

BRIDGING THE GAP: EXPLORING THE MECHANISM OF HIJAMA BI'L SHART (WET CUPPING) RELIEVING CERVICAL SPONDYLOTIC RADICULOPATHY (WAJA-UL-UNUQ)

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INTRODUCTION

Cervical spondylosis refers to a range of age-related degenerative changes in the cervical spine, affecting the intervertebral discs, facet joints, ligamentum flavum, uncovertebral joints, and laminae. These changes develop slowly over time and are commonly seen in individuals beyond their fifth decade. Problems arise when deteriorating structures press against the spinal cord or nerve roots, producing chronic neck pain and stiffness. One well-recognised consequence is cervical radiculopathy, in which a compressed or irritated nerve root generates pain, tingling, or weakness that travels down the arm.^[1] Symptoms typically begin as dull neck pain before spreading into the arm along a recognisable nerve distribution.^[2] In Unani medicine, Waja broadly denotes pain arising from either a temperamental imbalance (Su-e-Mizaj) or a physical disruption. Joint-related pain falls under Waja-al-Mafasil, which is further divided by cause and location.^[3] Subtypes include Nigras (gout), Waja-al-Warik (hip pain), Waja-al-Rukbah (knee pain), and Waja-al-Unuq or Waja-al-Raqabah for neck pain.^[4] Classical Unani texts do not use terms like "cervical spondylosis" or "radiculopathy," since these are modern classifications. However, their descriptions of Waja-al-Unuq and Waja-al-Asab Unuqiyah bear a strong resemblance to these conditions as understood today.^[3]

A fundamental tension runs through the existing literature on Hijama bi'l Shart and cervical spondylosis radiculopathy. Unani medicine attributes disease to the accumulation of *mawad fasida*, diminished *tab'i-quwwat*, and disruption of normal Mizaj. Biomedical research, by contrast, situates cervical radiculopathy in mechanical nerve root compression — from disc herniation, osteophyte formation, or ligamentous thickening — compounded by neurogenic inflammation and central sensitization that together perpetuate chronic pain. These are genuinely different ways of explaining the same

clinical problem. What is missing from the literature is a serious attempt to connect them. The question of how concepts such as the removal of morbid humoral matter or the restoration of vital function correspond to measurable physiological events i.e., reduced venous congestion, improved microcirculation, cytokine modulation, or normalized sensory processing, remains largely unaddressed. Equally, the specific mechanisms through which cupping acts, suction-induced ischemia-reperfusion, endogenous opioid release, segmental pain inhibition, have not been systematically examined

through a Unani conceptual lens. The practical irony is that both traditions point toward similar outcomes: pain reduction, restored neck mobility, and relief from radicular symptoms. Yet the theoretical distance between humoral-functional Unani reasoning and the neuro-pathophysiological account of radiculopathy have not been seriously worked through. Closing that gap is both a conceptual and a research priority.

Clinical Picture and Pathophysiology of Cervical Spondylotic Radiculopathy

Neck pain remains one of the more stubborn public health challenges of the modern era, resistant to meaningful reduction despite substantial clinical and research investment. Global estimates from 2021 recorded approximately 62.8 million people living with the condition (95% UI: 42.0–82.5 million), with around 15.4 million incident cases that year (95% UI: 10.2–21.2 million). The associated burden reached 6.4 million DALYs (95% CI: 3.8–9.8 million), reflecting the considerable loss of healthy life attributable to this condition worldwide. Between 1990 and 2021, raw case numbers among younger age groups rose in line with population growth, yet age-standardised rates for prevalence, incidence, and DALYs remained essentially flat. Three decades of stable proportional burden suggest that existing interventions have contained the relative load — but ongoing demographic ageing and global population growth mean absolute case numbers will continue climbing regardless.^[5] Cervical Spondylotic Radiculopathy (CSR) sits at the centre of this burden, accounting for 60–70% of cervical spondylosis presentations in clinical practice. Prevalence is estimated at approximately 3.3 cases per 1000, with an age-adjusted incidence of 0.8 per 1000 annually. Degenerative changes tend to concentrate at the C5–C6 and C6–C7 levels, placing the C6 and C7 nerve roots at greatest risk — with C7 emerging as the most frequently affected level across published clinical series.^[6] CSR does not, however, present uniformly across populations. A systematic review documented considerable variation in prevalence between sexes: male prevalence ranged from 1.07 to 1.76 per 1000, while female prevalence varied far more widely — from 0.63 to 5.8 per 1000. Variation of this magnitude reflects genuine biological and epidemiological differences between sexes, with implications for both clinical assessment and research design.^[7] Cervical radiculopathy develops when spinal nerve roots in the cervical region are compressed or inflamed. The two most common underlying causes are cervical intervertebral disc herniation and cervical spondylosis. Disc herniation, despite its prominence in lumbar radiculopathy, contributes to only around 21.9% of cervical radiculopathy cases. Cervical spondylosis is a considerably more frequent driver, arising from cumulative age-related wear across the cervical motion segments. Progressive disc degeneration reduces disc height and narrows the intervertebral foramina.^[8] Reduced disc height redistributes mechanical load onto the vertebral bodies and unconvertbral joints. Sustained

