Optimum VA

The Official Newsletter of the
National Association of Veterans Affairs Optometrists

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ISSUE HIGHLIGHTS

Low Vision and Traumatic Brain Injury Optometric Workgroups

As you are all acutely aware, the Low Vision Optometric and Blind Rehabilitation services are significantly expanding across the nation. We have support systems in place for both the newly hired VA Low Vision Optometrists and those VA Optometrists currently practicing Low Vision. The support is two fold. There is a bi-monthly conference call and a national e-mail group. The calls happen every other month, on the third Monday of that month at 4:00pm Eastern time. The next call is scheduled for Monday July 21st.

This group has worked together to write Low Vision Eye Templates, Low Vision Encounter Forms, Prosthetics guidelines and lists. We have a very open agenda....it is solely meant to be a support for us all.

We would like to extend an invitation to any VA Optometrist interested. Please assist us in extending this invitation to the new hires!! Please e-mail Dr Kara Gagnon if you wish to be involved in this group!

As you are aware, the visual consequences from TBI are much more in the fore front than ever before. Many of you are involved in the screening, diagnosis and treatment of these visual anomalies. Dr. Townsend has recognized the need to offer support and resources to us all. He has worked on national policies hoping to direct the nation in our long-term goals. In the short-term he supports the need for a VA TBI Optometric Workgroup. Much like the Low Vision Optometric Workgroup, the TBI Workgroup will convene on a bi-monthly conference call and have available a national e-mail group. The calls will happen every other month, on the third Monday of that month at 4:00pm Eastern time. Therefore monthly, there will either be a Low Vision Optometric Conference call or a TBI Optometric Conference call.

Many of you have already requested inclusion in this group. Your names have been placed on the e-mail group and will soon receive correspondence. The first call is scheduled for August 18th at 4:00pm EST.

We would like to extend the invitation to all VA Optometrists who may not have been aware of this support system/working group. The patient population demands pooling of resources and collaboration!

Thus far, the assistance and support the more experienced Optometric doctors have lent to the nation in attempts at best serving these brave young men and women has been remarkable.....it typifies attributes such as honor, valor, altruism...which make us all so proud to work in the VA System. This workgroup will facilitate such support.

Please e-mail Dr Kara Gagnon if you would like your name added to this group!

PROMOTION INFORMATION SHEET: NAVAO MENTOR PROGRAM

Association of Military Surgeons of the United States (AMSUS)

By Joseph F. Molinari, O.D., M.Ed., Col, USAF (Ret) & Lyman C. Norden, O.D., M.S., Lt Col ANG (Ret)

Thank you for allowing us to contribute to your request for information regarding the organization Association of Military Surgeons of The United States (AMSUS). When asked to compose this report I (JFM) counted the Baxter AMSUS mugs given out every year at the meeting and there were 28. Therefore, we have both been members for that period of time. This organization is "The" federal health care organization established in 1891 and is the only one incorporated by Congress in 1903. It spans all specialties in federal health care and all agencies. Initially, optometry was represented in the organization as the Air National Guard Optometry program which both of us served until the Optometry Section was established from both our efforts in 1986. This section is one of the newest sections in the organization and is searching for members to expand in the parent organization. The first Optometry Outstanding Service and Recognition Award was established and funded by Bausch and Lomb and given in 1994 and one of us (LCN) was the first recipient. It is now designated as the Adm William E. Sullins Award. We have had at least three VA optometrists to win this prize of one thousand dollars. Noted names besides Dr. Norden are Robert Newcomb, Murray Fingeret and John Townsend. This award has been given each year since at the annual AMSUS banquet held at the annual meeting.

The Veteran Health Affairs has been represented since 1935. Dr. Charles M. Griffith was the first VA representative to be the AMSUS president in 1935-36 and presided over the 44th annual meeting in Detroit in 1936. Since that time the VA Undersecretary of Health has rotated with the other service surgeon generals to occupy this position in a regular order. The VA's last time was 2006. We have participated in the last four meetings that the VA hosted at AMSUS. We must say that our programs have been second to none, compared with the other sister services, but we are highly biased. There are currently 225 VA members in AMSUS and the VA has a full day meeting, and a lunch, usually on Wednesday of the week long meeting. Usually at the lunch you can get to meet with the undersecretary and discuss topics in the VA. As of this writing there are only a few optometric members (less than 15). Benefits for the organization are:

AMSUS members can benefit by a unique educational reference perspective into what's going on in military medicine today. Our journal, Military Medicine, is a member benefit and is published monthly (http://www.amsus.org/journal/). The AMSUS Newsletter is also a member benefit, and is published quarterly (http://www.amsus.org/membership/newsletters.shtml). Another educational resource available to members is the AMSUS Online Resource Library, http://www.medinfonow.com/min/default.aspx?qs=C64EDEBEBC27B070969E779B1BFE1073511A40F1048D9FCF. Each year the Association of Military Surgeons of the United States (AMSUS) sponsors a meeting for healthcare professionals, with a focus on the activities of the health agencies of the United States Government. American attendees include military and civilian health care professionals of the Armed Forces, Public Health Service, and Department of Veterans Affairs. This year's meeting will be held 9-14 November in San Antonio,

Texas. At the Annual Meeting, attendees get both educational and networking benefits. Member discounts on Annual Meeting registrations is a popular member benefit.
For a whole list of member benefits, check out the AMSUS website at:
http://www.amsus.org/membership/benefits.shtml

The annual dues are 55 dollars. If anyone would like to discuss anything to do with AMSUS we would be happy to talk with them. You need to belong to this organization.

Understanding Corneal Hysteresis

By Ken Myers, O.D.

Introduction:

Many mechanical devices behave differently when they reverse their direction of motion. An extreme case are the spike rollers at rental car return lots that offer little resistance “entering” but rip your tires off “exiting”. A more common case are pneumatic door closures on office and hotel entrance doors that react differently to a push-in than a pull-out. Engineers describe both devices as exhibiting hysteresis. A material reacting differently being moved in one direction than in the reverse direction is said to show hysteresis and the more asymmetrical the reactions, the higher the hysteresis. Other definitions refer to “lagging” such as the device lags behind when made to reverse its direction of motion. Underlying either definition is the recognition the material exhibits a change in basic behavior while completing a full cycle of motion (a round trip); that the material reacts differently in one direction than the other in traveling back to the starting position.

There is no one standard unit of hysteresis since it is measured in whatever variable best describes the materials’ reaction to a change in direction of its motion. For example, roller spikes could have hysteresis measured in “tires” since the net result of an in and out, round trip is 1 or more tire losses. The pneumatic door closer could have its hysteresis measured in pressure since the pressure to push-in is more than the pressure to pull-out” in one open-close cycle. In general, we shall see that any material with both elastic and viscous properties, like the cornea, exhibits hysteresis.

While a commonly known and measurable characteristic of mechanical and electrical devices, little, if any, attention as been paid to the fact human tissues exhibit hysteresis and that abnormal hysteresis values can indicate disease or dysfunction. During a basic physical exam clinicians have long poked and prodded looking for an abnormal tissue response, rebound, or feel, without realizing they were, if fact, testing tissue hysteresis.

As far as I can determine, the first human tissue to have its hysteresis recognized and measured as such is the cornea; a viscous-elastic tissue. Although living organisms consist of many tissue types, practically all medical testing has centered around either anatomical studies (scans) that look for structural alterations or chemical analysis (lab studies) but there has been little study of how tissue reacts to external displacement forces (like tonometry). That the cornea is the first to have its hysteresis measured may not surprise us because it has been subjected for over a century to various types of external palpations (beginning with finger pressure) to diagnose glaucoma. This paper explains how efforts to better understand non-contact tonometry (an external displacement force) led to the first measurement of corneal hysteresis and the Ocular Response Analyzer (ORA).

Glaucoma studies led to the invention of the ophthalmoscope that first allowed examiners to visualize the living eye's interior to better understand why some became hard, red, inflamed and then blind. This led to new ocular observations of disc cupping and pallor, previously unknown. Tonometers were next developed to quantify another unknown and un-measurable ocular characteristic, IOP. And now the ORA measures corneal hysteresis, H, a third previously unknown and un-measurable ocular characteristic that, like ophthalmoscopy and tonometry is yielding new, measurable risk factors for several ocular disorders and diseases.

