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Corneal Sensitivity Measurement in Clinical Ophthalmology: The Science of Aesthesiometry

Kuldeep Raizada[1], Deepa Rani Diddi[2], Sayamprove Baruah[3]

[1]PhD, Akriti Ophthalmic Pvt. Ltd., Medical Devices Park, Patancheru, Hyderabad – 502319, Telangana, India
Email: *info[at]akritiophthalmic.com*

[2]PhD, Akriti Ophthalmic Pvt. Ltd., Medical Devices Park, Patancheru, Hyderabad – 502319, Telangana, India

[3]M.E., Akriti Ophthalmic Pvt. Ltd., Medical Devices Park, Patancheru, Hyderabad – 502319, Telangana, India

Abstract: ***Background: Corneal sensation, mediated by the trigeminal nerve's ophthalmic branch, is fundamental to ocular surface homeostasis. Reduced corneal sensitivity- termed hypoaesthesia or anaesthesia- is both a consequence and an accelerator of ocular surface disease. Accurate, reproducible measurement of corneal sensation is therefore essential for the diagnosis, staging, and therapeutic monitoring of a wide range of conditions, most critically neurotrophic keratopathy. Objective: To review the clinical science of corneal aesthesiometry, the history and operating principles of the Cochet-Bonnet aesthesiometer, its evidence base across key ocular surface and systemic diseases, and to present the specifications of the Smart Cochet-Bonnet Aesthesiometer by Akriti Ophthalmic Pvt. Ltd. Product: The Smart Cochet-Bonnet Aesthesiometer (Akriti Ophthalmic, Hyderabad) employs a nylon monofilament of 0.12 mm diameter with a length range of 5-60 mm, delivering calibrated pressures of 11–200 mg/0.0113 mm². The instrument features a retractable tip for filament protection, a clearly readable scale, and an ergonomic design optimised for rapid clinical and slit lamp examination. Conclusion: The Cochet-Bonnet aesthesiometer remains the most widely validated and clinically accessible tool for corneal sensitivity testing. The Smart Cochet-Bonnet Aesthesiometer from Akriti Ophthalmic delivers precision, durability, and ease of use, establishing it as an indispensable instrument for any corneal, anterior segment, or comprehensive ophthalmic practice.***

Keywords: corneal sensitivity, aesthesiometer, Cochet-Bonnet, neurotrophic keratopathy, corneal innervation, hypoaesthesia, dry eye disease, diabetes, herpes simplex keratitis, ocular surface disease, Akriti Ophthalmic

1. Introduction: Why Corneal Sensation Matters

The cornea is among the most densely innervated tissues in the human body, with a nerve fibre density approximately 300–600 times that of skin. This extraordinary innervation serves three critical functions: first, the afferent sensory arc of the blink reflex, which protects the ocular surface from trauma; second, neurotrophic support of corneal epithelial integrity and wound healing; and third, regulation of tear secretion and composition through interactions with lacrimal gland efferents. When this innervation is impaired- by viral infection, diabetes, surgical trauma, topical anaesthetic abuse, or neurological disease- the consequences for the ocular surface are severe, progressive, and potentially blinding.

Neurotrophic keratopathy (NK), the prototypical manifestation of corneal sensory nerve loss, is characterised by a cascade of epithelial breakdown, stromal ulceration, corneal melt, and perforation, driven not by infectious agents but by the withdrawal of trophic and protective neural inputs. NK affects an estimated 1.6 per 10,000 population and is classified into three progressive stages of severity (Mackie classification). Critically, its diagnosis hinges on the demonstration of reduced or absent corneal sensation- a finding that cannot be reliably established by slit lamp biomicroscopy alone.

Beyond NK, corneal sensitivity measurement has proven clinical value in monitoring disease activity in herpes simplex keratitis, assessing diabetic peripheral neuropathy through the corneal window, tracking post-refractive surgery nerve regeneration, and evaluating the severity of dry eye disease. The instrument at the centre of this clinical paradigm is the Cochet-Bonnet aesthesiometer- a device whose fundamental design, conceived in the 1950s, remains the gold standard for corneal sensitivity testing in routine clinical practice worldwide.

