Intermittent Pneumatic Compression as a Regulator of Physiological Processes: Problems, Hypotheses, and Research Directions

(A Conceptual Essay)

Dmytro Zaitsev

Department of Occupational Medicine, Psychophysiology, and Medical Ecology,

Shupyk National Healthcare University of Ukraine, Kyiv, Ukraine.

E-mail: suhanovik@gmail.com

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Abstract

This text offers a personal perspective on the future development of intermittent pneumatic compression (IPC) therapy. It is based not on a systematic literature review but on clinical practice, previous research, and conceptual synthesis.

IPC is presented as a regulatory intervention. Considering its reflexive effects and its influence on cerebral blood flow, IPC is discussed as both a neurotropic and neuromodulatory method. A separate hypothesis is proposed that IPC may serve as an endocrine-like tool — a kind of extracorporeal artificial "gland" that modulates humoral transport and indirectly stimulates the synthesis of regulatory substances.

The paper also attempts to outline the basic parameters that define an IPC procedure. It raises the problem of constructing an "alphabet" of IPC: a structured correspondence between parameters (and their values) and the resulting physiological effects at various biological levels.

In addition, around twenty possible directions for future IPC research are suggested, including applications in immunology, infectious disease, preventive and anti-aging medicine, and neuroscience.

“*What could be new in physiotherapy?*”

— Corresponding Member of the NAMS of Ukraine,

private conversation, ca. 2010

Introduction

What are the origins of this essay, and on what foundation does it stand?

The development of intermittent pneumatic compression (IPC) in Ukraine was initiated by Ihor Tarshynov between 1989 and 1992. His research team remained active until the COVID-19 pandemic. For many years, our communication with international colleagues was limited, and even with the emergence of the internet, this barrier persisted — partly because there were no consistent keywords in this field to guide information searches.

Until around 2012–2014, Ukrainian scientific thought in the area of IPC evolved largely independently, outside the influence of global research trends. As a result, some internationally accepted concepts remained unfamiliar within Ukraine, while certain domestic innovations did not gain global visibility.

Tarshynov’s group was twice recognized by the World Intellectual Property Organization: in 2003, for developing one of the first portable IPC devices for personal use, and in 2006, for introducing IPC in the treatment of asthenic syndrome in patients with erectile dysfunction.

From this original clinical practice and empirical observation, Tarshynov and his collaborators developed their own theoretical framework, which opened new directions for IPC use — not only in diabetes (since 1995) or atherosclerosis (since 2000), but also in neuropsychiatry (since 2006), ophthalmology (2009), and psychophysiology (2017).

Some practical consequences of this theoretical paradigm are the focus of the present work. What follows is a discussion of the unknown, and an attempt to outline what might be possible.

The future of IPC may lie in a world where its clinical effectiveness is no longer demonstrated through individual case reports, but through big data: thousands of procedures, patients, and parameters analyzed in real time, with consideration of genetic, biochemical, behavioral, and environmental factors.

In this future, IPC is not merely a therapeutic tool — it becomes part of a system of regulatory analytics, where algorithms search for response clusters, predictors of efficacy, and optimal parameter settings. At that point, IPC moves beyond classical physiotherapy toward a mathematically guided intervention that requires systems thinking and models grounded in population-level dynamics.

This publication is not a literature review and is not based on a structured analysis of previously published sources. Rather, it is a personal reconstruction of possibilities, drawn from clinical experience, empirical insight, and exploratory modeling using artificial intelligence.

For this work, ChatGPT-4o (OpenAI) was used as a supporting tool for general fact-checking, posing critical questions, and generating feedback. Some segments of the text are presented in dialogue form. Responses generated by the AI are presented as direct quotes.

What Is Intermittent Pneumatic Compression Today?

To begin the discussion, it's important to define a basic shared understanding of what IPC is. For this purpose, I will use the help of an AI-based summarization tool to generate a concise and widely accepted definition, reflecting general knowledge in the field.

ChatGPT-4o: "*Intermittent pneumatic compression (IPC) is a method of external mechanical stimulation applied to soft tissues using air-filled chambers or cuffs that rhythmically inflate and deflate. The goal is to improve venous and lymphatic drainage, prevent thrombosis, and stimulate recovery and metabolic activity. The physiological mechanism is based on the cyclic compression and release that enhances fluid movement through tissues and vessels, reduces stagnation, and supports local metabolic processes. IPC is commonly used in angiology, physical medicine, rehabilitation, sports medicine, neurology, and postoperative care. Its core concept is to simulate the effect of the muscle pump in cases where that function is impaired due to disease or immobilization.*"

At this point, it's helpful to clarify the distinction between what I call the "Tarshynov model" of IPC and the classical version of the method. Ihor Tarshynov himself never defined IPC formally — at the time, he didn't need to. He worked in a country where he effectively founded the practice. He once said:

"It's not about treating the disease. It's about adjusting the organism so it can handle the disease on its own."

Later, I tried to rephrase his idea in my own way:

"We treat one thing by affecting something else."