<u>Corneal Hysteresis:</u>

Corneal tissue was long considered to be purely elastic. It supports metabolism without vascularization using aqueous and tear films to supply nutrients and transport waste and maintains transparency by lamina arrayed uniformly at the optical level. But very little was known of its biomechanical properties until the Ocular Response Analyzer (ORA) appeared a few years ago.

When Goldmann developed GAT he assumed the cornea was a perfectly elastic membrane of standard and uniform thickness and curvature. Recent findings of inaccurate (reduced) GAT values post-LASIK show his purely elastic corneal model was incorrect and GAT errors result not from corneal thickness reduction but from lowering a new, global corneal property, hysteresis. A purely elastic cornea has zero hysteresis whereas one with both elastic and viscous properties does and its hysteresis increases or decreases as its tissue becomes more or less viscous.

The ORA shows the cornea has both elastic (spring-like) <u>and</u> viscous (shock absorbing) properties with its viscous nature only becoming evident as corneal forces are rapidly applied and removed. Its elastic-viscous nature explains why the cornea responds to GAT (a slow force) as mainly elastic tissue but as an elastic-viscous tissue (having hysteresis) during rapid NCT forces. If Goldman's purely elastic model had been correct the cornea would react identically to GAT and NCT forces, post-LASIK GAT readings would remain accurate and corneal tissues would not exhibit viscous damping and hysteresis.

The presence of hysteresis indicates the cornea has both elasticity and viscosity. Hysteresis values result from the interplay of the many corneal properties rather than a single static property such as center thickness and reflect overall, dynamic, corneal physiology.

While developing the ORA and in understanding corneal hysteresis, Grolman and Luce also derived new IOP measurements, Icc and Ig, (see later) and it became evident that H values reflect overall corneal integrity and can serve as risk factors for glaucoma, keratoconus, Fuch's and post-LASIK ectasia. Hysteresis values thus provide insight into the biomechanics and health of the cornea in a manner not previously measurable.

This paper explains the origin of corneal hysteresis, H, and how NCT data "ghosts" led to both two new IOP measurements (Icc, Ig) and the understanding and measurement of corneal hysteresis H.

The first explanations of H are qualitative, using analogies to familiar clinical situations viewed in a new light, while the second explanation is more quantitative.

QUALITATIVE EXPLANATION

Tissues With Elastic and Viscous Properties Have Hysteresis:

Many materials exhibit mostly elastic or viscous behavior. A tennis ball is mainly elastic while a putty ball mainly viscous. Dropped on a floor, an elastic tennis ball rapidly rebounds while a viscous putty ball "splats" on the floor with little (or no) rebound. Their degrees of elastic and viscous properties are revealed by how quickly (and high) they rebound after being deformed by external forces. High speed photography is needed to show how a golf ball is rapidly deformed at impact and rapidly rebounds from the golf club whereas a beach ball can be seen by the eye to be indented after being struck because it rebounds much slower.

Squeeze an elastic tennis ball until you slightly flatten it under thumb and forefinger and then reduce pressure. The flattened surfaces rebound so rapidly they stay in contact with your thumb and finger as you release pressure. But, squeeze a viscous putty ball and it stays flattened (or rebounds very slowly) after you release pressure. Squeeze an elastic-viscous sponge ball and it regains its spherical shape slowly enough to be seen by the unaided eye. The sponge ball's rebound speed (relaxation time) lies between that of the elastic tennis ball and the viscous putty ball. Elasticity produces a rapid rebound but viscous dampening causes a slower rebound. With the elastic tennis ball, rebound is so rapid you can not remove your fingers fast enough to see the rebound (or that of the golf ball) and high speed photography is needed to see rebound whereas an elastic-viscous sponge ball has such a slower rebound it is visible in real time.

In this way the relative mix of elastic and viscous properties of a material is revealed by measuring how it reacts to the application and release of an external pressure.

For example, if one pays close attention it is found that upon reducing finger pressure the tennis ball continues to push back whereas the sponge ball pushes back with far less (or no) pressure. Release an indented sponge ball fast enough and you feel little or no back pressure because your fingers retreat faster than the ball's rebound speed and lose contact. In the extreme case of a purely viscous ball of putty the fingers feel no pressure once released since the putty does not rebound at all.

One dictionary root of hysteresis is "to lag" and an elastic- viscous material whose rebound after deformation "lags", exhibits hysteresis. The amount of rebound "lag" reflects the material's ratio of elastic to viscous behavior with a high ratio indicating rapid rebound, little lag and low hysteresis and a low ratio producing slow rebound, large delay and high hysteresis. The rebound lag (hysteresis) of a material therefore reflects the dynamic interaction of its many physical parameters and characterizes its physiologic state as an organic whole. Importantly, a material's hysteresis can not be predicted by individual or multiple static measurements since it results from the dynamic interplay of all these properties and on the rapidity of the forces applied.

As a result, hysteresis values seldom correlate well, if at all, with individual physical properties like thickness because it results from all properties interacting dynamically. Corneal hysteresis reflects overall corneal integrity and correlates weakly to an individual property like center thickness, curvature, diameter or hydration because it arises from their dynamic interactions. Measuring corneal hysteresis therefore offers a new, valuable measurement of total, organic corneal status and recent studies show abnormal H values are linked to some corneal and ocular dysfunctions.

Specific Examples of Tissue Hysteresis:

Many components of the "physical exam" unwittingly test tissue hysteresis while "poking and probing" to evaluate how tissues "feel" and rebound.

Low Hysteresis Tissues

Consider chest or skin palpation or tendon reflexes in this light. The tissues being indented are, if normal, almost purely elastic and immediately rebound with little rebound. These tissues exhibit low hysteresis. While not explained this way during training, percussion tests gauge tissue hysteresis. Many external tissues, if normal, are very elastic, have little viscous damping and exhibit low hysteresis as shown by their rapid rebound. On the other hand, a fresh friction blister (see photo) is very taut and if compressed, immediately rebounds showing little hysteresis so in this case low hysteresis indicates non-normal skin. But tissue hysteresis values are of diagnostic use only if their normal values are known so that low or high values indicate abnormal tissue.

Poor Man's Corneal Model

A fresh blister has viscous-elastic tissue with a faster rebound time than pitting edema but much slower than cornea tissue. The blister will rebound to Q-tip indentation faster than it can be withdrawn and pitting edema is not visible to the naked eye. But, as the blister ages its outer covering "toughens" and the entrapped fluid beneath becomes more viscous (thicker) and rebound time increases to where a rapidly withdrawn Q-tip leaves a brief indentation apparent to the naked eye, exhibiting visible hysteresis.

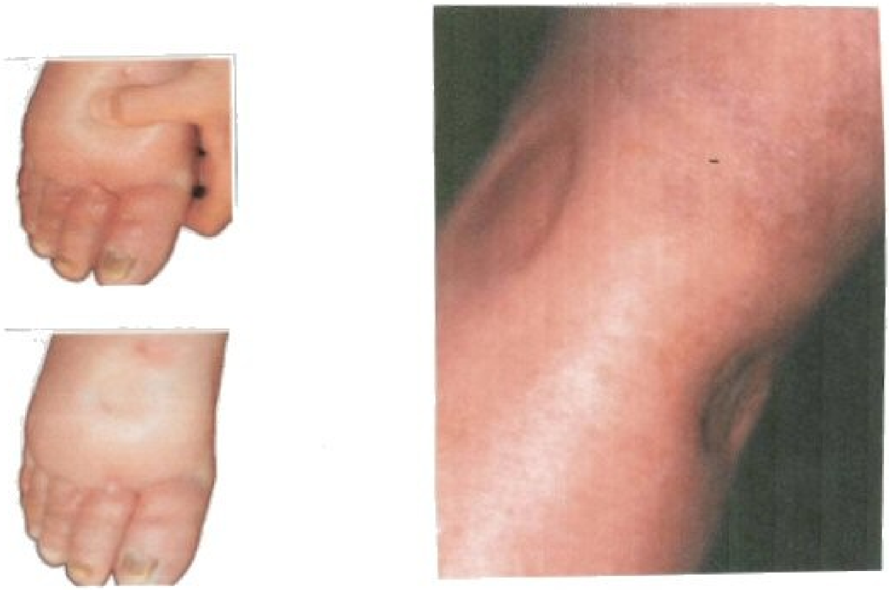
One way to differentiate between retinal schisis and frank detachment is to note the former reacts rapidly to slight eye movements as a taut, elastic tissue whereas the latter reacts slowly (waves) due to its more viscous tissue.