2. Anatomy and Physiology of Corneal Innervation

2.1 Neural Architecture of the Cornea

Corneal innervation is derived from the ophthalmic (V1) branch of the trigeminal nerve (cranial nerve V), specifically the nasociliary branch. Long posterior ciliary nerves penetrate the sclera near the optic nerve and travel anteriorly in the suprachoroidal space before entering the peripheral corneal stroma, where they lose their myelin sheath and branch extensively. This dendritic plexus bifurcates in the anterior stroma to form the subepithelial nerve plexus, from which terminal branches penetrate Bowman's layer to form the subbasal nerve plexus — a horizontal, whorl-like network visible on in vivo confocal microscopy (IVCM). Unmyelinated intraepithelial nerve endings terminate between epithelial cells throughout all corneal layers and represent the actual sensory receptors.

The human cornea contains approximately 7,000 nerve terminals per mm². Corneal nerves are predominantly A-delta and C fibres, transmitting mechanical, thermal, and chemical stimuli. A-delta fibres (thinly myelinated, medium conduction velocity) predominantly mediate sharp, localised pain responses to mechanical stimuli, while C fibres (unmyelinated, slow conduction velocity) mediate the duller,

burning quality of pain and are responsible for the ongoing protective signalling that supports epithelial homeostasis.

2.2 Neurotrophic Role of Corneal Nerves

Beyond sensation, corneal nerves exert critical trophic effects on the ocular surface. Neuropeptides released from nerve terminals- including substance P (SP), calcitonin gene-related peptide (CGRP), neuropeptide Y, and vasoactive intestinal peptide- act on epithelial cells to promote proliferation, migration, and adhesion. The corneal epithelial basement membrane is maintained in part by neural inputs, and loss of innervation leads to impaired hemidesmosome formation, reduced epithelial cell proliferation, and breakdown of the epithelial barrier. This explains why corneal anaesthesia invariably leads to chronic epithelial defects irrespective of other ocular surface factors.

3. History and Evolution of Corneal Aesthesiometry

The measurement of corneal sensation has a history spanning over 130 years. The first purpose-built aesthesiometer was described by von Frey in 1894, employing horse hairs of variable lengths to deliver calibrated tactile stimuli to the corneal surface. This principle- varying the length of a filament of known diameter to modulate applied pressure- remains the foundation of the Cochet-Bonnet instrument today.

In 1932, Francheschetti refined von Frey's approach, and in 1956 Boberg-Ans described a device using a single nylon thread of constant diameter but variable length, recognising that nylon's physical properties made it a more consistent and reproducible stimulus material than biological fibres. Henri Cochet and Roger Bonnet improved upon the Boberg-Ans design and developed the instrument that bears their names, which became the first commercially standardised corneal aesthesiometer. The Cochet-Bonnet aesthesiometer was widely adopted through the 1960s and 1970s and remains the reference standard against which all subsequent devices are validated.

Subsequent decades saw the development of non-contact alternatives- the Belmonte gas aesthesiometer (NCCA), which uses a controlled jet of air to deliver mechanical, thermal, and chemical stimuli; and the Swiss liquid-jet aesthesiometer- designed to address the limitations of direct corneal contact. More recently, the Corneal Esthesiometer Brill (CEB) was introduced as a further refinement. However, the accessibility, simplicity, and robustness of direct clinical validation data mean that the Cochet-Bonnet aesthesiometer continues to dominate clinical practice, particularly in centres where non-contact devices are not available.

4. The Cochet-Bonnet Aesthesiometer: Operating Principle and Physics

4.1 Physical Principle

The Cochet-Bonnet aesthesiometer operates on the mechanical principle of axial pressure transmission through a nylon monofilament of fixed diameter but variable length. The fundamental physics is described by Euler's column buckling formula: for a given material and cross-sectional diameter, the force required to produce a defined bending (buckling) stress decreases as the free length of the filament increases. Conversely, shortening the filament increases the bending threshold, delivering greater pressure to the corneal surface at the moment of buckling.

Key Physics: The filament transmits pressure axially until the bending stress threshold is reached and the filament buckles. The corneal surface receives pressure equal to the buckling force. Longer filament = lower pressure; shorter filament = higher pressure.