ChatGPT-4o: “*The Tarshynov model of IPC is a conceptual approach in which the procedure is seen not as a local intervention, but as a systemic regulatory stimulus intended to activate the body’s own adaptive mechanisms. This view brings IPC closer to other forms of systemic modulation, such as hormone therapy, psychotherapy, reflex therapy, or biofeedback.*”

Below is a list of clinical indications for IPC (Table 1). Because IPC includes a wide variety of settings and configurations, any statement about what is or isn’t appropriate should be interpreted within the context of at least conscientious, and ideally evidence-informed, clinical practice.

ChatGPT-4o generated a list of conditions where IPC is considered clearly appropriate and clinically justified. Conditions marked with an asterisk (*) are those where the benefit likely outweighs the risk, although the indication remains subject to discussion.

For comparison, I included a list I originally compiled in 2017 based on the best available data at the time. In that version, an asterisk (*) was used to mark effects that require independent verification or further clarification of their clinical relevance.

The sources I used came from a period when Ukraine's publication ecosystem was still disconnected from the global research infrastructure. Many of these data points were not formalized according to today’s standards but were documented in clinical reports, conference presentations, and institutional publications. They nonetheless influenced the shaping of real-world practice.

To simplify the comparison, conditions for which there was agreement across both lists — i.e., indications where IPC is universally accepted — were excluded from the table.

Table 1. Empirical Experience with the Use of Intermittent Pneumatic Compression

	Compiled by ChatGPT-4o (2025)	**Compiled by Author (2017)**
Deep vein thrombosis (I80.2). Spinal cord injury (S14, S24, S34)	✓	—
Varicose veins during pregnancy (O22)	✓ / ✓*	—
Chronic heart failure (I50). Thromboangiitis obliterans (Buerger’s disease) (I73.1). Pelvic venous congestion syndrome (I86.2). Multiple sclerosis (G35). Rheumatoid vasculitis (M05.2). Systemic lupus erythematosus (M32). Systemic scleroderma (M34). Chronic kidney disease (N18)	✓*	—

Stroke and spastic syndromes (G81–G83)	✓ / ✓*	✓
Atherosclerosis of lower limb arteries (I70.2)	✓ / —	✓
Cerebral palsy (G80). Arterial hypertension (I10–I15). Osteoarthritis (M15–M19)	✓*	✓
Diabetic encephalopathy (E11.9). Post-traumatic stress disorder (F43.1). Neurocirculatory dystonia (F45.3). Asthenic syndrome (F48.0). Sleep disorders (F51). Attention deficit disorder (F90). Epileptic syndromes (G40). Unspecified neuropathies (G62.9). Angiotrophonoeurosis (G90.9). Macular dystrophy (H35.3). Diabetic retinopathy (H36.0). Glaucoma (H40). Myopia (H52.1). Lipodermatosclerosis (I83.1). Cellulitis (L74.9). Trophic ulcer of the lower extremities (L97). Arthritis (M13). Joint contracture (M24.5). Dorsalgia (osteochondrosis, radiculopathies) (M54). Hyperglycemia (R73). History of myocardial infarction (Z86.7)	—	✓
Psychogenic erectile dysfunction (F52.2). Intellectual disability (F70–F79). Childhood autism (F84.0). Childhood enuresis (F98.0). Baroacoustic trauma of the ear (H83.3). Sensorineural hearing loss (H90.3). Chronic cerebrovascular disease (I67.8). Chronic nonspecific pneumonia (J18.9). Chronic sinusitis (J32.0). Meteoropathy syndrome (T75.2). Post-burn skin conditions (T95.0)	—	✓*

Note: Conditions with strong consensus in both lists (where IPC is widely accepted) have been excluded for clarity. Asterisked conditions indicate those requiring further validation or considered clinically debatable but potentially beneficial.

The table presented here does not aim to establish clinical recommendations, as it does not assess the expected efficacy of IPC in each case. Its purpose is to outline an empirical landscape that calls for further systematic investigation. A structured map of indications provides a foundation for future comparisons, hypothesis refinement, and the design of randomized clinical trials.

Of course, any comparison of lists that include both individual symptoms and full diagnoses (sometimes with staging), clusters of related conditions, broad clinical states, and therapeutic contexts is methodologically imperfect. However, even with these limitations, such structuring allows us to estimate the scale and scope of IPC's clinical reach. The methodological flaws do not prevent us from drawing conclusions — at least at a preliminary level.

- A newcomer exploring IPC for the first time might identify 5 to 10 common indications after a few days of searching in PubMed.

- The list generated by ChatGPT-4o includes around 30 conditions — reflecting a gap between the average clinician's expectations about IPC's range and what can be found in the "publicly available library" of common knowledge.
- My own list, filtered from decades of Ukrainian clinical observations and research, contains about 50 conditions. These are interventions that were actually attempted — regardless of whether they were "accepted" or not.
- A handwritten list by Ihor Tarshynov included nearly 90 items, and in conversation he would often say "more than 200" — although this number should probably be taken as figurative rather than literal.

This is our first pause for reflection:

Does IPC have unrealized potential? Without a doubt.

Today, the method is used at perhaps 10 to 20 percent of its actual capacity.

However, this essay is not about the 80 to 90 percent of indications that remain underutilized.

It is about entire domains that have not yet entered the clinical agenda at all.