<u>High Hysteresis Tissues</u>

Tissues with excess interstitial fluid become more viscous. Palpate them (the foot and arm shown) and they rebound slowly, like the sponge ball. Tissue viscosity is higher due to fluid retention and hysteresis is higher. The slower the tissue rebounds the greater the edema, retained fluids and hysteresis. Tissue rebound time could be recorded to accurately grade edema (and hysteresis) rather than the cruder +1, +2, +3 now used. The examiner could also press a pressure gauge in and out against the tissue and note the higher pressure "pushing in" then when "relaxing out" as the tissue lagged and exerted less pressure "going out". Hysteresis would then be the pressure in minus pressure out difference or Pin-Pout.

The units used to measure H depend on the method used. For pitting edema either time of rebound or difference between pressure "in" and "out" could be used. With either, higher H values indicate a higher ratio of tissue viscosity to tissue elasticity. Tissue edema usually produces increased tissue viscosity and hysteresis values and when extreme, rebound is so slow pitting edema results as shown in the figures.

PITTING EDEMA



The left photos show foot pitting edema from lymphedema. Excessive interstitial fluid increased tissue viscosity and lowered tissue rebound pressure so much the indentation remains after thumb pressure was removed. A pressure gauge under the thumb would have read higher during push in and much lower during removal and zero if the thumb is withdrawn quickly as is usually the case. When the thumb is removed quickly the tissue lags behind from increased viscosity and loses contact. The third photo shows arm pitting edema from being gripped by thumb and forefinger some time earlier.

Both tissues show slow rebound or lagging (hysteresis) because their rebound pressures are so low they fell behind thumb withdrawal. The amount of hysteresis depends on the amount of interstitual water that increased tissue viscosity and lowered rebound speed.

A pressure sensing "thumb" could quantify hysteresis by comparing resisting pressure in versus resisting pressure out. A difference indicates hysteresis. But a "thumb" speed slower than tissue rebound speed detects no hysteresis or pitting edema (no pit is left). To accurately measure if hysteresis is present, "thumb" speed must be appropriate to the tissue's rebound speed; that is, not too slow nor too fast.

Speed Is Critical

The speed of the indenting pressure is important. If an examiner's finger probes tissue too slowly, pitting edema will not be evident because the tissue will remain in contact with a very slowly retreating finger and no "pitting" will be seen. Indenting pressure release must be less than tissue "relaxation time" for hysteresis to be detected and measured. For example, to see how a golf ball rebounds when struck, high speed photography is needed because contact time with the club face lasts only milliseconds.

Since materials vary widely in their ratio of viscous to elastic properties, their rebound speeds, relaxation times and hysteresis values also vary widely, so to measure hysteresis the cycle time of the probing force can not be longer than tissue relaxation time. Because corneal tissue has a relaxation time in the millisecond range its hysteresis measurement requires application of a similarly brief external force.

The NCT Ghosts

To appreciate why the first human tissue to have its hysteresis measured was the cornea we review tonometry with the knowledge corneal tissue has elastic and viscous properties producing a relaxation (rebound) time of milliseconds.

Past Corneal Research:

For over a hundred years, corneal studies dealt with topology and chemistry; cell type counts, curvatures, thickness, diameters, hydration, cell generation and oxygen transport. As expected, these showed variations from average that became useful in clinical care. But, with the exception of oxygen transport, these dealt with a static cornea (anatomy). It wasn't until tonometry that forces acted on the cornea during examination but how the cornea reacted to them was not understood.

A New Corneal Science:

The ability to make accurate measurement of relevant physical properties is the basis of scientific knowledge. Both a relevant property and its accurate measurement are required. Corneal "hysteresis" was discovered and measured by Bernard Grolman, O.D. (NCT inventor) and his collaborator David Luce Ph.D., of Reichert Instruments. Mixed with discovering corneal hysteresis was their discovery of new ways to determine IOP (IOPcc and IOPg) and another new parameter Corneal Resistance Factor. Each is determined by the ORA and discussed in turn.

Past corneal measurements were akin to measuring the shape of a bell…without striking it to hear the ring. Static measurements can not accurately predict how a material reacts to moving forces just as the examiner does not just look but also "pokes and prods" human tissue searching for abnormalities.

Until NCT, the cornea's biomechanical (dynamic) properties remained hidden and its response to a brief force unknown since GAT measures IOP with the cornea essentially at rest. Grolman and Luce came to realize the NCT, by using a brief impulsive air pulse, had been measuring hysteresis since its introduction in 1971.

GAT and NCT Tonometry:

In designing his tonometer, Goldmann assumed the cornea was perfectly elastic in order to use static, stress-strain equations of thin spherical membranes. His assumption was appropriate because GAT slowly deforms and holds the cornea motionless while IOP is measured over a flattened 3.06mm diameter area. GAT is static and his assumption about elasticity was defendable whereas his others were not: corneas do not have the average composition, curvature and thicknesses listed in anatomy books and have viscous damping.

The very brief 25-35 msec NCT measurement puts the corneal apex in rapid motion--first in and then rebounding out-- unlike GAT that takes 2-5 seconds. During NCT the cornea passes through applanation while being pushed in by rising air pressure and a second time, as the cornea rebounds out as air pressure decreases. (NCTs use air pressure at inward applanation to determine IOP.)

GAT measures with the cornea at rest while NCT measures with the cornea in motion so it should not surprise one the cornea reacts differently to GAT and NCT and that GAT encounters little corneal viscous damping while NCT encounters a lot.

Despite this basic difference, decades of clinical trials showed the American Optical-Reichert NCTs meet ISO tonometry standards (are equal to GAT accuracy).

Some do not understand GAT, NCT or the ISO tonometry standards or how to check calibration of either tonometer and some believe NCT is less accurate despite the extensive literature and ISO certification.

This lack of understanding caused “myths”; that if NCT and GAT disagree, GAT is the “standard” though there are more ways for GAT to err. That NCT does not applanate (high-speed film shows it does). GAT and NCT are both excellent instruments and deserve their wide spread usage. That they occasionally disagree does not necessarily mean one is in error as their measuring techniques are influenced by different corneal properties and one or the other is sometimes less accurate. And, without a 3rd, absolutely perfect tonometer it remains impossible to determine which is more accurate when they disagree.

Ghost Data In The NCT:

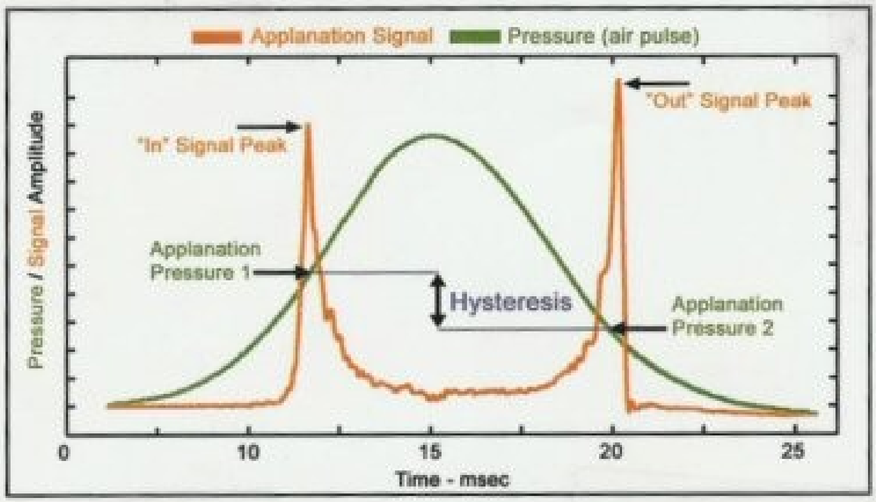
For some years prior to death, Grolman re-examined recordings of the internal signals generated during NCT measurements (Figure A) and their peculiarities nagged him.