4.2 The Smart Cochet-Bonnet Aesthesiometer- Technical Specifications

The Smart Cochet-Bonnet Aesthesiometer from Akriti Ophthalmic Pvt. Ltd. is engineered for rapid, reliable corneal sensitivity examinations with the following technical characteristics:

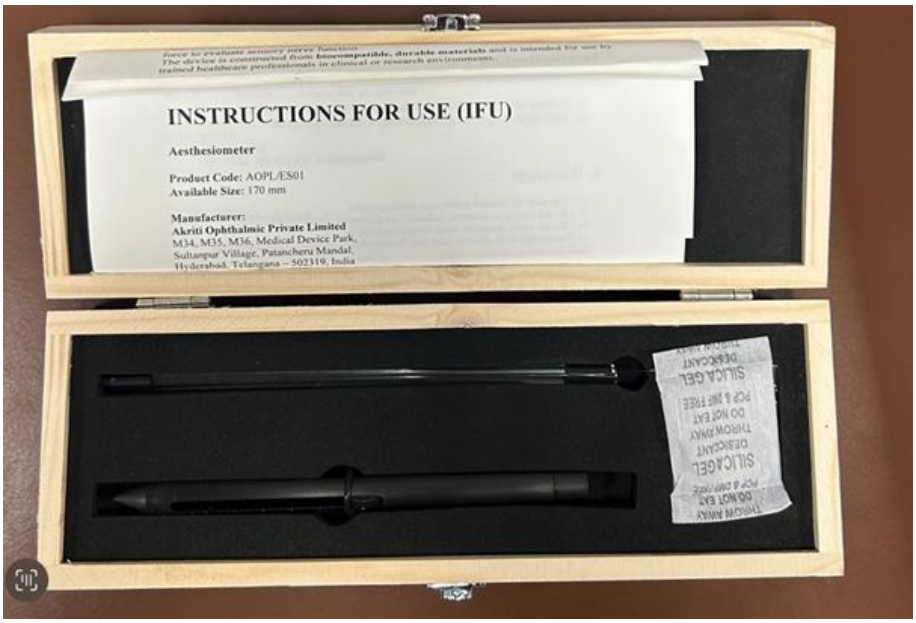

Image 1: Showing the full set of Aesthesiometer with Extra Nylon Filament and Lead Weights

Table 1: Technical specifications of the Smart Cochet-Bonnet Aesthesiometer (Akriti Ophthalmic Pvt. Ltd.). Source: akriti.co.in/products/cochet-bonnet-aesthesiometer

Technical Parameter	Specification / Detail
Monofilament Diameter	0.12 mm nylon monofilament
Length Range	5 mm to 60 mm (continuously adjustable)
Pressure Range	11 mg/0.0113 mm² (at 60 mm) to 200 mg/0.0113 mm² (at 5 mm)
Stimulus Type	Direct mechanical (tactile) stimulus — axial pressure at buckling threshold
Scale	Graduated scale readable during examination; directly indicates filament length
Control Mechanism	Forefinger-operated slider for precise, stepwise length adjustment
Tip Protection	Fully retractable monofilament tip into instrument body when not in use
Measurement Units	Millimetres (mm) of filament length; convertible to mg pressure via calibration chart
Sensitivity Range	0 mm (absent sensation) to 60 mm (full/normal sensation)
Clinical Threshold for NK	CS ≤ 40 mm is consistent with NK; CS < 50 mm warrants investigation
Application	Slit lamp-assisted or hand-held direct examination
Manufacturer	Akriti Ophthalmic Pvt. Ltd., Hyderabad, India
Price	Rs. 1,15,000 (available at www.akriti.co.in)

4.3 Pressure-Length Relationship

The inverse relationship between filament length and applied pressure follows a non-linear function. At maximum length (60 mm), the filament delivers the minimum pressure of 11 mg/0.0113 mm², representing the lightest detectable stimulus. As the length is progressively reduced, pressure increases, reaching 200 mg/0.0113 mm² at the minimum length of 5 mm, representing the maximum mechanical stimulus. This graduated range allows the clinician to map the complete spectrum from normal sensitivity through graded hypoaesthesia to complete anaesthesia using a single instrument.

Table 2: Pressure-length calibration guide and clinical interpretation for the Cochet-Bonnet aesthesiometer. NK = Neurotrophic Keratopathy.

Filament Length (mm)	Approx. Pressure	Clinical Interpretation	NK Staging Correlate
60 mm	11 mg/0.0113 mm²	Normal / Full sensitivity	Normal
50 mm	~30 mg/0.0113 mm²	Low-normal; early subtle loss	NK Stage I borderline
40 mm	~60 mg/0.0113 mm²	Mild hypoaesthesia	NK Stage I
30 mm	~95 mg/0.0113 mm²	Moderate hypoaesthesia	NK Stage II
20 mm	~140 mg/0.0113 mm²	Marked hypoaesthesia	NK Stage II–III
5 mm	200 mg/0.0113 mm²	Severely reduced / anaesthesia	NK Stage III
0 mm	No sensation	Complete anaesthesia	NK Stage III (advanced)

5. Examination Technique

5.1 Step-by-Step Protocol

Important: No topical anaesthetic should be instilled prior to aesthesiometry. All other slit lamp examinations should be completed first, as fluorescein dye and lubricating drops may alter sensitivity readings.

