Review Article

Exploring scraping therapy: Contemporary views on an ancient healing – A review

Eric Chun Pu Chu[1], Arnold Yu Lok Wong[2], Patrick Sim[3], Friso Krüger[4]

[1]New York Chiropractic and Physiotherapy Centre, New York Medical Group, Hong Kong SAR, China, [2]Department of Rehabilitation Sciences, The Hong Kong Polytechnic University, Hong Kong SAR, China, [3]Australian Chiropractic College, Adelaide, South Australia, Australia, [4]Chiropraktische Familienpraxis, Lüneburg, Germany

Abstract

Gua sha is a traditional healing technique that aims to create petechiae on the skin for a believed therapeutic benefit. Natural healings are mostly based on repeated observations and anecdotal information. Hypothetical model for healing does not always fit the modern understanding. Yet, the mechanisms underlying Gua Sha have not been empirically established. Contemporary scientific research can now explain some events of traditional therapies that were once a mystery. It is assumed that Gua Sha therapy can serve as a mechanical signal to enhance the immune surveillance function of the skin during the natural resolving of the petechiae, through which scraping may result in therapeutic benefits. The current review, without judging the past hypothetical model, attempts to interpret the experience of the ancient healings in terms of contemporary views and concepts.

Keywords: Gua Sha, immune response, petechia, scraping therapy, traditional healing

Introduction

There is significant demand for traditional and complementary medicine practices worldwide. More countries have gradually come to accept the contribution of traditional medicine for the individual health care and are considering traditional medicine to be integrated into health service delivery.[1] In wealth countries, such as Australia and the United States, older populations usually use complementary medicines for maintenance of health, pain management, lowering cholesterol levels and blood pressure, and improving sleep. Common modalities include natural products, acupuncture, chiropractic, and osteopathy. According to a national survey in China in 2009, the number of traditional Chinese medicine visits accounts for 18% of all medical visits to surveyed institutions. Due to an upsurge of the noncommunicable disease burden in older populations, there is accentuated emphasis on task sharing and shifting health care delivery to nonphysician medical providers.[2] This health care service is particularly important for lower income countries, such as the Lao People's Democratic Republic, 80% of the population lives in rural areas.[3] Gua sha is an ancient technique used in traditional East Asian medicine.

Gua Sha, also known as skin scraping, scraping therapy, or coin rubbing, has long been a traditional healing that is widely practiced in China and South East Asia. It is often used to treat nuchal pain, shoulder tension, myalgia, chronic pain, and other muscle issues.[4] Traditional healings were often embedded in the culture in which they were practiced. Gua Sha involves scraping the body surface with a tool (e.g. a buffalo horn scrape) with or without a skin lubricant to intentionally create petechiae, which is traditionally called *Sha* and can be loosely translated as stagnant blood. *Gua Sha* roughly translates into English as "dredging meridian stagnation". Protoscientific concepts of

Address for correspondence: Dr. Eric Chun Pu Chu, New York Chiropractic and Physiotherapy Centre, 41/F Langham Place Office Tower, 8 Argyle Street, Hong Kong SAR, China.
E-mail: eric@nymg.com.hk

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scraping therapy being responsible for removing interference have been reported as fictitious and simplistic.[5]

There are two western techniques that also scrape the skin but they differ from Gua Sha in terms of purposes. In ancient Greece, athletes lathered themselves with olive oil, and then scraped it clean with a scraper (called a *strigil*).[6] This gentle scraping aimed mainly for hygienic purpose and brought about a sense of relaxation rather than for any therapeutic outcome. Conversely, the Graston technique® is a form of deeper soft-tissue scraping mainly used in chiropractic care, attempting to stretch, separate, and break down adhesions.[7] Both of the above-mentioned techniques fall beyond the scope of the present review.

Cupping is another type of ancient healing of using heated cups to create petechiae for a therapeutic purpose. It has been used in the alleviation of pain and many other complaints for millennia[8] and is still commonly practiced as part of traditional Oriental, Persian and European medicine.[9] Cupping therapy is similar to Gua Sha in terms of its hypothetical, physiological and clinical basis. Primary care physicians at the frontline promote health for the greatest variety of patients. Through continued investigation into the mechanisms of complementary and alternative therapies, they will be able to optimize and apply certain ancient regimens to clinical practice, which may maximize benefit to patients. This review aims to shed light on the possible responses and potential outcomes of scraping therapy and provide proposed explanation made on the basis of an understanding of contemporary physiology.