The NCT monitors air pressure (green curve) as it rises and falls and at the two applanation points in each operation cycle---the cornea is applanated flexing in, briefly becomes concave until the air pump switches off, then rebounds back through applanation again as the air pulse decays to ambient pressure. NCT has access to two IOP readings---IOPin and IOPout--- but was calibrated using IOPin. All NCTs use just IOPin as the correlate to IOP and to see the Figure A curves the instrument must be opened and connected to an oscilloscope.

The red curve records the amount of infrared light reflected from the corneal apex and its two spikes signal the two applanations when reflected infrared light is maximal because the cornea apex is applanated (flat).

The second applanation---IOPout--- during corneal rebound results because the air pump can not instantaneously stop at first applanation and, for a few milliseconds, continues to pump, pushing the corneal into concavity during which reflected IR light decreases as shown. Then, as air pressure falls, the cornea rebounds through applanation a second time, generating the second red spike and IOPout air pressure measurement.

Each time NCT operates an internal Fig. A "signature" displays how the cornea responded to the standardized, brief air pulse. Because the cornea undergoes a brief deformation, its response depends not on a single corneal property like thickness (as assumed by Goldmann) but on all corneal parameters… thickness, hydration, curvatures, viscosity, diameter, interacting dynamically. In other words Fig. A reflects the dynamic response of the organic elastic-viscous cornea and the details of its rebound to a standard brief impulsive force. This means the shape of the red curve reveals the details of how the cornea rebounds and this, we know, is a result of corneal hysteresis.



Ocular Response Analyzer Measurement Signal

Figure A

A typical NCT operating signal showing rise and fall of external air pressure (green) impinging on the cornea and the peaks of reflected IR light (red) at first and second applanation when reflection maxmizes. Between these peaks the cornea is concave and IR reflection very low. A cycle lasts only 25 milliseconds and pressure 1 is always higher than pressure 2. Grolman and Luce came to realize this results from cornea tissue having viscous-elastic properties rather than elastic properties. Corneal tissue reacts differently if the pressure acting on it changes direction (tissue hysteresis).

For a viscous-elastic tissue, the pressure 1 (moving in) minus pressure 2 (moving out) it exerts at the same position, represents the degree of hysteresis much as a thumb causing pitting edema encounters higher pressure during tissue indentation than rebound. The in vs. out pressure difference at any corneal position could be used but the position at applanation is easily located by the IR signal spikes.

Eyes with identical IOP can have different values of hysteresis and low values are considered a risk factor for LASIK and an indicator and poorer prognostic sign of certain glaucomas as well as screeners for Fuch's and keratoconus.

The Nagging Puzzle:

It nagged Grolman that eyes with the same IOPin sometimes had very differently shaped Figure As and that the second applanation ---IOPout---always ocurred at a lower air pressure than IOPin and that eyes with the same IOPin could have different, but always lower values of IOPout.

Over several years Grolman and Luce came to understand Figure A revealed that each cornea reacted not as the purely elastic tissue assumed by Goldmann but as an elastic-viscous tissue whose rebound depended on each cornea's particular combination of elastic and viscous properties. This meant Figure A could be used to reveal not just IOP but other corneal properties including rebound lag---hysteresis. Figure A reveals how the cornea reacts to a very brief impulsive force and, since the interaction time is very brief (think of the golf ball and club face) only an electronic signal can capture it since it lasts only a few milliseconds.

Fig. A therefore details how each cornea reacts to a "palpation" that take place almost as fast as corneal rebound or relaxation time. Once applanated, the NCT air pulse decreases a hair faster than the cornea can rebound and the corneal reacts somewhat like a sponge ball due to its viscous nature and lags behind. Thus, when the second applanation occurs, the air pressure is below that at first applanation . Just as with pitting edema or the sponge ball when the examiner's finger felt less pressure while being removed, the NCT measures a lower IOPout because the corneal tissue lags behind the decaying air pulse due to viscous damping.

This is why timing is important in measuring tissue hysteresis. Because corneal rebound (relaxation) time is so brief, a very brief force must be used or else the corneal tissue will not show any lag (hysteresis) during rebound. (Think of measuring pitting edema for a tissue with a relaxation time of milliseconds.)

Stated another way; during NCT air pressure rise, both corneal elasticity and viscous drag resist the deformation but, as the air pressure rapidly decays and corneal elasticity pushes the corneal out, corneal viscous drag slows its rebound such that by the time the second applanation is reached the air pressure is below its value at first applanation. This is why IOPout is always less than IOPin and their difference measures hysteresis. This is similar to pitting skin edema or a sponge ball where the probing finger feels more pressure "going in" than "going out".

Grolman and Luce found hysteresis explained these puzzles posed by Figure A.

1. For every eye, air pressure at applanation #2 (rebounding) is below pressure #1 (going in).
2. Eyes with same IOP can have differently shaped Fig. As.
3. Two eyes of the same, normal subject exhibit similar Figure As.

Each cornea is uniquely characterized by its Fig. A in detail and can be "summarized" by four parameters of which only one was previously known.

1. Standard NCT IOP #1 measured "going-in".
2. How much IOPout lagged IOPin….Hysteresis
3. The shape of Fig A.

4. Corneal Resistance Factor (to be explained).

While eyes may produce different “corneal prints” due to normal variations, it always takes more pressure to applanate flexing in than when flexing out. IOPout always measures less than IOPin and this is the value of corneal hysteresis in mmHg.

No one had observed this with GAT because it’s reading is taken with the corneal surface at rest during applanation. GAT readings are, if looked at in detail and gear “lash” accounted for, identical whether one reads IOP at applanation while approaching from “below” (smaller mires-flexing in) or from “above” (larger mires, flexing out) because GAT is a static measurement unaffected by corneal viscous forces. The speed by which the GAT tip moves is extremely slow compared to the corneal time constant and hysteresis can not be detected or measured since viscous drag depends on the speed of corneal deformation and vanishes at slow speeds.

Therefore GAT readings do not depend upon direction at applanation but NCT readings do because GAT moves so slowly corneal viscosity is zero and corneal elasticity dominates. NCT however is so rapid the cornea exhibits considerable viscous damping causing the cornea to “lag” behind the decaying air pressure (like pitting edema).

Understanding IOPin – IOPout represented corneal hysteresis took time and during this period ways to use IOPout to improve NCT readings were discovered.

<u>New IOP Measurements</u>

Grolman and Luce determined empirically that a weighted combination of IOPin and IOPout is less sensitive to corneal variations (center thickness, et al) than GAT. The ORA displays this weighted combination as IOPcc, or cornea compensated IOP and it is not significantly affected by LASIK or non-standard thickness corneas.

WARNING: IOPcc does not mean “thickness compensated”. Corneal thickness is a poor predictor of how the cornea behaves during tonometry and can not be used to correct GAT readings.

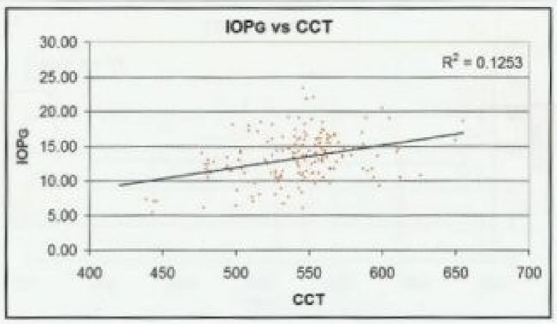
Attempts to “correct” GAT tonometry for corneal thickness, while done, are conceptually wrong. There are several “regression lines” used to make them and some yield <u>opposite</u> correction values. Worse, the straight line of a particular regression line is the least-squares fit to the many normal cornea IOPs having that thickness so there are normals with IOPs above and below the regression line. How can one know if a patient with thickness T should have GAT increased or decreased? This patient might be a normal lying above (or below) the regression line and not on the regression line (few are).