Intermittent Pneumatic Compression in Neurological and Mental Health Interventions

The nervous system exists to respond to everything that happens within the body. Naturally, it reacts to something as intense as mechanical stimulation — especially pneumatic massage. It hardly needs proving that IPC has neurotropic effects. And yet, when ChatGPT-4o was asked to define IPC, it made no mention of these effects. Moreover, it is reasonable to assume that IPC — when consciously experienced — also produces psychotropic effects.

There is no doubt that IPC can trigger a rapid and significant "release" response: muscular relaxation, thermoregulation (due to hyperemia and localized heat accumulation), and a reduction in perceived tension. The body receives rhythmic, slow tactile input across large surface areas — or, alternatively, targeted stimulation of highly sensitive zones such as the feet, hands, or ears. This makes IPC a multisensory event capable of activating a wide spectrum of neuropsychological mechanisms associated with safety, recovery, and embodied well-being.

Regarding autonomic modulation, it's still unclear whether IPC primarily reduces sympathetic activity while enhancing parasympathetic tone — or whether its effect is more about harmonizing autonomic balance in general, by lowering overactivity in either direction.

Perhaps the former is typical of a single session (a fast-acting effect), while the latter develops over a longer course (a slow-acting effect). This duality complicates intuitive recognition and makes it difficult to study using standard protocols. IPC's ability to modulate circadian rhythms remains an open question.

Several types of psychotropic effects should be considered. IPC may have anxiolytic effects (functioning as a kind of tactile “swing,” similar to deep pressure therapies), antidepressant effects (by imitating bodily movement and interpersonal interaction), and integrative effects on body awareness — helping users reconnect with their physical selves through enhanced proprioception and a sense of groundedness.

The muscle-relaxing impact of IPC, comparable to a warm bath, along with the rapid reduction of swelling, heaviness, or pain, can noticeably influence mood, motivation, and even self-image.

The drowsiness often observed during IPC sessions should be better characterized. It's worth exploring whether this represents true sleepiness or a form of dissociation — rest without sleep. Likewise, IPC should be evaluated for its potential as a quick-recovery method during short work breaks. In our practice, one coach observed that a hypnagogic state during IPC helped his athlete absorb verbal instructions more effectively — almost as if they were imprinted in muscle memory rather than cognitive memory. This suggests a kind of neuroplasticity, where somatic input leads to the formation of new body–brain templates that could persist over time.

In some patients — especially those in creative professions — this hypnagogic state was accompanied by rich mental imagery or spontaneous problem-solving. The parasympathetic dominance triggered by IPC slows down both mental and physical activity. The rhythmic nature of the stimulus may support theta-wave synchronization and activation of the brain's default mode network. On the other hand, the repetitive and predictable sensory input of a long IPC session requires minimal engagement from sensory and prefrontal cortex areas. This “freeing up” of cognitive resources may allow the brain to process signals from the limbic system and visceral organs instead.

Heart rate variability data supports this hypothesis: an increase in the power of very low frequency (VLF) bands is often recorded at the end of the session. The body seems to shift into a deep internal mode — not only of compensation but of reorganization.

This leads us to the concept of neuroplasticity through somatic induction: teaching the brain via modified bodily patterns — a kind of neural orthopedics or reverse biofeedback. IPC imposes a healthy physiological pattern onto the body. The body, in turn, sends corresponding signals to the brain. The brain adapts to this new norm — even if the change is not consciously perceived or voluntarily initiated.

Viewing IPC as a psychophysiological technique opens new research directions in neuroplasticity and mind–body integration.

Intermittent Pneumatic Compression as Hormonal Therapy

Imagine I show you a black box and tell you that inside it is something with the following characteristics:

- It interacts with the endocrine system. It acts as a synergist to hormones and chemical messengers by improving the delivery of bioactive substances to target organs and optimizing local metabolism in peripheral tissues — especially by reducing edema. It also influences endocrine gland activity through feedback mechanisms.
- It plays a role in humoral regulation. It triggers the synthesis of regulatory substances such as nitric oxide, prostacyclin, endothelin-1, adenosine, interleukin-6, insulin-like growth factor 1 (IGF-1), ATP/ADP, lactate, tissue plasminogen activator (tPA), von Willebrand factor, fibrinogen, and vascular endothelial growth factor (VEGF).
- It has regulatory effects on metabolism and certain processes in target organs. It produces coordinated responses such as muscle relaxation, arteriolar dilation, and enhanced vagal tone.

What’s in the box?

With these characteristics, you might expect it to be a hormone — or even a gland. But the description, of course, fits intermittent pneumatic compression.

This leads to a conceptual model in which the IPC device acts as an extracorporeal artificial endocrine gland — an external extension of the endocrine system and a legitimate participant in its regulation.

To speak plainly: from the moment it became clear that IPC stimulates the synthesis of nitric oxide, we were obligated to acknowledge it as a form of non-pharmacological hormonal therapy. The mechanism is not pharmaceutical, but the functional outcome is hormonally mediated. This is not a rebranding, but a shift in perspective. Thinking of IPC as a form of endogenous hormone therapy helps us reposition it within clinical reasoning.