loading at these sites promotes osteophyte formation and bony hypertrophy, and progressive joint enlargement further encroaches on foraminal space until cervical nerve root compression becomes sufficient to produce radiculopathy.^[9] The nucleus pulposus of degenerating cervical discs has been identified as a trigger for immune-mediated responses. Disc breakdown or disruption of local immune homeostasis initiates a biochemical cascade in which Neuropeptides are released, driving localized inflammation and sensitizing adjacent nociceptive pathways — producing the axial cervical pain patients typically report early in the condition. Concurrent compression of soft tissue adjacent to the disc reduces blood flow through the cervical and shoulder regions. Sustained vascular compromise at perineural vessels produces ischemia, tissue edema, and elevated pro-inflammatory mediators, while impaired microcirculation allows acidic metabolic byproducts to accumulate locally. Collectively, these processes reduce axonal conduction velocity in the affected nerve root, generating the radicular pain, sensory disturbance, and neurological deficits characteristic of CSR.^[10] Cytokine profiling at herniated cervical disc sites has consistently shown elevated interleukin-1 beta (IL-1 β), tumor necrosis factor-alpha (TNF- α), and interleukin-6 (IL-6) — each directly implicated in pain generation associated with cervical radiculopathy. Where nerve roots supplying the neck and shoulder are edematous or inflamed, disc degeneration is seldom absent; annular rupture allows inflammatory mediators to enter the spinal canal and reach the nerve roots directly. The clinical result is cervical radiculopathy — pain, sensory loss, and neurological deficit arising from a sustained mechanical and inflammatory insult to the nerve root.^[11]

Cervical Spondylosis and Radiculopathy in Unani System of Medicine

In Unani medicine, *Waja* refers to a sensation of bodily discomfort or unease caused by a derangement of temperament (*Su-e-Mizaj*) or a disruption in structural continuity. Unlike a single condition, *Waja-al-Mafasil* encompasses a range of joint problems—including pain, stiffness, and swelling—classified by their causes and the joints involved.^[12] Common subtypes include *Niqras* (gout), *Waja-al-Warik* (hip joint pain), *Waja-al-Rukbah* (knee joint pain), *Waja-al-Unuq* or *Waja-al-Raqabah* (neck pain), and others.^[4] While classical Unani texts predate modern anatomical terms and do not mention "cervical spondylosis" or "radiculopathy," a closer look at traditional literature shows that what is described as *Waja-al-Unuq/Waja-al-Raqabah* (neck pain) and *Waja-al-Asab Unuqiyah* (cervical neuralgia) aligns closely with what modern medicine identifies as cervical spondylosis and radiculopathy, respectively. According to *Sharah Asbab (Tarjuma-i-Kabeer)*, the Unani translation of *Sharah-Al-Asbab wa Al-Alamat* by Hkm. Kabeeruddin, *Waja-al-Unuq Asabi* (cervical radiculopathy pain) is a subtype of *Waja-al-Asab* (neuritic pain). It is characterized by pain originating at the affected nerve root and radiating along the

distribution of the involved nerve and its branches.^[13] The pain can vary from mild to severe, often worsens at night, and is accompanied by a tingling sensation. It does not follow a consistent pattern; with patients reporting sudden flare-ups of sharp pain that subside on their own. On physical examination, tenderness at the nerve root site is commonly observed, sometimes with sensory changes or muscle weakness in the area supplied by the nerve.^[13] *Waja-al-Unuq Asabi* may present in the following two forms:

I. *Waja'al-Unuqi Qamahduwi* (neck pain radiating to the occiput)

This form involves the upper four cervical nerves (*A'sab Fiqr al-Unuq*), with pain that can spread across the neck and work its way toward the nape (*Mu'akhkhar al-'Unuq*), the *Nuqrah*, and the occiput (*Mu'akhkhar al-Ra's* i.e., *Qamahduw*). It is not a particularly common presentation, but when it does show up, head movement becomes noticeably difficult — the pain is severe enough that patients often describe what they are feeling simply as a headache.^[13]