Yes, the average IOP of normal, thicker corneas is higher. But could your patient be one of the normals above or one of the normals below the straight regression line for that center thickness? (See“correction” chart.)

Several corneal specialists no lower “correct” GAT for corneal thickness and view this effort “unrewarding”. In their opinion, correcting GAT using thickness tables is futile. Doing this also adds to the misguided belief one can be very accurate in diagnosing glaucoma…that a change in IOP of 2-4 mmHg strongly affects the diagnosis. Glaucoma is multi-factorial, elusive, and difficult to diagnose and treat and subject to strong confirmation and prior diagnosis biases (how many false positive diagnoses are reversed because treatment is justified by the observation “with treatment no field loss occurred).

All that can be said for these correction curves is that <u>on average,</u> thicker, non-diseased corneas have slightly higher GAT readings but little can be inferred for an individual cornea. (A series of eyes measured by two different, calibrated GATs with skilled operators produce a mean standard error of differences of over 2mmHg which also negates the value of small “corrections” to GAT.)

Typical GAT “Correction” Methodology Is Wrong



Charts like this are used to justify correcting GAT for corneal thickness but this is faulty reasoning because each <u>individual</u> scatter dot represents a healthy eye.

The reasoning is since the IOP of thicker corneas is higher, on average, than the average IOP of thinner corneas, thicker corneal GAT readings underestimate the risk of glaucoma and vice versa.

This means a GAT reading of 20 for a thick cornea should be viewed as less a risk than a reading of 20 for a thinner cornea and should be corrected to a lesser value.

But how does a clinician know if the patient is average?

That this is misleading is shown by two patients, #1 and #2.

Patient #1 GAT=18 with CCT of 600
Patient #2 GAT=18 with CCT of 500

Patient #1 could be a normal and always had GAT of 18 with CCT 600.
Patient #2 could have had GAT of 6 for years but now has GAT of 18.

Yet common “wisdom” has it that a GAT of 18 for patient #1 indicates less risk than a GAT of 18 for patient #2. One does not treat the average but, rather, the individual patient.

Unless one knows that patient #1 has an “average” 600 CCT eye and patient #2 is an “average” 500 CCT eye there is no way to know which GAT reading should be corrected, or by how much, to better indicate risk.

Corneal factors affecting IOP include thickness, corneal rigidity, curvature, dioptric value, hydration, tissue integrity, age, and overall size. You can not accurately predict the rigidity of any material by measuring just thickness unless all materials are otherwise identical. Some normal, thin corneas are more rigid than some normal thick, corneas. Measuring thickness is futile.

Fortunately, using a weighted mix of the two applanation pressures allowed Grolman and Luce to produce an IOP measurement less dependent upon corneal variations than GAT.

Application of IOP cc to LASIK:

As noted, GAT gives false, lower IOPs after LASIK.

This can not be explained by LASIK leaving a thinner center thickness since the amount of cornea removed is miniscule. ("correction" curves ignore such minute changes.) Another aspect of corneal integrity must be affected by LASIX. An analogy might be glass cutting. A tiny scratch, forming a "fault line", reduces glass strength in one direction whereas the same reduction in thickness over the entire surface, will not.

Studies of pre- and post-LASIK patients show IOPcc is essentially uninfluenced. This provides continuity in IOP measurement. (And killed early hopes LASIK could treat glaucoma.) Later studies showed LASIK reduced corneal hysteresis so while GAT became invalid the ORA provided a clue ---LASIK reduced corneal hysteresis--- and those with low corneal hysteresis might not be good candidates to have their hysteresis reduced still more.

Other studies of IOPcc of corneas with larger variation in thicknesses than those following LASIK showed these also had little effect on IOPcc. So, we can abandon misguided attempts to "correct" GAT for corneal thickness and rely upon IOPcc

IOPg

Using another weighted mixture of first and second IOP air pressures, Grolman and Luce developed IOPg, which more closely mimics GAT measurements across a wider spectrum of patients than does NCT, including those with very low or high IOP readings and non-standard corneas.

This is not an adjustment to NCT to reach "correct" GAT readings. NCTs are already calibrated to meet ISO tonometer standards using the first applanation pressure.

IOPg is closer to what GAT would read for eyes that produce differing NCT and GAT readings and allows clinicians to better predict GAT readings for a particular eye which is useful if prior records contain only GAT readings. A clinician can now opt to record IOPg and NCT IOP to assist later examiners using GAT.

The value of IOPg is that switching between instruments will not change a diagnosis or produce "steps" in IOP logs if tonometer types are alternated.

IOPg better correlates to GAT readings and is a "nod to tradition" and continuity but not a corrected NCT reading since either NCT or GAT are sometimes less accurate.

Corneal Thickness:

ORA does measure corneal thickness. Not to "correct" GAT for corneal thickness but in recognition of NEI studies finding patients with "thin" or "thick" corneas are at higher risk for certain true or false glaucoma diagnoses. The NEI finding is a statement of how corneal thickness can be associated with misdiagnosis but not that one should correct GAT for corneal thickness. For example a "thin" cornea may be a symptom of an abnormal lamina cribrosa and cupping not due to an IOP only slightly higher than indicated by GAT. [This specific finding morphed into efforts to "correct" GAT for center thickness. No doubt aided by instrument companies who lobbied to make it billable.]

This shows the value of IOPcc as well as IOPg to the clinician in reaching treatment decisions.

Back To Hysteresis:

Hysteresis is due to corneal viscous drag opposing and delaying motion as the cornea flexes out. It takes more pressure to push the cornea into applanation than to reach applanation during rebound because viscous damping delays corneal rebound which means air pressure at IOPout has fallen below its value at IOPin. The greater the corneal viscosity the greater becomes corneal hysteresis and the more IOPout lags IOPin. That all corneas have a lower IOPout means all have some hysteresis.

The ORA uses IOPin-IOPout to measure corneal hysteresis. Normal corneas have hysteresis values clustering around 12 mmHg. Abnormal corneas exhibit lower than average hysteresis values which means they have below normal viscous damping and are less able to retain energy from the NCT air pulse. If their hysteresis loops are plotted (see next section) these have less area than those of normal corneas. This means abnormal corneas have below average hysteresis values, are more elastic than normal corneas due to reduced viscous damping and are less able to absorb energy from the air pulse.

At a normal (compared to NCT) time scale pitting edema shows tissue rebounding so slowly the indenting pressure has fallen to zero before it rebounds and hysteresis (pressure in – pressure out = pressure in - 0) is very high because the tissue is "spongy", edematous and retains the energy given it turning indentation. When hysteresis is high the tissue absorbs all the indentation energy and releases it only very slowly. A high hysteresis tissue tends to "retain" the energy given it during deformation. In the extreme case of a putty ball, it retains the energy completely and uses it to raise its temperature.

Only rapid NCT applanation and electronic tracings can reveal the viscous nature of the cornea because its rebound is so rapid, unlike pitting edema which can be watched in real time. While high or low tissue hysteresis can indicate abnormal tissue, to date only low corneal hysteresis has been correlated with corneal dysfunction.

Corneal hysteresis values are an indicator of the cornea's overall physiological status and do not correlate well, if at all, with static corneal measurements like center thickness which means hysteresis reflects overall corneal status as an organic entity and is a better indicator of corneal health than any single measurement of corneal anatomy.

Hysteresis is a proxy for the cornea's ability to absorb deformation energy and a cornea of low hysteresis absorbs less energy during rapid applanation because its physiological status at the polymer level is altered. (One possible cause is alteration in the "friction" between its laminas. Another may be related to a change in hydration.)

Abnormally Low Hysteresis Values:

Fuch's and Keratoconus:

Among the first results of measuring corneal hysteresis was the finding corneas with keratoconus and Fuch's dystrophy have below average hysteresis values. In one study 246 normals had an average hysteresis value H= 11.19 mmHg whereas those 62 with keratoconus averaged H=8.74 mmHg and 24 with Fuch's (24) averaged H=8.41 mmHg.