The Therapeutic Marks

The scraping marks (petechiae and ecchymoses) are formed when capillaries break open and blood leaks into the subcutis. These marks fade and completely resolve over 2–5 days.[10,11] Disappearance of petechiae and ecchymoses occurs via erythrocyte lysis. Cell debris is concurrently removed by microglia/macrophages. Haemolysis is associated with the release of haemoglobin and its catabolic products. Each haemoglobin molecule is made up of four heme groups surrounding a globin group, forming a tetrahedral structure. Haemolysis liberates a large amount of haemoglobin into the bloodstream or tissues that are taken up by macrophages via their scavenger receptor (CD163) with the subsequent induction of haem oxygenase (OH) for subsequent haem degradation.[12] The colour of the subcutaneous tissue reflects the physiologic sequences of haemoglobin catabolism and its conversion to bilirubin (BR) and haemosiderin. It is recognized that the ecchymotic region will have different shades of colour, reflecting the differential rates of haemoglobin catabolism.[13]

Haem catabolism

Haem (also known as iron protoporphyrin IX) is a large complex, comprising a haem ring (protoporphyrin IX) and an atom of ferrous (Fe^{2+}) iron located at the ring centre. Free haem is dangerous in excessive quantities which is why it is quickly removed from tissues.[14] The HO system is responsible for haem degradation, converting haem to biliverdin (BV), during which ferrous iron (Fe^{2+}) is released and carbon monoxide (CO) is emitted.[15] Three isoforms of HO enzymes (HO-1, HO-2 and HO-3) catalyze the initial reaction in haem catabolism.[16] HO-1, with the inducible character and widespread distribution, is regarded as the central part in the HO system.[17] HO-1 is recognized as having major immunomodulatory and antiinflammatory properties, which have been demonstrated in HO-1-deficient mice and human cases of genetic HO-1 deficiency.[18] HO-2 and HO-3 are the constitutive isoforms. HO-2 is expressed basically in neuronal populations and endothelial cells. HO-3 is poorly understood isoform and nearly identical to HO-2 (90%) in its amino acid sequence but shows only low catalytic activity.[19]

Bilirubin

BV is the primary product of haem degradation, which is then reduced by biliverdin reductase (BVR) into BR.[14] Once BR is formed it would interact with free oxygen radicals, and oxidized back to BV. This reduction and oxidation process will repeat itself by BVR and free oxygen radicals.[14] By the BR –BV redox cycle, physiological low BR concentrations can exhibit a potent antioxidant protection. Thus, BV and BR share similarities in the antioxidant and antiinflammatory properties.[20] Although BR was considered as a cytotoxic waste product of haem degradation for a long time, it is currently recognized as an endogenous cytoprotective and neuroprotective compound at physiological concentrations.[16]

Meanwhile, BR has been characterized as a potent antioxidant that prevents the oxidative damage triggered by a wide range of oxidant-related stimuli.[21] When plasma protein binding approaches saturation, free unconjugated BR may become neurotoxic. In particular, BR alters neuronal cell functions and causes cell death by mechanisms that may involve synaptic dysfunction, oxidative stress, impairment of mitochondrial oxidative phosphorylation, and apoptosis.[22]

Ferrous iron

OH-1 catalyzes the oxidative degradation of haem to liberate the central Fe^{2+} iron, CO, and BV. The ferrous Fe^{2+} iron has been considered to be free labile and metabolically active. It can catalyze the production of free radicals and thus act as a cytotoxic prooxidant.[23] At physiological pH, ferrous Fe^{2+} iron is rapidly oxidized to the insoluble ferric (Fe^{3+}) form. Transferrin (a blood-plasma glycoprotein), with a high affinity to ferric iron, plays a central role in iron metabolism and is responsible for ferric-ion delivery. Free haem and ferrous iron are strong prooxidant catalysts with the ability to promote oxidative stress and lipid peroxidation. Accordingly, there is little free iron in the circulation.[24]

Carbon monoxide

CO, once regarded as a metabolic waste, can act as an endogenous mediator of cellular signalling and vascular function.[15,25]

The main source of endogenous CO is derived from haem catabolism. Interleukin-10 (IL-10) is a vital antiinflammatory cytokine which inhibits the production of inflammatory cytokines (such as IL-1β, IL-6, IL-8 and TNF-α). Endogenous CO induces IL-10 expression in macrophages and *in vivo* through an HO-1-dependent pathway causing downregulation of many proinflammatory cytokines.[26] CO is known to have antiinflammatory, antiapoptotic and antiproliferative effects.[25] Recent evidence has shown that CO may also play a role in modulating neuropathic pain by increasing IL-10.[27]