1) **Step 1.** Patient positioning and preparation: Seat the patient comfortably at the slit lamp. Ensure no topical anaesthetic or lubricant has been instilled in the preceding 30 minutes. Instruct the patient to fixate straight ahead and to indicate verbally or by blinking when they feel the filament touch their eye.
2) **Step 2.** Initial filament length selection: Begin with the maximum filament length of 60 mm to deliver the lowest possible pressure, testing whether the patient has intact normal sensation before applying any higher-pressure stimulus.
3) **Step 3.** Approach and contact: Under low slit lamp magnification (or hand-held if slit lamp guidance is not used), advance the aesthesiometer toward the cornea perpendicularly, ensuring the monofilament contacts the corneal surface at a 90° angle. Oblique contact introduces friction artefact and underestimates true buckling pressure.
4) **Step 4.** Stimulus delivery: Allow the filament to make gentle contact and observe for buckling — the filament will bow slightly at the point of sufficient axial pressure. The patient should report a sensation of touch. Record the filament length at which a consistent response is elicited.
5) **Step 5.** Threshold determination: If no response is obtained at 60 mm, progressively shorten the filament in decrements (e.g., 60 → 50 → 40 → 30 → 20 → 10 → 5 mm) until the patient first responds. This length is recorded as the corneal sensitivity threshold.
6) **Step 6.** Corneal mapping: For full clinical assessment, measure sensitivity at five standardised corneal locations: central cornea, and four paracentral positions (superior, inferior, nasal, temporal) at approximately 2–3 mm from the corneal vertex. This topographic mapping identifies regional nerve loss patterns.
7) **Step 7.** Contralateral comparison: Always measure the fellow eye under identical conditions for interocular comparison, which is essential for detecting asymmetric hypoaesthesia.
8) **Step 8.** Retract and clean: After examination, fully retract the monofilament into the instrument body using the slider. Wipe the filament tip with 70% isopropyl alcohol and allow to air dry before the next patient.

5.2 Recording and Reporting

Results should be recorded as the filament length in millimetres at which a repeatable response is obtained. A clinical record might read: "Corneal sensitivity: R 35 mm (central), 50 mm (nasal/temporal), 40 mm (superior/inferior); L 60 mm all zones." Where slit lamp mapping is performed, a schematic corneal diagram with zonal sensitivity values provides the most informative record for serial monitoring.

5.3 Normative Values and Clinical Thresholds

Table 3: Clinical interpretation guide for Cochet-Bonnet aesthesiometer readings. NK = Neurotrophic Keratitis (Mackie Classification)

Sensitivity Finding	Clinical Significance
60 mm (full length)	Normal corneal sensitivity
≥50 mm	Low-normal; may warrant monitoring in at-risk patients (diabetics, post-herpetic)
≤40 mm	Consistent with Neurotrophic Keratitis Stage I - further evaluation mandatory
20–40 mm	Moderate hypoaesthesia- NK Stage II; risk of stromal ulceration
5–20 mm	Severe hypoaesthesia- NK Stage II–III; high risk of corneal decompensation
0 mm / no response at 5 mm	Complete corneal anaesthesia- NK Stage III; maximum risk

