similar reliability is expected for the hand-held version of GAT (ie Perkins tonometry). GAT has a robust, simple design (Figure 5), also helping to make it the instrument of choice for glaucoma specialists. Despite these advantages, however, errors can arise in GAT, and these are summarised in Table 4.

TABLE 4
Summary of potential sources of errors in estimating IOP with GAT

Potential source of error	Impact on IOP/solution
Width of tear meniscus	Wider menisci cause slightly higher estimates. Width of semi-circles should be ~1/10th diameter applanated (i.e. ~0.3mm, although they will appear to be ~3mm with 10x magnification)
Vertical alignment of semi-circles	Incorrect alignment can result in a higher reading, because vertical decentration of the prism will require a larger force to match the inner edges of the 'semi-circles'
Eyelids	Eyelids touching the probe can increase IOP, producing an effect similar to that caused by blinking/lid squeezing
Contact time between cone and cornea	Prolonged contact time can cause an apparent decrease in IOP due to 'aqueous massage'. Over a 5-minute period, the IOP can fall by ~3-4mmHg on repeated readings
Cornea*	Astigmatism >3.00D may cause an elliptical area of corneal contact. The flattest corneal meridian should be aligned at 43° to the apex of the tonometer cone.
Calibration	If the GAT is not regularly calibrated, systematic or random errors can result

*Note the effects of other corneal properties (eg corneal thickness, K readings etc) are discussed in the text

Non-contact tonometry

Grolman designed the first NCT which was introduced by American Optical in 1972, although the principle of measurement was considered by Erich Zeiss as early as 1951. There are several different NCTs which are commercially available, including: the Reichert (formerly American Optical) NCT II and auto NCT AT550 and portable PT100 instruments, the Keeler Pulsair EasyEye and Intelli-Puff, the Topcon CT80, the Nidek NT-2000/4000 and TonoRef RKT-7700, the Kowa KT-800 and the Canon TX-F.

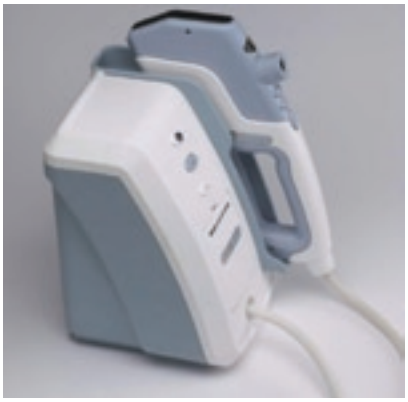

Figure 6
Keeler Pulsair 'Intellipuff' tonometer

In the original NCT, an air-puff is directed towards the cornea and the point of applanation is detected by an optical system. The time taken from the onset of the puff to applanation of the cornea is recorded electronically and this time is related to the IOP. In contrast, the later generations of NCT measure the air-pulse pressure within the instrument chamber at the moment of applanation. This measure has been found to correlate well with IOP, and is less sensitive to the effect of mechanical wear. The portable Keeler Pulsair (Figure 6), in contrast to most instruments, does not require the use of a chin rest for alignment, and being hand-held, can be used in any position. This tonometer creates a ramped air pulse which, at alignment, automatically applanates the cornea. Applanation is detected by an optical system and the sampling of the pulse pressure within the instrument is initiated. A revised and recalibrated version of the Keeler Pulsair (the '2000') was introduced in 1991 and the current fourth and fifth generations of the instrument are known as the Pulsair 'EasyEye' and Pulsair 'IntelliPuff'. The Topcon NCT was introduced in 1988 and has undergone several changes since. This instrument also samples the pulse pressure within the tonometer and converts it to an estimate of the IOP. This instrument uses a lower pulse pressure than the original Reichert instrument, and this particular feature is now common in modern NCTs.

Overall, the main advantages of NCT over contact tonometry have been reported to include the following:

- There is no need for an anaesthetic
- IOP can be recorded rapidly
- Repeat measures are unlikely to change the IOP
- Negligible risk of infection or cross contamination
- Reduced risk of corneal damage
- Operator-independent technique
- Improved tolerance where contact techniques are problematic
- Can be used by trained non-clinically qualified staff.