This model becomes easier to grasp when we compare IPC to dialysis machines, ventilators, insulin pumps, or pacemakers — devices that substitute or support physiological functions normally carried out by internal organs.

For the first time, IPC can be described and understood in the language of endocrinology. With some exceptions, IPC does not directly generate biochemical mediators. Instead, it produces effects that either resemble or counter those of classical hormones. It acts as a physiological trigger — typically initiated by hormones in natural conditions. In this framework, IPC effects can be formalized within the structure of the endocrine system, not by mimicking its mechanisms, but by aligning with its outcomes.

We can group these effects according to their hormone-like action — for instance, thyroxine-like effects or anti-angiotensin effects. And if we are to approach IPC as a regulator, the most useful analogy is to see it as an agonist or antagonist to real hormones.

(See Table 2: Known Hormonal Effects of IPC)

Table 2. Model of Known and Potential Hormone-Like Effects of Intermittent Pneumatic Compression

Interaction with Bioactive Substance	Known Effects of IPC	Potential (Hypothetical) Effects of IPC
Synergist of Thyroxine (T4/T3)	Increases tissue sensitivity to thyroid hormones (thermoregulation, metabolism)	May reduce cold intolerance, apathy, and slowness in mild hypothyroidism without medication
Synergist of Insulin	Improves tissue perfusion and insulin sensitivity	May reduce insulin resistance in metabolic syndrome; support weight reduction
Synergist of Nitric Oxide (NO) and Prostaglandins E2/I2	Stimulates vasodilation, anti-aggregation, vascular protection	May lower oxidative stress, enhance microcirculation, and protect endothelium
Antagonist of Catecholamines	Reduces sympathetic tone; stabilizes autonomic reactivity	May prevent stress-induced crises, arrhythmias, anxiety, and migraines
Antagonist of Aldosterone	Osmoregulatory effect; reduces volume overload	May decrease the need for diuretics; mild anti-edema effect
Antagonist of Angiotensin II	Blocks systemic vasoconstriction; improves microcirculation	May provide antihypertensive effect in resistant hypertension; requires

		clinical confirmation
Contextual interaction with Cortisol	Acts as antagonist under stress; synergist in adrenal hypofunction	May normalize stress response without impairing reactivity; requires studies assessing cortisol and ACTH levels
Potential Antagonist of Vasopressin (ADH)	Reduces water retention via alternative fluid elimination pathways	May reduce edema in functional ADH hypersecretion (excluding Schwartz–Bartter syndrome)
Indirect Potentiator of VEGF Activity	Enhances endothelial stimulation under ischemia	May support angiogenesis in chronically ischemic tissues; promising for rehabilitation
Possible Agonist of BDNF and Serotonin (hypothetical)	Promotes neuroplasticity; improves affective state	May aid in cognitive rehabilitation, improve sleep quality, and reduce anxiety

It is precisely the complexity, coordination, and wide spectrum of IPC's physiological effects that allow us to describe it not as a mere collection of stimuli, but as a systemic regulatory intervention. When IPC is combined with pharmacological hormonal therapies, synergies or antagonisms may arise — these interactions should be anticipated during protocol development.

The response to IPC will vary depending on the patient’s endocrine status, even when hardware parameters remain unchanged. The endocrine system functions as a buffering mechanism — under normal conditions, it is capable of compensating for external inputs. The effects of IPC may simultaneously involve:

- Suppression of glandular activity by enhancing the transport of hormones to their target tissues, triggering negative feedback (a peripheral effect, via enhanced uptake);
- And stimulation of glandular output by improving blood flow to the glands (a central effect, via increased production).

This triangular interaction makes IPC's impact on endocrine regulation typically mild and physiologically balanced — except perhaps for substances with high peripheral sensitivity (notably hormones from the endothelium, pancreas, and thyroid). Under conditions of mild, early-stage, or transient endocrine hypofunction, IPC may serve as a supportive or adjunctive therapy, potentially reducing the required dose of corresponding exogenous medications.

Even if we treat this model as metaphorical, it still offers explanatory power. It allows us to:

- Integrate scattered observations of IPC's endocrine effects into a coherent knowledge system;
- Establish new conceptual links between clinical phenomena;
- Anticipate possible outcomes;
- Generate hypotheses for future research.

We may not yet possess the full data, but we already understand the logical structure into which that data will eventually fit.

If we accept that IPC has hormone-like properties in terms of regulatory influence, then we can propose the following line of reasoning:

If IPC mimics the action of a specific hormone, and that hormone produces Effect X under Condition Y, then IPC may be expected to induce a similar Effect X under the same Condition Y, unless the original hormonal effect is strictly dependent on receptor specificity or membrane-level mechanisms.

(See Table 2: Predicted Hormone-Like Effects of IPC.)

Intermittent Pneumatic Compression as Immune Hygiene

There is strong reason to believe that IPC does not affect the immune system as a direct stimulator or suppressor, but rather as a facilitator.

Stress-related suppression of immunity may be alleviated due to reduced sympathetic tone, allowing the immune system to respond more actively — sometimes even excessively, especially in cases where it was previously overinhibited. In such cases, a brief pro-inflammatory activation may occur as part of the rebalancing process.