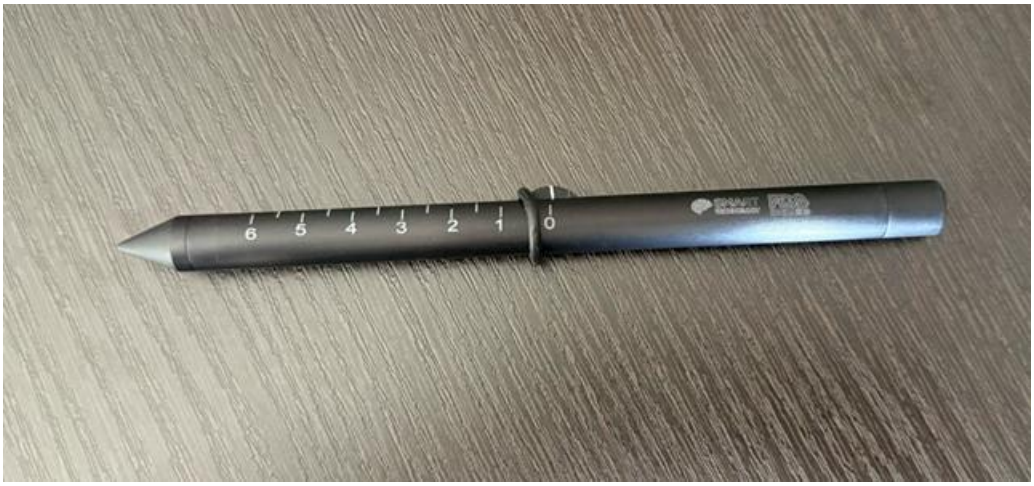

Image 2: Showing the Smart Aesthesiometer with O ring over the device on 0 Position

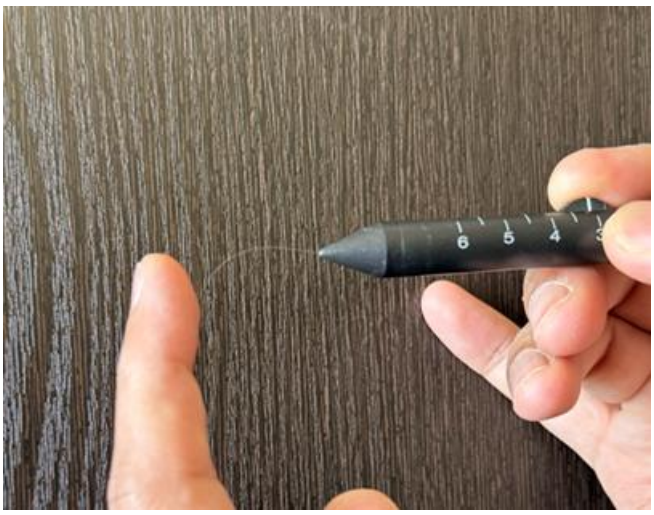

Image 3: Showing the Smart Aesthesiometer with 30 mm Extended filament and showing the force

Image 4: Showing the Smart Aesthesiometer with 60 mm Extended filament and showing the force

6. Clinical Applications of Corneal Aesthesiometry

6.1 Neurotrophic Keratopathy (NK)

Neurotrophic keratopathy represents the primary indication for formal corneal aesthesiometry. Defined as a degenerative corneal condition caused by impairment of trigeminal innervation, NK is staged by the Mackie classification into three progressive grades, with corneal sensitivity being the

defining diagnostic criterion at each stage. The Cochet-Bonnet aesthesiometer provides the sensitivity measurement that determines not only diagnosis but treatment eligibility-cenegermin (Oxervate®), the first approved pharmacological treatment for NK, requires documented reduced sensitivity as an inclusion criterion.

Clinical research has established that all patients with NK Stage I show a corneal sensitivity of ≤40 mm, and no healthy controls demonstrate values below 50 mm. This gives the Cochet-Bonnet a high discriminatory value at the clinically critical threshold of 40–50 mm for detecting early NK.

Common causes of neurotrophic keratopathy requiring aesthesiometric evaluation:

- Herpetic keratitis (herpes simplex virus, herpes zoster ophthalmicus)- the most common cause globally; HSV directly infects corneal nerve fibres, causing progressive denervation with recurrent episodes
- Diabetic peripheral neuropathy- corneal sensitivity reduction correlates with systemic neuropathy severity and HbA1c control
- Trigeminal nerve injury- surgical (neurosurgery, acoustic neuroma removal), traumatic, or iatrogenic (radiofrequency ablation, Gamma Knife radiosurgery)
- Topical anaesthetic abuse- direct toxic nerve damage from prolonged self-medication
- Chemical and thermal corneal burns- acute thermal and alkali injuries destroy nerve terminals
- Tumours of the trigeminal nerve or cavernous sinus-meningioma, schwannoma, nasopharyngeal carcinoma
- Post-refractive surgery (LASIK, PRK, SMILE)- transient or prolonged denervation from stromal nerve transection
- Riley-Day syndrome (familial dysautonomia) and other congenital trigeminal sensory neuropathies

6.2 Herpes Simplex Keratitis (HSK)

Herpes simplex virus type 1 (HSV-1) keratitis is the most frequent infectious cause of corneal blindness in the developed world. Beyond the direct epithelial and stromal inflammation it causes, HSV has a particular tropism for corneal and trigeminal nerve fibres. Corneal sensitivity is reduced in all forms of HSK, with stromal and disciform keratitis producing the greatest nerve damage. Serial Cochet-Bonnet measurements serve two purposes in HSK management: first, establishing the degree of nerve involvement at diagnosis; and second, monitoring nerve recovery following antiviral therapy or as a biomarker of subclinical recurrence. Recovery of corneal sensitivity toward normal values correlates with clinical quiescence and predicts epithelial stability.