II. *Waja'al-Unuq Adudi* (neck pain radiating to the humerus i.e., arm)

This variant involves pain stemming from irritation of the lower four cervical nerves (*Asab Fiqr al-Unuq*) and the first thoracic nerve (*A'sab Fiqr al-Sadr*). Discomfort typically radiates extensively across the posterior lower neck, posterior upper arm, wrist, and fingers.^[13] In Unani pathophysiology, sleeping in a prolonged abnormal neck posture is considered one of the main things that bring on *Waja-al-Unuq*. It may also arise from extreme cold exposure, which induces spasms in the neck musculature (*Al-Adalat al-Unuqiyyah*), particularly the sternocleidomastoid muscle (*Al Adalah al Qasiyah al-Halmiyah*). This condition generally presents unilaterally, with any abrupt head movements — such as coughing or laughing — aggravating the neck pain.^[13] When these muscular spasms take hold, patients tend to report *waja* (pain), *salabh* (stiffness), reduced mobility in the neck and shoulder, and *suda* (headache), along with localized swelling and tenderness.^[14,15,16] In Unani nosology, the condition is traced back to two core disturbances — simple dys-temperament (*Su-e-Mizaj Sada*) and material dys-temperament (*Su-e-Mizaj Maddi*) — both of which are seen as the root drivers of *Waja-al-Unuq* or *Waja-al-Raqbah*.

Ibn Zuhri's *Kitab al-Taisi* takes a close look at *Amrad-e-Raqbah* (diseases of the neck) and *Waja-al-Raqbah* (neck pain), tracing their origins mainly to *tafarruq-e-ittisal* (discontinuity/breach) and *sudda* (obstruction) within the neck's vascular structures (*uruq al-raqbah*) or neural elements (*a'sab*). Additional contributors to neck pain include *tashannuj* (muscular spasm/stiffness), *waram* (inflammation), *tamaddud*, *imtila* (congestion/distension), and *inkhizal-e-fiqr al-raqbah* (cervical vertebra displacement). On the question of *tashannuj*, Ibn Zuhri draws a clear line between two presentations:

the kind that creeps in gradually comes from *su-e-mizaj yabis* (simple dry dys-temperament) and tends to produce milder pain, while the acute form — *tashannuj-e-hadd* — is driven by *khilt fadil* (superfluous humor) pressing on neural structures and brings on severe pain. Ibn Zuhri also describes how *khilt fadil* building up between the *fiqr al-Unuq* (cervical vertebrae) can push them out of place — *inkhizal-e-fiqr al-raqbah* — which then triggers *waram-e-a'sab* (nerve inflammation) and sometimes *waram-e-nukha* (spinal cord inflammation), both of which produce pain. A shift to the side (*inkhizal-e-janibi*) leads to *tashannuj* and *tamaddud*, while a shift inward (*inkhizal-e-dakhili*) can cause *istirkha* (flaccid paralysis) and, in severe cases, *sudad-e-nukha* (spinal cord compression), which Ibn Zuhri considered potentially fatal.^[17]

Management in Unani Medicines

In the Unani Medical system, *Waja al-Unuq* (cervical spondylosis) and its associated manifestations are handled through three broad treatment tracks — *Ilaj bi'l Taghthiyah* (dietotherapy), *Ilaj bi'l Dawa* (pharmacotherapy), and *Ilaj bi'l Tadbir* (regimental therapy). Within *Ilaj bi'l Tadbir*, hands-on interventions like *takmid* (fomentation), *dalak* (massage), *Hijama* (cupping therapy), and *fasd* (venesection) are used in day-to-day clinical settings, sometimes on their own and sometimes alongside pharmacotherapy (*Ilaj bi'l Dawa*), depending on what the patient's condition calls for. Among these, *Hijama* (cupping therapy) has gained particular traction in contemporary Unani practice. At its core, Unani treatment for this condition works toward pain relief, reducing morbidity, and getting mobility back — all anchored in the principle of *ta'dil-e-mizaj* (temperamental normalization). More specifically, treatment focuses on restoring joint health by clearing out *khilt fadil* or morbid humors through *istifragh-e-mawad* (evacuation of superfluous humors), easing symptoms through *taskin-e-alam* (symptom palliation), and strengthening the affected joints via *taqwiyat-e-mafasil* (joint fortification). For pain, *musakkinat* (analgesic agents) and *muhallilat* (anti-inflammatory remedies) are the go-to options, while nerve and muscle strengthening medications are brought in to support overall musculoskeletal health.^[14,15,16,17]

Regimens of *Ilaj bi'l Tadbeer* for the Management of *Waja'al-Unuq* and *waram-e-A'sab*

