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Recontextualizing momentum transfer in shock wave therapy – insights into neurostimulation and beyond: correspondence

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Dear Editor,

The recent article authored by Wess and Mayer[1] piqued our considerable interest. They argue that the force generated by the change in momentum (momentum transfer) at tissue interfaces with varying acoustic impedance explains how shock waves could be used for the stimulation of nerve cells, healing of soft tissues (e.g., the brain and muscles), and fragmentation of hard structures (e.g., kidney stones)[1]. In soft tissues, small interfacial forces of ≤10 Newton (N) generated by the change in momentum (momentum transfer) stretch cell membranes, trigger mechanotransduction and mechanosensory pathways, leading to the reorganization of pathological memory patterns in the brain. Alternatively, at hard tissue interfaces, such as in body stones (e.g., kidney or gall bladder stones), much larger forces (≥200 N) cause mechanical fragmentation. The authors extended this principle to propose speculative applications in neurostimulation, suggesting that shock waves could reactivate neural circuits in neurodegenerative diseases such as Alzheimer's disease by mechanically exciting nerve cells or even reactivating neural circuits in the brain. Their framework positions momentum transfer as the core physical driver of the diverse clinical effects of shock wave therapy, bridging lithotripsy, soft tissue repair, and emerging neurological applications including chronic pain relief. Although conceptually innovative, the hypothesis lacks robust experimental validation and overlooks critical biological and physical complexities. This commentary integrates recent evidence to refine the framework and highlight key gaps. The work has been reported in line with the TITAN criteria, and cite in your references[2].

First, the authors extend the momentum transfer paradigm to neurostimulation, suggesting that shock waves can reactivate neural circuits in neurodegenerative diseases via transcranial pulse stimulation (TPS). However, a recent systematic review of TPS in Alzheimer's patients found only mild cognitive improvements in uncontrolled trials, emphasizing the lack of sham controls and small sample sizes[3]. Although functional MRI revealed increased hippocampal connectivity after transcranial pulse stimulation, these changes were not validated through larger randomized controlled trials[3,4]. In addition to the randomized controlled study design and objective neurophysiological measures (e.g., EEG, fMRI), we emphasize the significance of incorporating other experimental designs or conditions, such as age-based cohort studies (e.g., patients ≤70 years of age vs. >70 years), studies evaluating inflammatory biomarkers (e.g., TNF-α, IL-6), cross-arm comparisons (e.g., verum TPS, sham TPS, and other established interventions such as low-level vagus nerve stimulation), and longitudinal follow-up studies (e.g., 6–12 months)[5,6]. Without robust evidence that momentum directly modulates synaptic plasticity rather than nonspecific inflammation or placebo effects, neurostimulation claims remain conjectural.

Second, while Wess and Mayer speculated that shock waves could reactivate neural circuits and support nerve regeneration, recent research offers tangible evidence for this potential. Recently, Ham *et al*, developed a focused shockwave therapy to stimulate deep brain regions without surgery[7]. Unlike traditional treatments that require the insertion of electrodes, this new approach delivers highly localized mechanical signals to areas such as the hypothalamus and hippocampus, which are critical for neurogenesis, memory and cognitive function, without harming the surrounding tissues. Notably, this method allows region-specific mechanical stimulation, directly supporting the idea that targeted physical forces can drive neural repair and functional recovery. This aspect, however, remains overlooked in the authors' discussion.[1].

Third, the authors briefly discussed mechanotransduction of membrane stretching, triggering VEGF/NO release. Other pivotal mechanisms involved in inflammatory signaling and healing have not been discussed. Shock waves activate intricate signaling cascades involving PI3K/Akt, MAPK, and Wnt/β-catenin pathways, which regulate cell proliferation and inflammation[4,8]. In addition, low-intensity shock waves polarize macrophages

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toward an anti-inflammatory phenotype, accelerating tissue repair[9]. This omission risks overselling momentum transfer as a primary mechanism, ignoring synergistic biochemical feedback loops.