LASIK:

Earlier, the value of IOPcc for monitoring post-LASIK IOP was noted and for avoiding the trap of correcting GAT for center thickness. Probably more importantly, LASIK has been shown to significantly reduce corneal hysteresis; reflecting a fundamental alteration of corneal physiology and increase in corneal elasticity to viscosity ratio (E/V). Since corneal hysteresis only weakly correlates with corneal center thickness it is a better indicator of corneal biomechanical status and that LASIK lowers its value may mean corneas with low hysteresis are poor LASIK candidates and measuring H is a better screener than corneal center thickness for post-LASIK ectasia.

Glaucoma:

A significant finding of the NEI Ocular Hypertension Study was that eyes with "thin" corneas were at higher risk of glaucoma (not because GAT read low) and there is mounting evidence a "thin" cornea is not the cause but a correlate of glaucoma because it my reflect a non-normal lamina cribrosa more susceptible to damage. This may result from the fact corneal and cribrosa tissue are linked during ocular development (genesis).

Evidence for this is found in studies that low corneal hysteresis correlates with glaucoma risk and prognosis (independent of corneal thickness). Perhaps the cribrosa, with which the cornea shares tissue types, may be similarly affected, has lower than normal hysteresis and is less able to absorb the mechanical energy of IOP at every level.

There is also evidence low corneal H is found in eyes more likely to have normal-tension glaucoma, but again, not correlated with corneal thickness.

Corneal Resistance Factor:

The ORA introduces this new parameter also derived empirically from IOPout and IOPin. Termed Corneal Resistance Factor (CRF) it can be thought of as the overall "resistance" or "rigidity" of the cornea and, to some extent, the eye and surrounding tissues. CRF and hysteresis have been found to have abnormal values for eyes with Fuch's, keratoconus and POAG and normal-tension glaucoma. (While this paper concentrates on how the cornea responds to NCT pressure pulses, in fact the entire eye and orbital tissues, to some degree, respond to these pulses. Future study may show that other details of Figure A "reflect" the status of several ocular-orbit tissues much as plucking a violin string reveals information about not just its string but the instrument's shape, size, wood type and even varnish.)

Importance of Corneal Hysteresis and Resistance Factor:

This paper's goal was to provide understanding of corneal hysteresis and not review the many papers now linking abnormal H values to ocular diseases. Corneal hysteresis is important because:

1. It is a new, quantitative, measurement of a global corneal parameter previously unknown and unmeasured. Because corneal H (and CRF) reflects the complex interaction of different corneal properties as a whole, abnormal values can derive from and signal difference diseases and dysfunctions of which several have now been linked.

2. Because measurement of corneal H (and CRF) is easily done by ORA, its routine measurement may link abnormal values to additional ocular diseases and dysfunctions and provide insight into their causes. For example high values of hysteresis may signify yet undiscovered ocular dysfunctions.

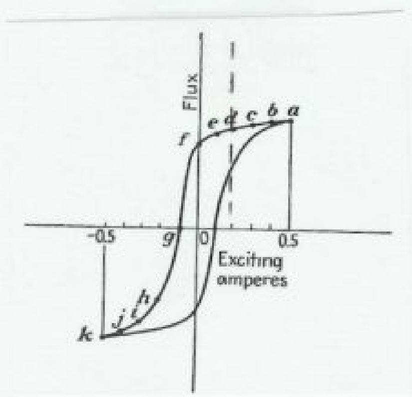
For these reasons clinicians will benefit from understanding the cause and meaning of corneal hysteresis and see the value of including its measurement on a routine basis as a new, independent risk factor indicator.

For those desiring a more detailed understanding of how hysteresis results, the following physical explanation is offered.

QUANTITATIVE EXPLANATION

In engineering, hysteresis describes how iron magnetizes when placed in a varying external magnetic field. The induced magnetic flux in the iron lags behind the external exciting field and has different values depending on whether the external field was increasing or decreasing. This creates two responses, one for an increasing and one for a decreasing external field. (See illustration)

TYPICAL HYSTERESIS LOOP



An iron bar placed within an exciting magnetic field exhibits hysteresis as the field is increased and decreased through one full cycle. The vertical axis is the magnetic flux induced in the iron at each value of the exciting field. At any particular exciting value [0.1 amperes shown by the dashed line], two possible flux values result. Less flux is induced when the exciting field increases from k up to a [right curve] and more flux is induced later when the exciting field decreases from a down to k [left curve].

Induced magnetic flux lags behind the exciting field during the cycle since it is higher [left curve] as the exciting field decreases than when the exciting field increases [right curve]. The amount of hysteresis (lag) and energy absorbed during a cycle is proportional to the area enclosed by the loop. Its shape depends upon the exact composition of the iron and prior heat treatment or magnetism and can be used to detect abnormalities.

As with viscous-elastic tissue in the arm, foot or eye, the amount of hysteresis measured, and the shape of the hysteresis loop, in iron depends on the speed (frequency) of the external force cycle.

The right segment is induced magnetism (flux) with the exciting force (amperes) increasing (k to a) and the left is induced flux with it decreasing (a to k). One cycle produces this S-shaped curve whose enclosed area represents the energy absorbed by the iron during one cycle. For an exciting force of +0.2, the induced iron flux depends on whether the force was increasing or decreasing. This difference in response for different directions is hysteresis.

In our case, the material is the cornea undergoing one cycle of flexing due to air pressure. Viscous damping causes the cornea to absorb energy and reach the applanation point at two different pressures depending upon the direction of motion. The cornea, like iron, exhibits hysteresis because it absorbs energy and applanation occurs at different air pressures depending on if the air pressure is rising or falling. Just as the “thumb” testing for pitting edema felt more resistance pushing in than when withdrawing.

The ORA uses the difference between first and the second IOPs to measure the amount of corneal hysteresis. Normal corneas have values clustered around 12 mmHg while non-normals have lower values because they are less viscous and absorb less energy.

We now see pitting edema as extreme hysteresis because the tissue's resistance fell to zero as the thumb withdrew just before the tissue began to rebound. In pitting edema, an extreme case, the "thumb" feels zero pressure as it withdraws and not just less. If the cornea had pitting edema the second IOP reading would be 0.

What follows are two mechanical models of the cornea that demonstrate the cause of corneal hysteresis, why GAT never encountered it and the NCT.

GAT CORNEAL MODEL
(Figure 1)

An elastic material like a coil spring produces a resisting force in direct proportion to the amount it is compressed or stretched. The more compressed the more it resists. The resistance does not depend on the direction or speed at which you pull or push on the spring.

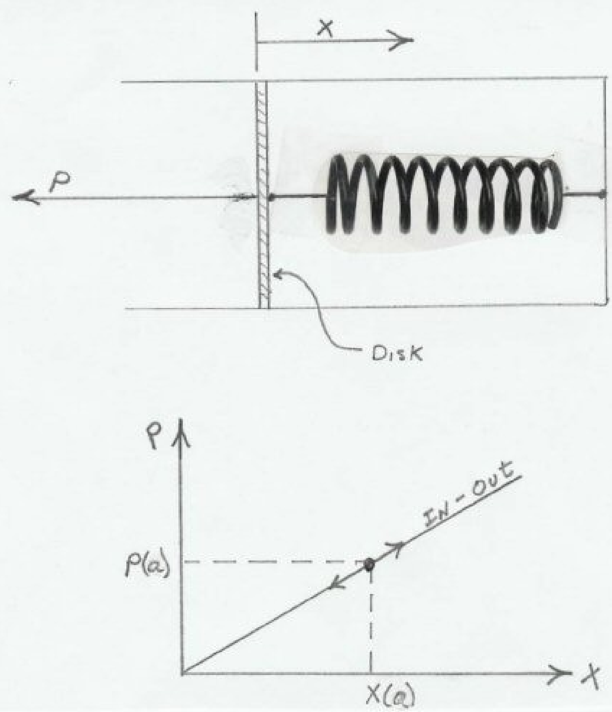
Place a coil spring in a smooth tube, attach one end at the back and push on its other, free with a "thumb" to compress it 2 inches, stop and hold still. A pressure gauge between thumb and disc reads, say 1. Compress it to 4 inches and stop. The gauge now reads 2. At 8 inches in, the spring pushes against the "thumb" with 4. Within its elastic limits a spring's resisting pressure to compression is linear and plotting compression vs. spring resistance gives a straight line of Fig. 1. Stiffer springs have steeper sloping lines while weaker ones have less steep slopes. In general, the relation between a spring's displacement (X) and the force (F) it exerts is $F = kX$ with k (slope) a constant depending upon the spring's stiffness.