Some of these advantages could become important limitations if the NCT operator is unaware of the possible sources of error (eg variations summarised in Table 3). For example, with all NCTs it is essential to take at least three to four readings per eye to balance out the effect of the ocular pulse, whereas with GAT, a single measure is usually adequate because the ocular pulse is visible at the point ▶

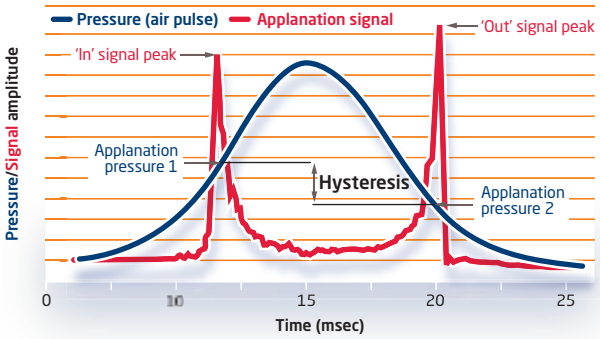

Figure 7 The ORA instrument (left) and measurement signal (above). The ORA records an inward IOP estimate ('Applanation pressure 1') and an outward IOP estimate ('Applanation pressure 2'), estimates that may differ in individuals by an amount referred to as 'corneal hysteresis'

Figure 8 The Dynamic Contour Tonometer (Pascal)

of applanation in the form of an oscillation of the semi-circles, permitting the recording of an average or better still maximum end-point. In general terms, method comparison studies conclude that NCTs provide clinically meaningful measures of IOP which equate to those obtained by GAT, although NCT has not replaced GAT as the technique of choice among glaucoma specialists. Indeed, there is some evidence that the accuracy of NCTs is still doubted by some clinicians, a large proportion of whom believe, at least to some extent, that the instruments 'read high',[5] a finding which is counter to the results from published comparison studies. However, it is likely that in some patients this effect is explained by a statistical sampling phenomenon, known as the 'regression towards the mean effect' whereby, regardless of the form of tonometry used in the primary care setting, patients with high IOPs may not have this high reading confirmed on examination in secondary care, simply due to diurnal IOP variation. For example, while most primary care optometrists use a combination of test results, and not simply IOP, when deciding whether or not to refer a patient with suspect glaucoma, they are, arguably, often presented with a situation where the IOP is raised and the optic discs and visual fields are normal. In this circumstance, an optometrist may use a simple cut-off criterion for referral (eg if IOP >28mmHg, then refer). However, because IOP is affected by a number of sources of variation (eg those listed in Table 3), sometimes a raised IOP value will be measured just by chance, even though the reading will, on average, be lower when repeated, thus lying closer to the mean value of the 'real' IOP than the first reading. Although this form of sampling bias cannot be eliminated, the practice of repeat checking of IOP before referral will help inform the decision, especially in cases when raised IOP is considered to be the primary reason for referral.

Developments in tonometry

Potential errors in measurements of IOP due to variations in the properties of the cornea were discussed briefly above. There are a number of different pachymeters that provide 'correction' for estimates of IOP, although as was stated earlier, there is no single 'correction' factor that has been universally adopted. Potentially of greater significance is the development of instruments that quantify biomechanical properties of the cornea, such as the Reichert Ocular Response Analyser (ORA). This NCT-like instrument (Figure 7) employs a bi-directional dynamic applanation method to provide a measurement, corneal hysteresis (CH), a parameter which quantifies specific viscoelastic properties of the cornea and can determine the total corneal resistance (including the aggregate effects of thickness, rigidity and hydration) to the tonometer force during the measurement of IOP. Results from high quality clinical trials are necessary before evidence-based decisions can be made as to the value of this type of measurement in managing patients, although studies performed to date suggest that CH may be of clinical value.

Dynamic contour tonometry (DCT) is a novel method of measuring IOP based on the principle of contour matching. The DCT tip features a miniaturized piezoresistive sensor built flush with the centre of a concave contact surface of 7mm. The Pascal DCT is a slit-lamp mounted instrument (Figure 8) that is operated in a manner that is similar to GAT. IOP is sampled continuously and both IOP and ocular pulse amplitude outcomes are displayed. It has been suggested that DCT gives measurements of IOP that are not influenced by CCT. Indeed, one recent study suggested IOP values from DCT are closer to manometric levels than those from GAT.

The ORA and DCT are relatively recent developments, whereas pneumatonometry, a method that gives a continuous measurement of IOP from which the magnitude of the ocular pulse can be measured, has a considerably longer history, although a more clinically useful instrument has been available only within the past decade. The relationship between changes in blood volume and the ocular pulse at a known heart rate enables the pulsatile ocular blood flow (POBF) to be estimated, and because decreased POBF pulsatile has been observed in patients with COAG, it has been suggested that the pulsatile component of ocular blood flow might provide useful information on the vascular aetiology of COAG.[6] The OBF tonometer is portable and comprises a small base unit with data storage capability, a sensor (which attaches to a slit lamp) and a printer. The instrument automatically generates a measure of POBF, taking no longer than GAT. While the OBF tonometer has been shown to provide reproducible data, the value of the OBF pneumotonometer as a technique for measuring IOP has been questioned,[7] and, contrary to expectations, based on the theory of measurement of IOP, CCT appears to affect measures obtained with the OBF tonometer more than they affect GAT.[8]

References

A list of references is available from the clinical editor: william.harvey@rbi.co.uk