6.3 Diabetic Peripheral Neuropathy

The cornea has attracted growing interest as a non-invasive window into systemic diabetic neuropathy. Corneal nerve density (measured by IVCM) and corneal sensitivity (measured by aesthesiometry) both decline in parallel with sural nerve conduction velocity and sudomotor function — established systemic neuropathy markers. Several landmark studies have demonstrated that corneal sensitivity measured by the Cochet-Bonnet aesthesiometer correlates significantly with the duration of diabetes, HbA1c levels, and the severity of diabetic peripheral neuropathy by nerve conduction studies. This positions corneal aesthesiometry as a potential non-invasive screening tool for systemic diabetic neuropathy in ophthalmology clinics, where patients with diabetes are routinely evaluated.

6.4 Dry Eye Disease (DED)

The relationship between corneal sensitivity and dry eye disease is bidirectional and complex. Severe aqueous-deficient dry eye (particularly Sjögren's syndrome-associated) is associated with reduced corneal sensitivity, as chronic epithelial stress and inflammation damage the subbasal nerve plexus. Conversely, corneal anaesthesia impairs the neurotrophic support necessary for goblet cell maintenance and mucin production, worsening tear film instability. Aesthesiometry in DED helps distinguish neuropathic from inflammatory aetiologies, guide treatment escalation decisions, and monitor the response to neurotrophic agents such as cenegermin and AM transplantation.

6.5 Post-Refractive Surgery Monitoring

LASIK creates transient corneal denervation by severing stromal nerve trunks during flap creation, resulting in a predictable reduction in corneal sensitivity that typically reaches its nadir at 1 month post-operatively and recovers substantially by 6–12 months. In a subset of patients with delayed or incomplete recovery, reduced corneal sensitivity contributes to persistent dry eye symptoms, recurrent erosions, and impaired epithelial healing. Serial Cochet-Bonnet measurements at 1, 3, 6, and 12 months provide objective documentation of nerve regeneration and identify patients who may benefit from neurotrophic therapies or intensified lubrication.

6.6 Glaucoma and Contact Lens Use

Long-term topical glaucoma medication, particularly with preservative benzalkonium chloride (BAK), has been associated with progressive reduction in corneal sensitivity attributable to both direct neurotoxicity and ocular surface inflammation. Switching from preserved to preservative-free formulations has been shown to partially restore sensitivity. Aesthesiometry provides an objective endpoint for monitoring this toxic effect. Extended contact lens wear similarly reduces corneal sensitivity through mechanical compression of the subepithelial plexus; sensitivity monitoring is relevant in patients with corneal complications attributable to contact lens-induced hypoxia.

6.7 Fuchs' Endothelial Dystrophy and Keratoconus

Both Fuchs' endothelial dystrophy and keratoconus are associated with reduced corneal sensitivity, with reductions correlating with disease severity. In Fuchs', the chronic endothelial failure and oedema produce secondary stromal and nerve changes; in keratoconus, the mechanical and structural deformation of the corneal architecture distorts and damages the nerve plexus, particularly in advanced disease. Post-Descemet membrane endothelial keratoplasty (DMEK)

and post-corneal collagen crosslinking (CXL) sensitivity recovery can be documented serially with aesthesiometry.

7. Comparison with Other Aesthesiometers

Table 4: Comparative overview of available corneal aesthesiometers. NCCA = Non-Contact Corneal Aesthesiometer; CBA = Cochet-Bonnet Aesthesiometer.