S. No	Name of Regimental Therapy	Definition	Site	Reference(s)
1.	<i>Fasd</i> (venesection)	Venesection is a therapeutic intervention aimed at the systemic removal of pathogenic humoral constituents, thereby facilitating the restoration and maintenance of physiological humoral equilibrium.	<i>Qifal</i> (cephalic vein) <i>Akhal</i> (median cubital vein)	(Hamdani,2011; J; Sina, 2010)
2.	<i>Hijama bi'l Shart</i> (wet cupping)	Scarification-based cupping therapy, performed to induce local bloodletting, facilitates the evacuation of pathological substances from the targeted tissue.	<i>Kahil</i> (over the interscapular region), <i>Akhda'ayn</i> (sides of the neck), and <i>Nuqra</i> (over the nape of the neck).	(Hamdani, 2011; J. A. Khan et al., 2017; Sina, 2010; Zuhr, 1992)
3.	<i>Hijama Bi'la Shart</i> (dry cupping)	Dry cupping, frequently referred to as wind cupping, is a type of cupping in which the skin is sucked and the surrounding area is massaged without removing the unclean blood.	<i>Kahil</i> , <i>Akhda'ayn</i> , <i>Nuqra</i> .	(Hamdani, 2011; J. A. Khan et al., 2017; Sina, 2010; Zuhr, 1992)
4.	<i>Tal'iq al-Alaq</i> (Leech Therapy)	One way of evacuating morbid matter is the regular administration of leeches, which is particularly helpful in situations where venesection or <i>Hijama</i> is not performed correctly.	At the site of the pain.	(Hamdani, 2011; J. A. Khan et al., 2017; Sina, 2010; Lone et al.,2011; Zuhr, 1992)
5.	<i>Dalak</i> (therapeutic massage)	Massage involves techniques ranging from light, moderate, and deep pressure and regimen involving manual manipulation of muscles.	At the site of the pain.	(Hamdani, 2011; J. A. Khan et al., 2017; Sina, 2010; Lone et al.,2011; Zuhr, 1986)
6.	<i>Naful</i> (Irrigation/douche)	It involves slowly and steadily dripping a medicinal liquid over a chosen body part for a predetermined duration from a fixed height.	At the site of the pain.	(Hamdani, 2011; J. A. Khan et al., 2017; Sina, 2010; Lone et al.,2011; Zuhr, 1986)
7.	<i>Bukhoor/ Inkebab</i> (Vapour bath)	Exposure of a portion of the body or the entire body to fumes from boiling water or medication decoction.	At the site of the pain	Mohammad et al.,2014
8.	<i>Imala</i> (Diverting morbid matter)	Diversion of morbid matter/humour from the affected site to another site, or increase in the flow of humours towards a specific site.	<i>Kahil</i> , <i>Akhda'ayn</i> , <i>Nuqra</i>	(Hamdani, 2011; J.A. Khan et al., 2017; Sina, 2010; Lone et al., 2011; Zuhr, 1986)

History of *Hijama* (Wet Cupping)

Hijama (cupping therapy), an Arabic term derived from *al-hajm* meaning "to suck" or extract, is a bloodletting practice that has shown up across a wide range of cultures going back to ancient times. In Muslim traditions, wet cupping therapy (*hijamah bi'l shart*), commonly termed *hijama*, follows a set sequence: skin demarcation, sterilization, initial cupping (dry phase), superficial puncturing, and secondary cupping (wet extraction phase), and final sterilization.^[18]

Different traditional and complementary medical systems have their own ways of making sense of cupping, but what runs through most of them is the idea of using

negative pressure via specialized cups to draw out toxic or morbid substances from subcutaneous tissues.^[19,20]

The Ebers Papyrus — an ancient Egyptian medical text dating to around 1550 BCE — is where cupping first shows up in writing, with basic instruments and their uses sketched out alongside other treatments.^[21] The WHO has taken a more favorable stance on traditional and complementary medicine — cupping therapy included — noting that the evidence points to real health gains, a track record of reasonable safety, and a side effect burden that sits well below what most pharmaceutical options carry.^[22] Cupping instruments are made from glass, plastic, rubber, or wood and come in

different sizes depending on which part of the body is being treated. In Unani medicine, cupping is thought to work by driving *Imala* (diversion) of morbid material away from the diseased site, pushing out pathological humours through *Istifragh* (expulsion), and improving blood circulation both locally and more broadly. Whether or not the skin is scarified determines which type of *Hijama* is being used — *Hijama bi'l Shart* involves making superficial incisions before reapplying the cup, while *Hijama bi'la Shart* skips the incision entirely.^[23]

Cupping has a long history — long enough that pinning down exactly where it started is not straightforward. The Ebers Papyrus (1550 BCE) from ancient Egypt already describes using cupping and bloodletting to clear unwanted matter from the body, and Greek physicians including Galen picked it up and built on it from there. It fell out of favor in Europe for a time, but Islamic medicine kept the practice alive, worked it into humoral theory and Prophetic medicine, and Unani practitioners today are still drawing on that same tradition when they reach for cupping as a regimental therapy.^[24]