IPC may also engage neuroimmune mechanisms — for example, by activating the inflammatory reflex through the vagus nerve, as a consequence of enhanced parasympathetic activity. When applied to the abdominal region, IPC may improve gut motility, potentially reducing endotoxinemia, a known risk factor for systemic inflammation.

Mechanically, IPC supports the synthesis of anti-inflammatory mediators such as interleukin-10 and nitric oxide (NO). The combination of edema reduction and enhanced microcirculation can shift the functional conditions for macrophages, dendritic cells, and tissue-resident lymphocytes in chronically inflamed areas.

Theoretically, IPC may serve an immunotropic role as a suprasystemic modulator. It may help suppress excessive immune responses (e.g., in autoimmune or para-inflammatory conditions), while also supporting secondary immunity by reducing systemic inflammation — essentially unburdening the immune system from the load of edema, hypoxia, and sympatho-stress.

Potential Directions for Further Clinical Exploration

When teaching physicians, I often recommended that even localized IPC procedures be viewed as having systemic effects. If IPC influences circulation and autonomic regulation, then it should primarily be understood as a regulatory intervention. What began as an empirical observation has now become a theoretical principle — and perhaps explains how Ihor Tarshynov once came up with his famous “200 indications”. Of course, IPC is not a miracle cure — or perhaps more precisely, it is a panacea with very real limitations (which we will address separately). But these broad horizons reflect how Tarshynov envisioned the purpose and place of IPC in medicine. It may even be appropriate to use it prophylactically in clinically healthy individuals.

§1–3. As an adjunctive and background therapy, IPC may help ease the course of some infectious diseases (when used alongside adequate primary treatment). To clarify its role, at least two foundational studies are needed:

- Does IPC influence lymphatic or hematogenous dissemination of pathogens? If so, under what conditions and to what extent? More broadly, does IPC affect the dynamics of toxin clearance or the pace of immune response?
- A parallel line of investigation — high risk, but potentially groundbreaking: could IPC affect the production or breakdown of pro-inflammatory cytokines? Might it dampen or amplify the cytokine response during septic states?
- The so-called “detoxification effect” of IPC — fact or myth? If real, at what stages does it act: local toxin generation and containment; systemic spread and immune reaction; storage and biotransformation (e.g., hepatic load reduction); final elimination?

These lines of inquiry could guide future studies not only in infectious disease, but also in allergology and critical care medicine.

§4. By improving local microcirculation and overall tissue perfusion, IPC may accelerate drug absorption, especially for injected substances, and influence volume of distribution, particularly for hydrophilic agents. IPC could alter drug distribution between peripheral and central compartments, especially for drugs with low plasma protein binding. If IPC is found to affect portal circulation, it may also influence the first-pass metabolism of certain drugs. Enhanced lymphatic drainage could impact the kinetics of lipophilic compounds or nanomedicines. IPC might also accelerate elimination via renal clearance or sweating. Clinically relevant topics may include: delivery of medications to poorly vascularized tissues; duration of action for hormone-based treatments; adjustments to drug dosing based on tissue saturation; changes in peak effect and half-life duration.

§5–6. Using IPC as a targeted therapy in oncology patients raises complex ethical issues — each case requires individual clinical judgment. Risk must be assessed not by diagnosis alone, but by how the tumor might behave under changes in microcirculation, hydrodynamics, or immune activity. Promotion of micrometastases or reactivation of dormant lesions remains a theoretical concern. Although there is no empirical evidence that IPC triggers such complications, caution is warranted due to the vascular sensitivity of some tumors. IPC might be appropriate in palliative care, post-treatment rehabilitation, or as an adjunct during pharmacotherapy (particularly for hydrophilic or angiotropic drugs).

We often think of IPC only after disease has occurred, underestimating the preclinical and subclinical phases. Risk factor modification may become a new domain for IPC — especially if it enhances local immune responses through stimulation of lymphatic flow and humoral regulation. It may also, in theory, reduce the risk of oncogenic transformation in tissues affected by hypoxia or chronic inflammation.

The broader question of IPC as a preventive tool (not only for oncology, but also for seasonal viral infections and age-related decline) reflects classic challenges in preventive medicine: the need for long-term, large-scale trials and the economic dilemma of treating some vs. protecting all. One possible solution: establish a registry of clinically healthy IPC users — athletes, office workers, transportation personnel — possibly integrated with a mobile app to collect physiologic data in real time.

§7–8. Further research should focus on timing of therapeutic effects — to better predict treatment outcomes and inform patients not just about expected benefits, but about the minimum time required for those benefits to emerge. Conversely, if the length of the planned course is fixed, we should be able to estimate likely outcomes. Understanding IPC's temporal dynamics would bring it into the domain of clinical planning. The same applies to duration of remission — knowing when to schedule repeat treatments in advance. This could form the foundation for optimizing long-term care and support the case for continued follow-up.

§9–10. Myths and realities in cosmetology. There is a need for thorough dermatological research into the effects of IPC on skin condition. Anecdotal and follow-up observations have suggested improvements in regeneration, barrier function, and microcirculatory normalization. Some have even reported hair regrowth, likely due to overall physiological enhancement.