Device	Stimulus	Advantages	Limitations	Availability	Cost
Cochet-Bonnet (incl. Akriti Smart CBA)	Mechanical (nylon filament)	Gold standard; validated; simple; portable; slit-lamp compatible	Contact only; no thermal/chemical stimuli; learning curve for perpendicular contact	Widely available globally	Low-moderate)
Belmonte NCCA	Mechanical, thermal, chemical (air jet)	Non-contact; stimulates A-delta and C fibres separately; sensitive to low intensities	Expensive; lab-based; not widely available clinically; complex calibration	Research centres only	High
Swiss Liquid-Jet	Mechanical (liquid jet)	Non-contact; precise pressure control; repeatable	Limited clinical adoption; requires liquid reservoir system	Very limited	High
Corneal Esthesiometer Brill (CEB)	Mechanical (nylon)	Portable; disposable monofilament; standardised	Newer device; less validated evidence base vs CB	Emerging	Moderate
KeraSense® (Dompe)	Mechanical	Disposable; sterile; single-use; validated against CB (r>0.001 agreement)	Less experience; primarily for NK screening	Growing	Low-moderate

The Cochet-Bonnet aesthesiometer remains the most extensively validated instrument for corneal sensitivity testing with decades of peer-reviewed evidence establishing its normative values, disease-specific thresholds, and inter-rater reproducibility. While non-contact alternatives offer theoretical advantages in measuring discrete nerve fibre subtypes, their complexity, cost, and limited clinical availability restrict their use to specialised research settings. For the comprehensive ophthalmologist, corneal specialist, or diabetologist incorporating ocular endpoints, the Smart Cochet-Bonnet Aesthesiometer provides the optimal combination of clinical validity, practical usability, and cost-effectiveness.

8. Integration with Other Ocular Surface Diagnostics

Corneal aesthesiometry should be interpreted within the context of a comprehensive anterior segment evaluation. The following complementary investigations contribute to a complete picture of corneal nerve health:

- In vivo confocal microscopy (IVCM): Provides direct quantitative imaging of subbasal nerve fibre density (NFD), tortuosity, and morphology. IVCM and aesthesiometry are complementary — IVCM assesses structural nerve architecture while aesthesiometry measures functional sensory output. Parallel reduction in both confirms organic nerve damage; dissociation (reduced NFD with preserved sensitivity, or vice versa) suggests compensatory mechanisms or selective fibre type involvement.
- Optical coherence tomography (OCT) / OCT-A: Anterior segment OCT documents epithelial and stromal thickness changes; loss of epithelial thickness over the visual axis in the setting of reduced CS confirms neurotrophic epitheliopathy. OCT-A may reveal limbal vascular plexus changes associated with severe surface disease.
- Corneal topography: Irregular astigmatism, focal anterior elevation, and progressive keratometry changes in an eye with reduced sensitivity suggest progressive NK-related stromal degradation or ectasia.
- Tear film assessment (TBUT, Schirmer's, osmolarity): Essential for distinguishing NK-associated DED from primary aqueous-deficient or evaporative DED and for identifying the reflex tear arc impairment that accompanies reduced sensitivity.
- Slit lamp biomicroscopy with fluorescein: Documents the size, depth, and morphology of any epithelial defect; staining pattern in NK (rose bengal, lissamine green) reveals devitalised epithelial cells not visible with fluorescein alone.

9. The Smart Cochet-Bonnet Aesthesiometer by Akriti Ophthalmic: Design Advantages

The Smart Cochet-Bonnet Aesthesiometer by Akriti Ophthalmic Pvt. Ltd. embodies the essential features that make the Cochet-Bonnet the reference standard, while incorporating ergonomic refinements suited to high-volume clinical use:

- Precision nylon monofilament (0.12 mm diameter): This specific diameter produces the clinically validated pressure range of 11–200 mg/0.0113 mm², matching the physical parameters of the original Cochet-Bonnet design on which all published normative data are based.
- 60 mm maximum length: Full range from 5–60 mm enables measurement across the entire clinical spectrum from complete anaesthesia to normal sensitivity, using a single standardised instrument without the need for supplementary filaments.
- Forefinger-operated control: The slider mechanism allows one-handed, real-time adjustment of filament length while maintaining the instrument in position at the slit lamp, minimising patient movement artefact between successive measurements.
- Clear graduated scale: Directly readable during the examination without requiring the clinician to withdraw the instrument, enabling rapid threshold determination and efficient multi-zone corneal mapping.
- Fully retractable tip: When not in use, the monofilament retracts completely into the instrument body, protecting it from mechanical deformation that would alter calibration. This is the single most common cause of measurement error in poorly maintained aesthesiometers.
- Robust construction: Designed for long-term clinical use with standard sterilisation protocols (alcohol wipe of

filament tip between patients; instrument body with moist-wipe compatible housing).
- Slit lamp compatibility: The instrument handle and balance are optimised for slit lamp-guided use, enabling precise perpendicular approach to the corneal surface under magnification.