Clinical Evidence: Efficacy of Hijama Bi'l Shart in Cervical Spondylosis

The integration of *Hijama bi'l Shart* (wet cupping) into contemporary evidence-based pain management has gained substantial empirical support in recent years. ➤ Recent clinical trials, evidence-mapping studies, and systematic reviews have consistently highlighted its efficacy in mitigating both the acute and chronic symptoms of cervical spondylosis (traditionally categorized in Unani medicine as *Waja-al-Unuq*).^[25]

Key Clinical Trials in Cervical Spondylosis

1. Tausif K et al., 2017- Hijama vs Tens (NIUM Bengaluru)^[26]

- Design: Open, randomized, controlled trial; 50 patients with radiologically confirmed *Wajaur Raqba* (Cervical Spondylosis), 25 in *Hijama* group and 25 in TENS groups.
- Intervention (*Hijama bi'l shart* protocol): 2 medium cups (5.5cm) on bilateral supraclavicular fossa; 1 large cup (6.5 cm) at C7-T1 junction (posterior neck); 8 sittings on alternate days over 15 days.
- Control: TENS to cervical paravertebral region, 20 minutes, 8 sittings over 15 days.

➤ VAS pain reduction

- *Hijama*: VAS $5.16 \pm 1.21 \rightarrow 2.36 \pm 1.47$ by day 15 ($\Delta \approx 2.8$, $p = 0.001$ within-group).
- TENS: CAS $5.36 \pm 1.25 \rightarrow 3.56 \pm 1.36$ ($\Delta \approx 1.8$, $p = 0.001$).
- Inter-group: Day-15 VAS difference 1.20 in favor of *Hijama* ($p = 0.001$).
- Functional outcome (Neck disability Index/ Vernon & Mior)
- *Hijama*: NDI $20.20 \pm 4.20 \rightarrow 11.00 \pm 3.73$ ($p = 0.001$).

- TENS: NDI $19.92 \pm 4.42 \rightarrow 15.72 \pm 4.47$ ($p = 0.001$).
- Inter-group: DAY-15 NDI significantly lower in *Hijama* group ($p = 0.001$).

Conclusion from authors

Both interventions were effective, but *Hijama* produced greater reduction in pain and disability; inter-group analysis showed *Hijama* had a superior effect to TENS in short-term management of *Wajaur Raqba*.

2. Siddiqui et al., 2024- Clinical and hematological study of Hijama Bi'l Shart (AMU, Aligarh)[27]

- Design: Interventional, open-label observational clinical and hematological study; 110 patients with musculoskeletal pain were screened, 90 enrolled, and 60 completed 4-week *Hijama bi'l Shart*.
- Intervention (*Hijama bi'l Shart*): 4 sittings, once weekly for 4 weeks (total study duration 28 days); 5 medium cups; Sites: 1 cup at C7 vertebra (**Nuqra**)-posterior cervicothoracic junction, 2 cups at **right and left supraclavicular fossae (Katifain)**, and 2 cups on both **scapular regions**.
- Procedure: Cups applied after antisepsis, primary dry suction for 10 minutes until erythema, then multiple superficial incisions and re-application for 5 minutes with EDTA-coated cups.

VAS pain reduction

- Baseline: 7.9 ± 0.73
- Day 14: 6.6 ± 0.51
- Day 28: 3.8 ± 0.63 ; $p = 0.0001$, $t = 17.57$, indicating a highly significant reduction in neck pain over 4 weeks of *Hijama*.

Conclusion from authors: Looking at both clinical outcomes and hematological markers, *Hijama bi'l Shart* (wet cupping) shows a consistent ability to bring pain down across a range of musculoskeletal conditions, cervical spondylosis among them, with meaningful VAS improvements recorded over 4 weeks. The study also maps out where cups are placed for cervical spondylosis specifically, C7 *Nuqra*, bilateral supraclavicular fossae *Katifain*, and the scapular regions.

Studies indicate that cupping works primarily by improving hemolymphatic circulation and clearing metabolic wastes and toxins from the body. The localized negative pressure created by wet cupping appears to drive this process, drawing out toxins accumulated in exudates and purulent fluids just below the skin. Additionally, the targeted bloodletting involved in wet cupping promotes drainage through both vascular and lymphatic channels, removing harmful substances from the skin's microcirculation and surrounding interstitial spaces.