Several questions remain open:

- Can IPC prevent or reverse varicose transformation of veins?
- Does it help recovery after physical exertion — and if so, under what conditions? Can it improve athletic performance?

The absence of evidence is not proof of absence. It is possible that the current lack of observed benefits is due to limitations in study design or insufficient sample sizes. Alternatively, elite athletes may have already maxed out their circulatory and regulatory systems, leaving little reserve for IPC to activate. This brings us into the domain of "borderline physiology" — semi-pathological or overtrained states where a regulatory stimulus might no longer be interpreted as helpful.

Here, a paradox emerges: "panacea poisoning." If the system is already operating at full capacity, it may become unresponsive to external stimuli that push in the same direction. In cases of maladaptive homeostasis, IPC may no longer serve as a signal for normalization — the body might interpret it as interference and trigger counter-regulation. Furthermore, the system may be unable to process new physiological input if it's already overwhelmed by competing or higher-priority signals.

In borderline states of reduced reactivity, IPC may neither help nor harm — it becomes neutral. Still, it might regain effectiveness if the body's adaptive mechanisms recover. In such cases, IPC could be integrated into supportive therapy, provided there is individual clinical assessment. The safety of IPC during phases of reactive exhaustion requires focused investigation in appropriate patient groups.

§11. Precise quantification of ATP and other indicators of baseline metabolism during IPC could serve as a "micro-brick" in explaining its broader effects. If confirmed, such findings would support IPC as a method of core metabolic modulation.

§12. More attention is needed on what happens after IPC — not just during the session or treatment course. Many effects that currently appear diffuse or non-specific may in fact be delayed, complex, and underrecognized aftereffects. This may explain why IPC sometimes works in situations where it "shouldn't."

§13. Anti-inflammatory and Immunomodulatory Effects. What exactly do these effects consist of, and what are their causes? Which components of the immune response are most affected — humoral, cellular, innate, or adaptive? Which markers are most sensitive, both quantitatively and qualitatively?

Key research questions include: Are there changes in inflammatory mediators like IL-6 and IL-10 after IPC? Can IPC reduce TNF-α levels in systemic inflammatory conditions? Is IPC simply a non-specific stimulator, or does it act as a harmonizer of immune function? Can it influence lymphocyte, macrophage, or neutrophil activity? Does it alter the function or distribution of B and T cells? Are CRP and other inflammation markers reduced after a course of IPC?

§14. Further EEG and fMRI studies are needed to investigate the impact of IPC on CNS function, neuroplasticity, central sensitization, and viscerosomatic interactions.

§15. There are already data supporting the role of IPC in alleviating gestosis symptoms. This suggests that its application in managing pregnancy-related pathologies deserves deeper clinical exploration.

§16–18. Ideas from I. Tarshynov on future directions:

- IPC and stem cells.
- IPC and aging.
- IPC as a functional test, i.e., its potential diagnostic value. For example, a lack of response to IPC in athletes might indicate "full exertion" and depleted adaptive reserves. In contrast, heightened responsiveness during periods of adaptation might help reveal hidden chronic or pre-pathological conditions.

Ideas developed in collaboration with ChatGPT-4o:

§19. IPC During Active Movement. In traditional IPC settings, the patient is expected to remain still. Most devices are not built for changes in body position or abrupt muscle tension during sessions. But perhaps we're limiting the method unnecessarily. New research could explore IPC during active motion: compression-assisted exercise, neurophysiological reconditioning, and movement-enhanced drainage. Potential applications may include muscle rigidity, spasticity, myofascial pain, venous stasis in limbs, post-op rehab, and athletic recovery.

§20. Another direction is dynamic mapping of tissue stress, shearing, and pressure gradients during IPC sessions. Using elastography, we could visualize tissue-layer displacement and evaluate the depth of mechanical effect in real time.

§21. Finally, is there a link between IPC and appetite modulation? Could IPC influence leptin or ghrelin pathways?

The overconcentration of IPC research on deep vein thrombosis prevention has created a misleading impression of its narrow scope. But if we were starting this work today, I believe we could already say: we're sitting on 12–15 years' worth of discoveries waiting to happen.

Limitations

We can do some things slightly faster and slightly more purposefully than the body can do on its own — and without paying too high a physiological "price". But what exactly can't we do?

By definition, we cannot influence the direction of non-specific effects. These are outcomes clearly caused by the IPC procedure but independent of its specific settings.

We cannot do anything with an organ that no longer exists. Perhaps we can support regeneration or tissue remodeling, but disrupted anatomy casts a shadow even when the light is switched on.

We cannot help when the body has taken a course toward self-destruction. This is pathological homeostasis — a reorganized regulation that we merely stimulate rather than correct. For example, in an acute autoimmune reaction, we are likely to accelerate the self-damage much sooner than we facilitate resolution.

The core limitation — or rather, the intrinsic weakness — of IPC is its relatively slow action. When timing is measured in minutes or hours, IPC (except perhaps for antishock suits) is not suitable. We accelerate biological time — but not infinitely. I tried to estimate how far this acceleration can go. Tentatively, I assumed it to be comparable to that of moderate physical exercise. Still, it would be useful to quantify the boundary — a coefficient of acceleration for biological processes under IPC.