Nitric oxide drives skin repair

Nitric oxide, also called nitrogen monoxide or simply NO, is generated from the amino acid L-arginine by the enzyme nitric oxide synthase (NOS). There are three NOS isoforms: Neuronal NOS (nNOS or NOS-1), cytokine-inducible NOS (iNOS or NOS-2) and endothelial NOS (eNOS or NOS-3). NO used to be considered as a molecule relevant to air pollutant. However, NO is now recognized as a potent endogenous vasodilator and plays a role in neurotransmission, platelet aggregation, innate immunity and inflammation. NO has an extremely short half-life ($t½$ <4 s), which makes it difficult to store. NO is produced as needed whereupon it promptly diffuses through the membrane of target cells, bypassing conventional neural receptors.[5] Under normal physiological circumstances, NO gives an antiinflammatory effect and drives skin repair.[28] Paradoxically, NO is considered as a proinflammatory mediator that induces inflammation due to overproduction in abnormal situations.[28]

Low and constant NO production in the skin seems to be responsible for the maintenance of barrier function and the regulation of blood flow rate in the microvasculature.[29] Higher levels of NOS activity, stimulated by skin wounding or ultraviolet light, initiate a cascade of reactions that require various cell types in a variety of coordination of responses. The enhanced NOS activity in skin wounds appears to be crucial in facilitating leukocyte infiltration and inflammation. In response to injury, activation of the constitutive NOS isoforms (NOS-1 and NOS-3) proceeds and overlaps with the expression of inducible NOS isoform (NOS-2). Thus, at a macrolevel due to injuries, at least three different rates of NO production can occur in the skin.[29]

Potential Mechanisms Induced by Scraping Therapy

Gua sha is often used to treat ailments that cause chronic pain and relieve symptoms of stress. Various mechanisms have been proposed to explain the observed efficacy of scraping therapy. Evidence regarding the possible mechanisms of action following scraping therapy are summarized as follows.

Dampening of pain-promoting substances

Certain inflammatory cytokines (such as IL-1β, IL-6 and TNF-α) involve in the generation of inflammatory pain, by directly activating nociceptive sensory neurons or acting on several signalLing pathways.[30] During the period of scraping marks (petechiae) resolution via haemolysis, endogenous CO derived from haem degradation may induce IL-10 (an antiinflammatory cytokine) expression in macrophages and cause a general downregulation of proinflammatory cytokine production, resulting in antiinflammatory[26,31] and antinociceptive effects.[26,32]

An animal study involving rats with lumbar disc herniation induced by autologous nucleus pulposus has shown that the pain-relieving effects and the mechanism of Gua Sha might be related to the attenuation of inflammatory cytokines (IL-1β, IL-6 and TNF-α in a time-dependent manner).[33] The authors also noted that the effect of antinociception by Gua Sha was more intense in the second treatment course. After three treatment courses, the levels of inflammatory cytokines decreased dramatically. Therefore, the authors deduced that the reduction of inflammatory cytokines following scrapping had a key influence on antinociception.[33]

Inhibition of neuronal responses

Microglial cells are known as resident macrophages in the central nervous system (CNS, including the spinal cord), which rapidly respond to a wide range of stimuli that threaten physiological homeostasis.[34] Nociceptive signals from peripheral nerves lead to a dramatic activation of microglia in the spinal dorsal horn. Furthermore, activated microglia cells are observed mainly in the medial side of the dorsal horn, an area that has been shown to receive inputs from these nerves.[34] Antinociception and neuroprotection are predominately mediated by an autocrine role of antiinflammatory cytokines (IL-10) in the spinal microglia, which upregulate β-endorphin expression in the CNS.[35] HO-1 is among the most critical cytoprotective mechanisms that are activated during times of injury and stress.

Nascimento and Branco conducted a rat model to determine synergism between peripheral (hind paw) and spinal HO–CO pathways of nociception. Typically, 1% formalin was injected into the skin of the hind paw to cause pain-related flinching. The HO substrate (haem-lysinate) was injected intrathecally prior to the formalin test and produced a dose-related antinociception (flinching nociceptive response). The hind paw injections of haem-lysinate also resulted in an antinociceptive effect. These findings support an antinociceptive synergy exists between the peripheral and spinal HO–CO pathways.[36] The alleviation of pain-related flinching behaviours in rodent model could be due to the inhibition of inflammatory responses that are linked to the microglia (resident immune cells) activation in the spinal cord.[36] Gua Sha has beneficial short-term effects on pain. The upregulation of OH-1 is thought to represent an antioxidative response to circulating haemoglobin products following the Gua Sha procedure.[37] Activation of the HO pathway at the level of the spinal cord can modulate nociception originating in peripheral tissues,[36] as mentioned above.