Proposed Mechanistic Pathways of Hijama bi'l Shart in CSR

For a long time, nobody could quite explain how cupping therapy actually worked — but research over recent years has started filling in that picture, pointing to subatmospheric pressure suction, immune system activity, and improved peripheral blood circulation as the main drivers. What the data shows is that cupping raises cutaneous blood flow, shifts skin biomechanics, pushes nociceptive thresholds higher, kicks local anaerobic metabolism into gear, dials down inflammation, and adjusts how the cellular immune system behaves.^[28] A few different theoretical frameworks have been put forward to make sense of how cupping does what it does. The immune modulation theory suggests that stimulating the skin in this way disrupts the local microenvironment enough to send biological signals that trigger the neuroendocrine arm of the immune system. According to a meta analysis, cupping and acupuncture are similar.^[29]

Shaban and Ravalia took a different angle — what they called the genetic theory — arguing that the suction cupping creates puts the skin under mechanical stress, and when that combines with the local anaerobic metabolism it triggers, the resulting signals are strong enough to switch certain genes on or off. In wet cupping, the small skin cuts that are made seem to take this further, setting off gene-expression responses and wound-healing activity in a more targeted way.^[30]

(1) Pain Gate Theory

Injury to local skin and capillary vessels appears to act as a nociceptive stimulus in its own right.^[31] From a neural standpoint, cupping seems to intercept and reshape pain signals as they move through spinal cord nociceptors toward the brain, disrupting how chronic pain gets processed at both ends. Reviews pulling together data from randomized controlled trials have consistently found cupping to be an effective option for managing a range of pain conditions.^[32]

The theory that keeps coming up most often to explain this is the Pain Gate Theory.^[33] Back in 1965, Melzack and Wall laid out the idea that thin and large nerve fibers both carry pain signals from the site of damage to two locations in the dorsal horn of the spinal cord, where inhibitory interneuron can step in and block transmission cells from getting the signal through to the brain.^[34] Both thin (A-delta and C) and thick (A-beta) fibers excite transmission cells. However, thin fiber activity hinders inhibitory cells, whereas large-diameter fibers stimulate them. When these inhibitory cells are excited by large fiber activity, they impede the transmission of pain signals to the brain. The more large fibers are stimulated by movement — touch, pressure, vibration — the less pain is felt. With cupping and other reflexology techniques, the thinking is that activating transmission cells also brings large "A" fibers into play alongside "C" fibers within the spino-thalamic pain pathway.^[34] On top of this, metabolic byproducts like pro-inflammatory

cytokines, lactate, and adenosine triphosphate are well known for sensitizing peripheral nociceptors — and cupping appears to interfere with that process.^[35] When the skin receives a stimulus, the number of active receptor-fiber units goes up and the brain gets fed information about what is happening. So when something like the negative pressure of cupping lands on the skin, it sends out an afferent volley dominated by large-fiber impulses that partly close off the "T" cells while also firing up the presynaptic gate.^[33]

(2) Diffuse Noxious Inhibitory Controls (DNICs)

DNIC gives us another way to think about why cupping relieves pain. At its core, DNIC is about applying a second, localized noxious stimulus somewhere away from the main pain site, which then damps down the activity of wide dynamic range (WDR) nociceptors — this is the physiological backbone of what clinicians have long called "counter-irritation," the idea that one pain can quiet another.^[36] This descending inhibitory pathway is easy enough to trigger in experimental settings, but the field has since tightened up its language to stop animal and human findings from getting lumped together. The term now used for the human experience of this pain-relief effect is Conditioned Pain Modulation (CPM), while DNIC has been kept specifically for the lower brainstem-mediated inhibition seen in live animal studies.^[37]

(3) Reflex Zone Therapy

The Reflex Zone Theory helps explain both the local and wide-reaching effects of cupping from a neurophysiological standpoint. Drawing on traditional European medical practice, cupping applied to the shoulder triangle has shown real clinical usefulness for peripheral neuropathies like carpal tunnel syndrome. This seems to work because the dermatomes being stimulated share segmental neural innervations with the median nerve. Looking at this from a biomechanical angle, the vacuum-induced negative pressure pushes red blood cells (RBCs) out of the microvasculature and into the interstitial compartment — and it does this without tearing the capillary walls. That controlled extravasations, working alongside the strong focal vasodilation cupping produces, improves local hemodynamic in a meaningful way. The resulting hyperemia then helps flush out accumulated metabolic waste and gets cellular repair going within the affected reflex zone.^[33] Running underneath all of this is the principle of cutaneovisceral and viscera cutaneous reflex arcs. What the clinical picture often shows is that trouble in an internal organ tends to show up on the skin surface in specific dermal regions — something that happens because those areas share neuroanatomical, musculoskeletal, and biochemical pathways with the organ in question. When an internal organ is under stress, it sends nociceptive afferent signals through the autonomic nervous system out to the dermatome it corresponds to. That signal from the organ then surfaces as localized cutaneous hyperalgesia, edema, and

disrupted vasomotor tone — showing up on the skin as either reddened, vasodilated patches or pale, vasoconstricted tissue.^[39] Left to run chronically, this kind of reflex activation starts to leave structural marks — the subcutaneous connective tissue thickens and becomes fibrotic, synovial fluid production drops off, and the cartilage and ligaments nearby begin to break down. Applying cupping directly to these dermatomal zones gives cutaneous mechanoreceptors a strong mechanical input to work with. Those sensory signal then moves through autonomic reflex arcs, turning up microcirculation at the surface while also adjusting blood flow and function in the corresponding visceral organs.^[40] Getting to grips with how these reflex pathways work matters — it is what connects traditional therapeutic approaches to modern neuroscience, and what gives interventions like cupping therapy a firmer footing in evidence-based practice.