It makes no difference to the spring how it got compressed in 8 inches. It resists by the same amount, 4 , whether compressed "in" from 6 to 8 inches or stretched "out" from 15 inches to 8 inches. At 8 inches it always exerts 4 units of resistance

It also makes no difference to the spring how fast it is moved to, or past, 8 inches. Whether approaching slowly, holding steady, or whizzing in or out past 8 inches the spring exerts the same resistance, 4, the instant it is at 8".

Even if forced to move rapidly by the "thumb", an elastic spring's resisting pressure will be given by Fig. 1 for each value of X and it therefore has zero hysteresis. There is no lag between "thumb'" withdrawal and spring rebound. There is no "pitting edema".

Figure 1 (GAT Model)



A circular disk is attached to the left end of an elastic spring inside a tube with the spring's right end anchored. No matter how rapidly the disk is pushed in or out by an external agent it resists with a pressure P that depends only on position X. The disc's resisting force P(a) after being moved to X(a) is the same irrespective of its direction, or speed, at X(a). It pushes out with the same pressure at X(a) whether held there steady, is being compressed in and passes X(a) or rebounding out and passing X(A). All elastic materials behave in this manner.

This models the elastic cornea assumed by Goldmann. If X(a) corresponds to applanation, the cornea pushes out with the same pressure whether it reached applanation while moving in or out or was held there steady. This assumption of elasticity, while incorrect, works with GAT because it operates so slowly there is no viscous damping by the cornea.

If one thinks of the corneal as small adjoining disks attached to tiny springs the result is the elastic cornea assumed by Goldmann. Because the GAT tip moves very slowly with the cornea at rest at applanation, cornea viscosity has no effect and GAT encounters only an elastic cornea. (Like checking for pitting edema by moving the indenting finger so slowly the indent is never visible.)

REALISTIC CORNEAL MODEL
(Figure 2)

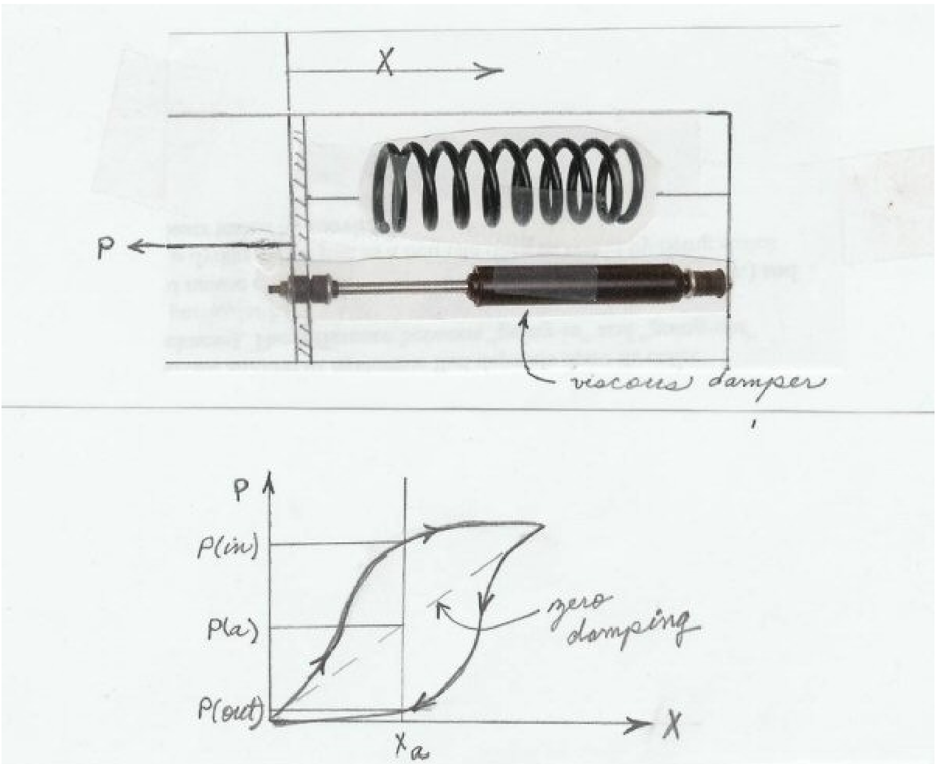


Figure 2 (NCT Model)

A viscous damper is added whose drag increases with speed. This model is viscous-elastic but when an external pressure moves it slowly, drag is low and disc resistance P still depends only on position X (zero-damping line) as in elastic model 1.

But if external pressure moves the disc rapidly in, viscous drag develops and adds to spring resistance causing higher disc resistance P at each position (left curve). P(in) at X(a) becomes higher than in the elastic model P(a). Near the start, where the disc moves slowly and drag is still small, P(in) follows the zero damping line until drag increases.

Once compressed, if the external pressure rapidly decreases, viscous drag again builds as the disc speeds up and opposes the spring's efforts to push the disc out. Disc resistance P against the external pressure is less (right path) at each position than when the spring acted alone. At X(a) the disc exerts less pressure, P(out), compared to P(in) and P(a).

P(in) – P(out), results solely from viscous damping and P(out) must always be lower. The elastic-viscous exhibits hysteresis when moved rapidly. The area of the hysteresis loop indicates how much energy was absorbed by damper viscosity. Unlike foot or arm tissue, low corneal hysteresis is associated with dysfunction.

In this model corneal tissue is viscous and not just elastic. The elastic coil spring slides in and out but now with a viscous shock absorber (damper) added. This combination resembles the pneumatic door closers we encounter everyday that contain a spring to close the door and a damper to keep it from slamming. It reacts like a cornea or other viscous-elastic tissue when made to move rapidly. On cars the combo is called a “strut” (see illustration) or, in older cars, a spring and shock absorber.

The viscous damper is not linear like the spring and resists more strongly the faster you attempt to move it. Push in on the disc and both spring and viscous drag resist but, pull out (reduce your push) and viscous drag reduces the pressure by which the spring pushes on your thumb. The net force of resistance now depends on speed and the direction of motion.

Experiment with a pneumatic door closer. If you slowly move the door you feel only the elastic spring resisting. But, move the door rapidly and its resistance quickly increases from viscous drag. Open the door and remove your hand and it slowly closes much as the pitting edema indentation slowly rebounds out.

You will find it takes more force to quickly open the door since both the spring and shock absorber oppose you than to quickly close the door since the shock absorber resists but the spring is helping.

Figure 2 illustrates how this viscous-elastic combination resists being moved during one cycle in-and-out. The curve shows hysteresis. It is not a straight line and the faster you attempt to make the device move through one cycle the more the curve bulges away from the straight line as viscous drag increases and more energy is absorbed by the damper.

The dashed straight line shows if we move very slowly only the spring resists and we have Fig. 1 again. This is why the slow GAT encounters no viscous drag and its measurements are the same whether its tip is moving in, out or not at all at applanation.

Typical Strut

An elastic spring and a viscous damper combined as one unit is found in many common devices such as automobiles (termed struts) and storm door closers. In this strut the elasticity is clearly produced by the coil spring and the viscous damping by the fluid filled shock absorber. In tissue the demarcation is not so clear since the elastic and viscous properties result from a "blend" of various tissue constituents acting together.

The viscous-elastic nature of the cornea and hence its hysteresis depends upon the parameters of thickness, hydration, curvature, diameter and tissue integrity interacting with each other. Since these each react differently to slow vs. fast external forces, the cornea can act primarily as elastic tissue having little hysteresis during Goldmann tonometry but as viscous tissue having high hysteresis during air pulse tonometry.