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MULTIPLE-CHOICE QUESTIONS – take part at www.opticianonline.net

1 Which of the following statements about IOP is untrue?
A Raised IOP can be caused by increased aqueous production
B Raised IOP can be caused by reduced aqueous outflow
C Aqueous outflow is reduced in normal ageing
D The trabecular meshwork and Schlemm's canal are not implicated in the mechanism of IOP elevation

2 Which of the following statements about the trabecular meshwork is untrue?
A Endothelial cell populations lining trabecular fibres are reduced with age
B The cribriform layer of the trabeculum is continuous with the inner wall of Schlemm's canal
C Pathological changes in Schlemm's canal anatomy are the primary cause of IOP elevation
D Trabecular beam thickening occurs with age

3 Which of the following statements best describes the population distribution of IOP?
A IOP is normally distributed with an average of 21mmHg
B IOP is negatively skewed
C IOP is positively skewed with an average of 15-16 mmHg
D IOP is positively skewed with an average of 21mmHg

4 Which of the following statements about use of IOP in screening is correct?
A An IOP cut-off of 22mmHg perfectly discriminates between normal individuals and glaucoma patients
B No single IOP level has high sensitivity and specificity
C The sensitivity of an IOP cut-off of >21mmHg is good but the specificity is poor
D An IOP cut-off of 30mmHg has very high sensitivity

5 Which of the following statements about the findings of the Baltimore Eye Study is incorrect?
A 13% of individuals with an IOP below 16mmHg have POAG
B All individuals with IOP ≥ 35mmHg had glaucoma
C Compared with an IOP <16mmHg, individuals with IOPs of 22-24mmHg are twice as likely to have glaucoma
D 88% of all glaucoma cases had IOPs of ≤ 29mmHg

6 Which of the following statements about corneal characteristics and IOP is untrue?
A A thicker than average cornea will generally have greater resistance to flattening and so the recorded IOP is likely to be higher than the true IOP
B A thinner than average cornea will generally have lower resistance to flattening and so the recorded IOP is likely to be lower than the true IOP
C A steeper than average cornea will generally have lower resistance to flattening and so the recorded IOP is likely to be lower than the true IOP
D Corneal rigidity or 'stiffness' is most likely to have a greater impact on IOP readings than corneal thickness and corneal curvature

7 Which of the following statements about the long-term variation in IOP is untrue?
A IOP rises slightly with advancing age, by 1-2mmHg between the third and seventh decades of life
B In the younger age groups, females have been shown to have marginally higher IOP than males
C People with a family history of glaucoma tend to have higher IOPs
D IOP may be lower than normal in an eye with acute anterior uveitis

8 Which of the following statements about the short-term variation in IOP is untrue?
A The arterial pulse causes a cyclic variation IOP of ~6mmHg
B The IOP is on average higher in the morning and lower in the afternoon and evening
C A tight collar or necktie can increase IOP by up to 4mmHg
D Drinking a large quantity of water will tend to increase IOP

9 In respect of diurnal variation in IOP, which of the following is correct?
A Diurnal variation is typically 10mmHg in normal eyes
B The characteristic lowering in IOP from mid-late afternoon is often more pronounced in males
C The primary source of diurnal variation in IOP is measurement error inherent in tonometry
D Phasing refers to the practice of measuring the variation in IOP when supine, sitting and standing

10 In respect of the Goldmann Applanation Tonometer (GAT), which of the following is untrue?
A The GAT is a variable force, fixed area, contact applanation tonometer
B A prolonged contact time between the probe and the eye can cause an apparent drop in IOP
C Wider menisci cause slightly higher IOP estimates
D GAT measures the force required to flatten an area of 3.06mm

11 In respect of NCT, which of the following statements is correct?
A The first NCT was introduced by American Optical in 1951
B Method comparison studies show that modern NCTs tend to provide accurate estimates of IOP
C When an optical system detects the presence of corneal indentation, the pulse pressure within the instrument is sampled
D An average of 3-4 readings is required with an NCT because of limited accuracy and 'over reading' of IOP

12 In respect of developments in tonometry, which of the following statements is correct?
A IOP readings with dynamic contour tonometry (DCT) are more influenced by central corneal thickness than those estimated with the OBF tonometer
B The Ocular Response Analyser (ORA) is a combined tonometer/pachymeter that 'corrects' IOP for central corneal thickness
C The ORA measures 'corneal hysteresis', a measure that is believed to reflect the aggregate effects of corneal thickness, rigidity and hydration
D Pneumatonometry permits the simultaneous and continuous recording of IOP and systolic and diastolic blood pressure

Successful participation counts as two credits towards the GOC CET scheme administered by Vantage and one towards the Association of Optometrists Ireland's scheme. **The deadline for responses is April 17**