Clinical Studies Related To the Effects of Wet Cupping on Pain

A recent systematic review and meta-analysis comparing different cupping approaches for low back pain found wet cupping to be clearly the stronger option when it comes to pain relief. The numbers backed this up — wet cupping produced a statistically significant drop in LBP, with Present Pain Intensity (PPI) scores falling meaningfully ($P < 0.1$) against standard care controls. Dry cupping, by contrast, did not manage to move the needle on pain intensity in any clinically meaningful way for LBP patients.^[41] Wet cupping's reach goes beyond local tissue effects — it also appears to work on the psychological side of pain, which matters particularly in conditions like migraines. One recent trial found that a wet cupping session brought down both physical pain and mental distress within ten minutes, and those gains held up over a one-month follow-up. What stood out was that after treatment, sleep duration — not mental distress — became the main factor predicting physical pain levels, hinting that wet cupping's pain-relieving effect may be tied closely to how it handles the psychological dimension of pain.^[42] A retrospective clinical analysis found that even a single wet cupping session could keep pain at bay for up to a month across a range of chronic musculoskeletal and neurovascular pain conditions.^[43]

(1) Clinical Studies Related to the Effects of Wet Cupping on Neck Pain

More recent clinical work adds to the case for wet cupping in cervical musculoskeletal conditions, particularly mechanical neck pain. A 2025 clinic-based experimental study found that one targeted Hijama session was enough to produce noticeable pain relief within 48 hours. Post-treatment measurements showed a 65% drop in pain intensity on the Visual Analog Scale (VAS) and a 46% reduction in functional impairment on the Neck Disability Index (NDI) — both statistically significant. Taken together, these numbers make a strong case for wet cupping as a fast-acting option for bringing cervical pain down and getting mobility back.^[44] That picture carries over into degenerative conditions too.

A clinical case series testing a standardized Unani protocol (Hijama-Bil-Shart) over 42 days found much the same thing — patients who received wet cupping over the suprascapular and C7-T1 regions saw statistically significant reductions in both localized and radicular neck pain ($P = 0.001$).^[45] Pulling the evidence together, cupping therapy has a reasonable clinical track record across a range of pain conditions, with neck pain being one of the better-supported applications. The quality of the evidence sits somewhere between very low and moderate, but the overall direction is consistent — cupping appears to be safe, effective, and worth considering as a non-pharmacological add-on in the management of chronic musculoskeletal and neuropathic disorders.^[46]

Adverse Effects

Adverse events (AEs) following *Hijama bi'l shart* are infrequently documented, with most incidents exhibiting mild to moderate severity. The predominant adverse side effects associated with cupping therapy include scar formation and burns. Additional reported complications encompass bulla formation, localized petechiae or tenderness at the cupping site, abscess development, cutaneous infections, transient skin discoloration, insomnia, post-inflammatory hyper-pigmentation and vasovagal syncope.^[21]

Sites of Hijama (cupping therapy) for Cervical Spondylosis Radiculopathy

S.No	Anatomical Sites
1.	Nuqra: refers to the nape, specifically the lowest section of the back of the neck centered around the prominent bump of the 7th cervical vertebra (Hamdani, 2011). The historical physician Ibn Sina advised against performing wet cupping (<i>Hijāma bi'l Shart</i>) on this area. He reasoned that because the rear portion of the brain is responsible for retaining memory (<i>Hafiza</i>), applying cupping therapy to the Nuqra could compromise this function and lead to forgetfulness (Ibn Sina, n.d.).
2.	Akhda'ayn: This corresponds anatomically to the lateral aspects of the neck, situated inferior to the mastoid process and just below the hairline. This site is also described as immediately posterior and inferior to the earlobe (Hamdani, 2011). Ibn Sina notes that <i>Hijama bi'l Shart</i> at <i>Akhda'ayn</i> is therapeutically equivalent to venesection of the <i>al-Qifal</i> (cephalic vein) and proves efficacious for <i>irtiash al ra's</i> (vertigo) (Ibn Sina, 2010).
3.	Yafukh: This corresponds to the midline joining both parietal bones, situated approximately 10-14cm above the posterior hairline, representing the central vertex of the cranial vault (Hamdani, 2011).