Fourth, cavitation, which is the collapse of microbubbles during shockwave propagation, is a well-documented contributor to tissue effects. Cavitation generates secondary shock waves and microjets[10], which enhance drug delivery across the blood-brain barrier and excite nerves via bubble-induced action potentials[7,11]. A recent review on extracorporeal shock wave therapy (ESWT) in neurological diseases explicitly links cavitation to axonal regeneration, contradicting the authors' momentum-centric narrative[7]. The authors should have acknowledged the potential roles of these other mechanisms and discussed how they could be distinguished experimentally from the effects of momentum transfer. A balanced comparison of these differing perspectives would provide a more comprehensive understanding of the complex actions of shock wave therapy (SWT). In this line, Regen *et al* developed a Monte Carlo-based detector model to simulate laser speckle imaging, enabling the non-invasive, real-time measurement of momentum transfer in various tissues, including the brain[12]. This approach can be reinforced by controlling cavitation experimentally, for example through the use of high-viscosity polyvinyl alcohol (PVA) medium that restrict gas bubble development.

Based on this approach, Schelling *et al* demonstrated that extracorporeal shock waves stimulate sciatic nerves via a cavitation-mediated mechanism[13]. In addition, Goller *et al* showed that cavitation can be visualized in brain surrogate models using high-speed videography (10,000 fps), where the cavitation behavior varied depending on the physical properties of the medium (e.g., transparent polycarbonate filled with degassed water vs. Sylgard silicone gel)[14]. Together, these experimental approaches offer a robust framework for differentiating momentum-driven effects from those primarily mediated by cavitation or downstream biochemical signaling.

Fifth, the authors considered momentum transfer as the sole driver of healing, neglecting the adaptive responses of the body. Shock waves are known to provoke an orchestrated healing response in vivo, including the modulation of inflammation and immunity. For example, low-intensity shock waves can shift macrophages toward a regenerative and anti-inflammatory phenotype in injured tissues[15]. It is essential to recognize that patients are not passive recipients of physical forces; their biological resilience (e.g., antioxidant responses, stem cell recruitment) determines their outcomes. A holistic model integrates physics and biology.

Finally, the authors predominantly cited internal studies using proprietary devices, perhaps because of their affiliation with Storz Medical AG. Independent research by other authors on ESWT's effects on nerve conduction velocity reported mixed results: while motor latency improved, sensory outcomes showed no significant changes[4]. The study relies heavily on the authors' own prior studies and device-derived data (for example, internal measurements of shock forces on model stones)[16], with relatively little reference to independent research that might offer different interpretations. This predominant reliance on proprietary studies limits a holistic view and may skew the broader implications of shock wave effects. In this context, Zhu *et al* demonstrated that shear stress waves and cavitation are the key mechanisms of kidney stone fragmentation in shock-wave lithotripsy (SWL)[17]. Indeed, the same mechanisms underlie SWL kidney injury[18]. However, another study by Alkhamaali *et al* reported that cavitation-mediated tissue repair and the healing process play a key role in shock wave therapy for treating chronic plantar fasciitis, plausibly through an initial phase of stress-induced remodeling, followed by tissue repair[19]. In this line, a fairly recent systematic review on the effects of SW underscores that the effects and mechanisms of shock wave-mediated tissue injury or healing vary widely according to the tissue type, and that the clinical translation of these effects is highly challenging[20].

The momentum transfer hypothesis offers a valuable lens for understanding shock wave therapy, but requires refinement in terms of empirical validation, integration of multifactorial mechanisms, and clinical translation. Future studies should prioritize interdisciplinary collaboration to disentangle the role of momentum transfer from confounding physical and biological variables.

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A.M.: conceptualization, data curation, writing – original draft preparation, supervision; C.O.E.: conceptualization, writing – original draft preparation; M.M.: data curation, writing – original draft preparation; writing – reviewing & editing; T.B.E.: validation, writing – original draft preparation, writing – reviewing & editing. All the authors read and approved the final manuscript.

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