The antinociceptive effects of nitric oxide

Tissue injury (scraping) results in the release of inflammatory mediators from damaged cells including nitric oxide (NO), histamine, ions, etc., Recruited immune cells release further mediators including cytokines and growth factors.[38] Among the cytokines, IL-10 is an antiinflammatory cytokine that suppresses nitric oxide synthases (NOS1 and NOS2) activity. The suppression of NO production leads to reduction in inflammation-related nociception.[39,40] The role of NO plays in pain is not simple. It may show pro- or antinociceptive effects depending on the circumstances.[28,41]

Actually, NO offers pain relief in a number of ways.[42] Inflammation causes swelling or oedema and exerts pressure on the nerves. Often the compromised circulation to the nerves is first perceived as pain. NO-mediated vasodilation will increase the delivery of oxygen and nutrients to poorly perfused nerves to reestablish a normal membrane potential (the firing threshold).[42] Given the similarities between the chemical structures and biological actions of CO and NO, both gases are involved in the modulation of blood vessel function, including vasodilation and inhibition of platelet aggregation.[16] Moreover, NO may reduce pain by increasing cyclic guanosine monophosphate levels, the mechanism by which opioids work.[42] Experimental data in mouse models have also demonstrated that NO inhibits nociception in the peripheral and also the central nociceptive system.[43]

The modulation of pain by counterirritation

A counterirritation is an artificial irritation of the skin at certain body part in order to relieve a morbid irritation (pain) elsewhere. Counterirritation may be explained by the gate control theory.[44] Mechanoreceptors are sensory receptors that are activated by mechanical pressure or distortion rather than noxious stimuli. Both mechanical (myelinated) and nociceptive (nonmyelinated) signals enter the same neurons in the dorsal horn of the spinal cord. However, since mechanoreceptors have lower activation threshold, they generate high-rate action potentials.[44] When the mechanoreceptor pathway is activated, it causes interneurons to inhibit nearby nociceptive axons, suppressing the painful afferent from ascending the spinal cord.[45] Most people have experienced the gate-control phenomenon in daily life by rubbing (soothing) a painful spot following an injury.[44] Many therapeutic ointments containing cooling agents and mild caustic substances used for treating muscle and joint pain also adopt the property of counterirritation.

Scientific literature supported that intense manipulation of naturopathic approaches (i.e. Gua Sha, massage or cupping technique) can activate mechanoreceptors to dampen or modulate central (spinal) nociception via the gate-control pathway. Antinociceptive effects of counterirritation on pain perception and spinal nociception have been confirmed by experimental models on functional magnetic resonance imaging. Attention plays an important part in the pain relief experienced from counterstimulation.[46,47]

The placebo effects

The placebo effect is an achievement of some therapeutic effect after getting a sham procedure (placebo treatment). It has long been known that a placebo can provide the perception of pain relief simply due to a profound expectation of patients. Recent reviews suggest that placebo interventions can improve physical disease processes of peripheral organs more easily and effectively than biochemical processes.[48] Now science has found that under some circumstances, these placebos can be as effective as real medical treatments. Expectation can also influence treatment outcomes.[49]

Neurophysiologically, a placebo effect might be attributed to another pain-gating mechanism of the CNS. The midbrain periaqueductal grey matter (PAG) is an anatomic checkpoint that receives inputs from the frontal cortex and hypothalamus and sends signals through descending projections to the dorsal horn of the spinal cord.[45] Endorphins (endogenous opioid neuropeptides) are produced as a response to certain stimuli, especially stress, fear or pain. The hypothalamus responds to pain signals by releasing β-endorphin through the PAG network, which in turns sends its output on the descending inhibitory pathway and produces pain-inhibiting effects peripherally. In this way, profound analgesia is produced.[45] Through this pathway, strong emotion, stress or even high expectation can blunt the pain perception.[43] A recent study utilizing C-11 methyliodide with positron emission tomography has shown that a placebo administration (an intravenous introduction of 1 mL of 0.9% physiological saline) can trigger the endogenous analgesic activity through the site-specific activation of the μ-opioid receptors of the brain.[50]

Summary and Conclusion

This narrative review draws up a survey of scientific sources on an ancient healing, scraping therapy. It is hypothesized that the skin, the nervous system and immune system interact with one another to generate a cascade of physiological responses to the scraping, through which scraping may result in therapeutic benefits. Within the scope and limitations of this review, only a brief overview could be given of the potential relationship between the observed outcomes and scraping therapy. Implementing effective traditional healings within health systems will require appropriate knowledge translations and future prospective studies.

Key points

1. The observed therapeutic effects following scraping therapy may be a physiological response to the minor bruising.
2. Scraping is assumed to be a mechanical signal to elicit the immune function of the skin.
3. Through natural resolving of the scraping marks (petechiae) a cascade of physiological responses are generated.
4. Counterirritation and placebo effect can also contribute to positive effects for symptom relief.

Ethical clearance

This narrative review using publicly accessible documents as evidence did not require ethical approval. This research did not need informed consent as human subjects were not involved. References and quotations were written as per the journal guidelines.

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Conflicts of interest

There are no conflicts of interest.

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