ChatGPT-4o: "*A hypothetical coefficient of biological time acceleration K under IPC might be around 1.5–3 for vascular effects (drainage, perfusion); K ≈ 2–4 for recovery from fatigue and functional blockages; K ≈ 1.1–1.5 for systemic metabolism and hormonal reactions. K for the immune and nervous systems would depend on the specific task.*"

The impact of IPC works on the scale of the organism. Mechanical energy enters the body like a ripple in water — the further the wave travels, the fainter it becomes. The strongest effects appear at the level of organs, systems, and the whole organism. On levels below (cells, molecules) or above (social environment, population), the effects are weaker. To find the true effect of IPC, we must look at it on the scale of systems — and not expect molecular precision or a psychosocial breakthrough, even though either might occur as an echo.

The Problem of the "Alphabet" of Intermittent Pneumatic Compression

One of the most promising yet underdeveloped areas in intermittent pneumatic compression research is what I call the "alphabet" of IPC. By this, I mean a structured map of all parameters that influence the clinical outcomes of an IPC procedure — as well as the expected biological responses associated with specific parameter values across different levels of physiological organization. A "parameter" here refers to any feature of IPC that can be expressed in measurable units, unambiguous qualitative descriptors, or binary "yes/no" properties — and which can be practically used to distinguish devices or techniques.

This is a systemic gap in the development of IPC. Today, the idea of an "alphabet" remains a working concept on the desks of a few dozen researchers worldwide. No attempt so far has resulted in a public, broadly accepted, or even conditionally complete document. The reason is simple: such a task demands an unusually high level of interdisciplinary coordination. It creates a paradox — we develop treatment protocols without a fully defined coordinate system. Like writing a driving manual without being entirely sure how many wheels the vehicle has.

Over the last 15 years, various authors have raised this issue. And yes, it may seem that significant progress has already been made. For example, we now have detailed insights into how lymphatic outflow responds to different pressure levels — particularly thanks to some outstanding studies from the Polish school of IPC research. In fact, it is already feasible to build a basic matrix of known effects, listing procedural parameters, their values, and associated responses on different biological levels or treatment phases. This data can be collected from published literature, especially from studies comparing two or more IPC methods. Later, it could be aggregated as a meta-analysis.

However, a practical attempt to build such a matrix (Table 3) quickly reveals a serious limitation. Even without numerical values or references, each cell contains complex, multidimensional data that resists standardization. The resulting table would be massive and nearly impossible to publish in a traditional format. Each cell needs an explanation the size of a small chapter. In reality, this “table” is not a table at all, but a reference system — an encyclopedic structure, or a multi-layered database.

In this context, the idea of an “alphabet” is both a prerequisite for IPC standardization and a research challenge in itself. It deserves recognition as a standalone scientific agenda. I hope that this conceptual outline, presented within the boundaries of this essay, may serve as an initial stimulus for a deeper and more systematic exploration of IPC parameters in future work.

Table 3. Intermittent Pneumatic Compression Parameters And Biological Targets

Target	Pressure	Duration	Direction of flow
Interstitial fluid		Local compression duration: 2–4 s. Minimum total session duration: 6 minutes.	From periphery to heart. Reverse flow is acceptable only on the trunk or in limbs with lymphatic congestion. Alternating flow — only briefly and at low pressure.
Veins	50–70 mmHg	Local compression duration: 1–2 s. Minimum session duration: 6–12 minutes.	Same as for interstitial fluid.
Lymphatic vessels	70–100 mmHg	Local compression duration: 1–6 s. Minimum session duration: 18–24 minutes.	Same as for interstitial fluid.
Arteries	Same as for veins	Local compression duration: ~1 s. Minimum session duration: 12–18 minutes.	Alternating flow. Flow from heart to periphery is possible in severe ischemia, but only locally and briefly. Flow toward the heart on the legs has a mild effect but requires a longer course.
Autonomic	Same as for	Session duration up to 6 minutes	

balance	veins for cutaneous receptors, 70–90 mmHg for proprioceptors.	activates sympathetic tone; over 12 minutes — activates parasympathetic tone.	
Arterial pressure			For lowering BP: direction from head to back, from heart to periphery. For increasing BP: the opposite direction.
Muscles	Up to 60 mmHg	Local compression duration: 2–3 s. Total session duration up to 6 minutes increases muscle tone; over 12 minutes causes muscle relaxation.	

At present, we do not know which parameters of IPC are clinically significant. Pressure is always mentioned. Duration of inflation and pause is often discussed. Total session time is sometimes considered. But the direction of the pneumatic wave or its frequency are rarely mentioned. That gives us six commonly discussed parameters — while theoretically, there could be up to twenty.

We still don't know which of them truly matter, or which are therapeutically indifferent. We don't know how precisely they should be measured, or how to quantify subjective sensations.

Here is a possible list of IPC parameters that might have clinical relevance.

Topographical Parameters:

- Anatomical location: visible body parts, with attention to lateralization and segmental zoning.
- Projection-based location: areas that correspond to internal organs or pathological processes.
- Target location: preparatory, primary, compensatory, or indirect zones of action.
- Area of influence. Since "area ≠ significance," it might be helpful to propose a weighted scale (e.g., based on number of receptors, volume, or vascular accessibility) with a weighted unit like $cm^2 \times$ sensitivity coefficient. The shape of contact (flat or circular compression) and relative area (percentage of the available surface) may also be considered.