4.	Mankib: Unani classical literature delineates the <i>Mankib</i> site as the region on each shoulder situated precisely posterior to clavicle (Masihi, 2986). Anatomically, this corresponds to the posterior aspect of the deltoid muscle adjacent to its origin at the scapular spine and acromion process. Across various traditional medical systems, cupping and acupuncture are applied at this locus to address arm, shoulder, and neck pain; paresis; and cervical stiffness.
5.	Qamḥadūwah: Corresponds anatomically to the external occipital protuberance on the posterior skull, overlying the cerebellum and brainstem (Hamdani, 2011). However, <i>Hijama bi'l shart</i> at this site carries the risk of inducing dementia (Baqar, 1939).
6.	Kahil: Corresponds anatomically to the interscapular region between the shoulders, serving as the designated cupping site in this area (Hamdani, 2011). Ibn Sina posits that <i>Hijama bi'l shart</i> at <i>kahil</i> ; is therapeutically equivalent to venesection of the <i>al-basaliq</i> basilica vein) and confers benefit for shoulder and neck pain (Ibn Sina, n.d).
7.	Acromion Process: Anterior and inferior to the acromion, on the upper portion of the deltoid muscle, in a depression formed when the arm is in full abduction. <i>Hijama bi'l shart</i> on the site is applied in case of pain, limited motion and paresis in the shoulders and arms (Domink Irnich,2013)
8.	Scapula: This anatomical landmark-positioned posteriorly between the scapular spine and the acromion-serves as a primary intervention point for managing musculoskeletal dysfunction. It is specifically indicated for patients presenting with referred pain or restricted range of motion in the upper extremities and dorsal trunk (Chirali,2014)
9.	Lateral aspect of trapezius muscle: Application of <i>Hijama bi'l Shart</i> to the bilateral depressions of the lateral trapezius muscle, located at the nuchal hairline, yields significant therapeutic effects. This intervention induces relaxation of the cervical musculature and alleviates discomfort associated with shoulder pain, upper back pain, and occipital haeadche (Cirali,2014).
10.	Below C2: Situated inferior to the second cervical vertebra, along the medial border of te scapula, approximately 10cm lateral to the midline spine on each side. <i>Hijama bi'l shart</i> at this locus is indicated to alleviate pain and stiffness in the neck, shoulder and back (Irnich, 2013).
11.	Coracoid Process: Wet cupping applied near the shoulder-specifically on the upper arm close to the coracoid process- can help treat C5 and C6 nerve compression. This technique targets pain that starts at the front of the shoulder and travels down the back of the arm and forearm to the hand. .
12.	Humerus: In cases where the C7 and C8 nerves in the neck are compressed, wet cupping is performed on the upper-outer back of the arm (near the triceps). This helps alleviate pain that travels between the shoulder and the upper back, as well as shooting pains that extend down the back of the arm to the forearm ((Dommerholt & Fernandez-de-las-Penas,2018).
13.	Below the occipital bone: In the space between the sternocleidomastoid and trapezius muscles. In cases where the C2 and C3 nerve roots are compressed, <i>Hijama bi'l shart</i> on this location is helpful.
14.	Below the spinous process of the 7th cervical vertebra: Near the acromion process level. On this site, <i>Hijama bi'l shart</i> is recommended for shoulder and neck pain, particularly searing agony. Stronger suction is utilized for acute or severe pain, whereas weak to medium cupping is done for ten to fifteen minutes in cases of chronic discomfort.

Methodological Consideration for Future Research

Hijama bi'l Shart has history and theory behind it — but the research catching up to it has real problems. Studies in this space tend to pull in different directions methodologically, and too many of them lean on self-reported outcomes that are hard to compare across settings. Getting this treatment taken seriously in clinical protocols for Cervical Spondylotic Radiculopathy (CSR) is going to require trials that are better designed, more consistent in how they are run, and actually built to answer questions about what is happening physiologically.

Rigorous Randomized Controlled Trials (RCTs)

The field needs to stop relying on preliminary observational work and step up to something more rigorous. What is genuinely missing right now are large, well-powered, multi-centre RCTs that bring in CSR patients whose radicular signs have been confirmed both clinically and radiologically. If the goal is to pin down

what wet cupping is actually doing — separate from placebo effects or the general experience of receiving clinical care — then trials need to be designed with multiple comparison arms, setting *Hijama bi'l Shart* against:

- **Sham intervention:** Using validated sham-cupping devices (e.g., cups with stimulated suction and blunt scarification tools that do not breach the epidermis).
- **Active physical therapies:** Such as Transcutaneous Electrical Nerve Stimulation (TENS) or targeted cervical physiotherapy.
- **Conventional pharmacological care:** Standard-of-care NSAIDs or neuropathic pain modulators (e.g., gabapentinoids).

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