Clinical Note: The retractable tip is a critical feature. Bent or kinked monofilaments deliver unpredictable buckling forces and systematically overestimate corneal sensitivity. Clinicians should inspect the filament for straightness before each use and replace the instrument if deformation is observed.

10. Limitations and Considerations

- Contact method: Direct filament contact with the corneal epithelium carries a theoretical risk of microabrasion and infection if hygiene protocols are not followed. Filament tip wiping with 70% IPA between patients and inspection for deformation are mandatory.
- Mechanical stimulus only: The Cochet-Bonnet is unable to deliver thermal or chemical stimuli, which test C-fibre subtypes separately from A-delta fibres. Pathologies selectively affecting specific nerve fibre types may produce dissociated sensitivity loss not captured by purely mechanical testing.
- Subjectivity: As a psychophysical test, results depend on patient attention, cooperation, and verbal or motor response. Patients with cognitive impairment, language barriers, or poor fixation may produce variable results.
- Inter-examiner variability: Technique standardisation- particularly the perpendicularity of filament approach and the rate of advancement- contributes to measurement variance. Training of all clinicians using the instrument in a given practice is strongly recommended.
- Limited low-stimulus sensitivity: At the lower end of the pressure range (60 mm), the instrument cannot detect subtle sub-threshold nerve dysfunction detectable by the more sensitive non-contact gas aesthesiometer. Early diabetic and post-refractive nerve loss may be missed if sensitivity is reduced but still above the 50–60 mm range.

11. Discussion

Corneal sensitivity measurement occupies a unique position in ophthalmic diagnostics: it bridges the gap between structural imaging (IVCM, OCT) and functional assessment, providing a clinician-friendly, quantitative, and reproducible measure of corneal nerve health that directly informs clinical decisions. In the era of neurotrophic keratopathy-specific therapy- including topical nerve growth factor (cenegermin), serum-derived eye drops, and amniotic membrane transplantation- the ability to diagnose and stage NK accurately is no longer merely academic but has direct treatment and reimbursement implications.

The Cochet-Bonnet aesthesiometer achieves the clinical ideal of simplicity and validated precision. Its absence from many clinical settings reflects a historical perception that corneal sensitivity testing is a specialist-only procedure, a perception that the instrument's practical design actively challenges. Any ophthalmologist with a slit lamp can perform a Cochet-Bonnet examination in under three minutes, obtaining clinically actionable information that cannot be derived from any other standard test in the ophthalmic clinic.

The Smart Cochet-Bonnet Aesthesiometer from Akriti Ophthalmic addresses the practical barriers to wider adoption by combining the calibrated precision of the original design with ergonomic refinements- the forefinger control, retractable tip, and readable scale- that reduce examiner-dependent variability. The availability of this instrument through Akriti Ophthalmic's pan-India distribution network of over 4,000 hospitals positions it as a genuinely accessible tool for any practice seeking to integrate formal sensitivity testing into their diagnostic workflow.

Looking forward, the complementary use of corneal aesthesiometry with IVCM-derived nerve density measurements represents the most comprehensive approach to corneal nerve health assessment. As evidence continues to accumulate linking corneal nerve parameters to systemic neuropathy in diabetes, multiple sclerosis, and Parkinson's disease, the cornea is emerging as a non-invasive 'neural biopsy' site- with the Cochet-Bonnet aesthesiometer as the functional component of this assessment.

12. Conclusion

Corneal sensitivity is a fundamental indicator of corneal nerve health, ocular surface homeostasis, and — increasingly — systemic neurological disease. The Cochet-Bonnet aesthesiometer, with over 60 years of validated clinical use, remains the reference standard for its measurement in routine practice. The Smart Cochet-Bonnet Aesthesiometer from Akriti Ophthalmic Pvt. Ltd. delivers this gold-standard capability in an instrument engineered for durability, precision, and clinical ease of use, at a price point accessible to the full range of ophthalmic practices across India and globally.

Every corneal specialist, comprehensive ophthalmologist, and diabetologist managing patients with ocular surface disease should have access to formal corneal sensitivity testing. The Smart Cochet-Bonnet Aesthesiometer makes this possible. By incorporating aesthesiometry into the diagnostic routine for dry eye, herpetic keratitis, diabetes-related ocular surface disease, post-refractive surgery follow-up, and any patient presenting with corneal epithelial breakdown, clinicians can detect neurotrophic disease earlier, stage it accurately, monitor treatment responses objectively, and ultimately prevent the progression to corneal blindness that unchecked neurotrophic keratopathy invariably produces.

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