An example of the dependence of response to the rapidity of an applied force is demonstrated by driving a car down a washboard road at different speeds. The struts react differently as speed increases and, while the car faithfully follows the bumps up and down at slow speed the struts begin to "average" the bumps out (lag behind) at higher speeds and the car begins to ride smoother at higher speeds.

Let's imagine going through one cycle, starting with X=0. We rapidly apply a force sufficient to reach full compression when only the spring was present and measure the disc's resistance P at each position X. Before picking up much speed there is little viscous drag and resistance P follows the zero-drag line of Model 1 because drag is low and only the elastic spring resists. But, as the disc gains speed, drag increases rapidly and resistance P becomes greater with greater X than with just the spring acting and the resistance curve P bows to the left. (Looked at another way, the disc resistance reaches it values after less movement in than before due to drag.)

As we approach the end point and resistance P becomes close to our external force the disc moves slowly again, drag decreases and the curve flattens out.

Now rapidly decrease the outside force. It there is no drag, the disc's resistance to movement at every position would follow the zero-drag line once again. And, when the disc begins, moving slowly, there is little drag and P follows that line. But, as speed picks up drag increases again and acts opposite to the spring's force. On the return trip the disc exerts less force of resistance because viscous drag opposes spring force. The net resistance to outward motion is less than that of the spring alone and the curve bows to the right. At a midpoint, say X(a), the disc resisted movement going outward less than it resisted movement going inward. This is hysteresis again.

This means the force needed to move the disk past a given point is greater going-in than going-out. This is shown by P(1) being greater than P(2) at X(a).

P(1) - P(2) represents corneal hysteresis and depends on the amount of corneal viscosity.

GAT operates too slowly to encounter hysteresis. Only a rapid applanation can reveal elastic-viscous properties. Digital palpation operates quickly enough to show hysteresis in foot and arm tissues with long time constants (pitting edema).

After study, Grolman and Luce saw corneal viscosity as the cause for applanation to always occur at a lower air pressure while the cornea was rebounding and that Figure A enabled corneal hysteresis to be measured.

Hysteresis is an indicator of the cornea's overall physiology and does not correlate strongly with a single static parameter like thickness, meaning it reflects overall corneal status as an organic entity. Just as pitting edema does not stem solely from tissue thickness but on overall tissue physiology.

Conclusions:

One need not build a watch, discover how to smelt iron, or understand television and cell phones to use them. (Nor wade through this lengthy tome.)

But, clinicians should understand their instruments because it improves care, distinguishes them from technicians and gives greater appreciation of their instruments in front of which they like to pose. There are too many clinicians who blindly accept the readings of their "machines" without understanding how they can go "bad" or be misleading. One of the ironies of life is that inventors of equipment may end up earning less than those who use them.

I hope relating how Grolman and Luce discovered the "ghosts" in the NCT appeals to those wanting to more fully understand tonometry and corneal hysteresis. Grolman, in fact, discovered the NCT process by accident which leads us back to ringing bells. His initial idea for measuring IOP (without anesthetic) was to use sound pressure waves to excite the eye into vibrating because pressurized structures have a frequency at which they most easily "ring" (resonant frequency) which rises with higher pressure. So, measure resonant frequency of an eye and one can determine IOP.

But, high-speed photographs of sound waves striking rabbit eyes did not show vibrations. Failure. Then, one day, studying the film, Grolman noticed the sound waves did slightly flatten the rabbit corneas although they did not vibrate. Sound waves are a series of air pressure pulses and some years later the NCT appeared in 1971 and optometrists had an accurate means of measuring IOP before DPAs became available.

Unknown to Grolman, there was still information within the NCT waiting to be unlocked some 40 years later when he and Luce revealed the NCT "Ghosts" for what they were.

Open VA Optometrist Positions

			position will be eligible to apply for an award up to the maximum limitation under the provisions of the Education Debt Reduction Program. The incumbent must be a citizen of the United States; must be proficient in spoken and written English as required by 38 U.S.C. 7402(d) and 7405(f); and must hold a license valid in any state.
Battle Creek, MI VAMC	Christi Feighner, HRMS	(269) 223-5241	Will perform low vision evaluations, prescribe appropriate low vision devices, and work effectively with multidisciplinary team in prescribing a training course for the Clinic (low vision therapist, orientation and mobility instructor) to provide visual impairment rehabilitation training.
Saginaw, MI VAMC	Edward Lesko, HR	(989) 497-2500	The incumbent will provide low vision examinations, prescribe appropriate low vision devices, work effectively with the multidisciplinary low vision to provide optimal visual impairment rehabilitation service in an advanced ambulatory low vision program. In addition, while not in low vision, provide high level full-scope primary eye care including glaucoma, diabetic and urgent care management.
VAMC, Biloxi, MS		(228) 523-4607	The incumbent functions as an integral part of the eye care team and the Surgical Service at the VA Gulf Coast VA Care System. He/she is responsible for examining the human eye and ascertaining defects of the human visual system with ophthalmology available for consultation.

Reviewed/Updated Date: June 27, 2008

Web Links

Associations, Institutes, Organizations, Societies

All About Vision
Alliance for Aging Research
American Academy of Ophthalmology
American Academy of Optometry
American Academy of Pediatrics
American Optometric Association
American Optometric Foundation
Association of Regulatory Boards of Optometry (ARBO)
Council for Refractive Surgery Quality Assurance
Eye Advisory
Eye Surgery Education Council
Glaucoma Research Foundation
Healthy Vision 2010
International Glaucoma Association
NASA Vision Group
National Eye Research Foundation
National Keratoconus Foundation
National Optometric Association
NBEO
Optometric Extension Program
Optometric Refractive Surgery Society
Optometrists.org
ORMS
Parents Active for Vision Education
RGP Institute
Schepens Eye Research Institute
Vision Council of America

World Council of Optometry

Conventions and Meetings

Great Western Council of Optometry
Heart of America Contact Lens Society
Midwest Vision Congress & Expo
Southern Council of Optometrists
Vision Expo East
Vision Expo West

Government Health

Armed Forces Optometric Society
Centers for Disease Control and Prevention
ClinicalTrials.gov
Federal Registry
NAVAO

Journals

American Journal of Ophthalmology
American Society of Cataract and Refractive Surgery
Archives of Ophthalmology
British Journal of Ophthalmology
Digital Journal of Ophthalmology
Eyeworld
Ocular Surgery News
Ophthalmology Times
Ophthalmology
Primary Care Optometry News
Primary Eye Care News
Review of Ophthalmology
Review of Optometry

Low Vision and Vision Therapy

American Foundation for the Blind
Canadian National Institute for the Blind
Eschenbach Optical
Foundation for Fighting Blindness
Lighthouse International
Low Vision Gateway
NORA - Neuro-Optometric Rehabilitation Association
Ocutech
Prevent Blindness America
State License Renewal Requirements
Vision and Computers

Ocular Disease

AMD Alliance International
American Macular Degeneration Foundation
Lutein Information
Macular Degeneration Foundation
Macular Degeneration International
Macular Degeneration Network
Macular Degeneration Partnership
Macular Disease Society
MAXIVISION
Center for Keratoconus
Chua Eye Page
Collaborative Longitudinal Evaluation of Keratoconus Study (CLEK)
EyeCancer Network
Ophthoguide
Ophtholinx
Trials Summary

Systemic Disease

American Diabetes Association
AskPhysicians.com
National Headache Foundation
Sjögrens Syndrome Foundation

Ophthalmic Equipment and Medications

Alcon Laboratories
Allergan
BOTOX®
Carl Zeiss Meditec
Hilco
Lombart Instruments
Medtronic Solan
Merck
Novartis Ophthalmics
Novartis
Reichert Ophthalmic Instruments
Reliance Medical Products
Topcon
Wilson Ophthalmic

Optometry Schools

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Michigan College of Optometry at Ferris State University
New England College of Optometry
Northeastern State University
NSU College of Optometry
Ohio State University
Pacific University
Pennsylvania College of Optometry
Southern California College of Optometry
Southern College of Optometry
SUNY State College of Optometry
UAB School of Optometry
University of California - Berkeley
University of Houston
University of Missouri - St. Louis

State Optometry Associations

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Virginia
Washington
West Virginia
Wisconsin
Wyoming