- Compression dose: an integrated index combining pressure, area, and time — to reflect the energetic or neurophysiological impact.

Pressure Parameters. Much of this data is better visualized via pressure curves or cyclegrams, allowing synchronized analysis of multiple zones or types of pressure:

- Phase pressure dynamics: contact force (P_0, compression from strap only), baseline compression pressure (P_{min}, before inflation), peak or plateau pressure (P_{max}), and inflation/deflation gradients.
- Peak pressure kinetics: screen value (nominal pressure), actual internal chamber pressure, effective pressure on the skin (depends on body position, fit, and tightness), subjective pressure perception (often used to gauge "dose"), and tissue-depth gradient.
- Anisotropic pressure in tissue layers: includes shear, stretch, local gradients (in fascia), directional shifts (in muscles and fascia), tensor deformation (at receptor or interface zones), hydro-mechanical gradients (in fluids), and cellular-level tension gradients.
- Dynamics of pressure pattern during the session.

Time Parameters:

- Segment-level base timings: inflation time (t_1), plateau time (t_2), deflation time (t_3), pause time (t_4).
- Derivatives: relative ratios of these phases.
- Cuff-level parameters: duration of compression and pause phases, full cycle length, and total procedure time. Derived: compression wave speed.
- Session-level and treatment plan timing: duration per session, interval between sessions, course length, interval between courses. Derived: time ratios.
- Phase-based parameters: intersegmental phase shifts and overlap; positional mapping of active segments.

Frequency Parameters:

- Absolute activity metrics: number of inflations and plateaus per minute; duration of inflation, plateau, deflation, pause.
- Relative metrics: percentage of time spent in each phase (e.g., plateau) within a cycle or full session; phase-time ratios.

Cuff Construction Parameters:

- Chamber properties: material (elastic or inelastic), wall thickness, thermal conductivity, absolute and relative size, shape, skin contact conformity.
- Multichamber layout: number of chambers, relative overlap, size gradients.
- Pneumatic tubing: length (resonant or not), pressure control mode (global or per-chamber).
- Cuff tailoring design: type of closure (single or sectional), adaptability.
- Working medium (air inside the chamber): temperature, vibration, containment (sealed or open circuit).

The list of parameter groups presented here is obviously incomplete and should be treated as an initial attempt toward forming an IPC "alphabet." It does not yet account for software-driven control algorithms, clinical context, or subjective factors. The next steps would include identifying clinically significant parameters and developing systems for their evaluation — especially for complex indices, multivector relationships, and non-linear biological effects.

Compression is not about force per se, but about how tissues respond to it. This means the same pressure value may produce very different physiological effects in different patients. To personalize the compression level, we could introduce empirical coefficients, such as: $P = (P_0 + P_{max}) \times K_1 \times K_2$, where P is the target compression for a specific patient; K_1 reflects tissue compliance — how soft and deformable the tissue is; K_2 reflects tissue rigidity — how tense or fibrotic it is.

Tissue quality could be assessed manually using simple semi-objective scales:

Tissue compliance index (K_1). Low (0–3 points), moderate (4–6), high (7–9):

- Softness under pressure: easy indentation with no resistance (3), elastic rebound (2), firm with resistance (1), rigid (0).
- Lateral skin shift: moves easily by 2–3 cm (3), moves ~1 cm (2), barely moves (1), fixed (0).
- Recovery after pressure: immediate rebound (3), slow rebound (2), persistent local dent or swelling (1), no recovery (0).

Tissue rigidity index (K_2). High (0–3 points), moderate (4–6), low (7–9):

- Resistance to finger pressure: none (0), moderate (1), strong (2), very strong (3).
- Deep tissue density: loose/soft (0), average (1), firm (2), hard (3).

- Pain with pressure (if present): no pain (0), mild discomfort (1), pain without resistance (2), pain with strong resistance (3).

The optimal area for IPC has high compliance and low rigidity. Areas with low compliance and high rigidity may be painful and less responsive, and require caution. Mixed profiles often lead to unstable or unpredictable results.

Another unsolved question is how to define derived parameters, ratios, and integrated indices. For instance, pressure alone means little without time. We need to understand which combinations or ratios actually matter for clinical outcomes.

Building a corresponding big data system is theoretically feasible, but extremely challenging. And even if developed, it would be nearly impossible to apply in clinical routine without the help of artificial intelligence. Yet this project requires a deeply interdisciplinary approach — mathematical modeling well beyond the expertise of physicians, biologists, or biomedical engineers, who currently dominate IPC research. Ironically, these same professionals would be the main users of such a system — yet they are least equipped to create it.

Let us now ask a simple question: can such a complex technology become popular among physicians? Affordable? And therefore, widely accessible? That sounds like utopia. Here we encounter a new problem — the collapse of Tarshynov's IPC. High-quality solutions are rarely cheap, and low-quality ones are not worth paying for.

An alternative approach would be to build the "alphabet" not through computational modeling, but by conducting a series of direct clinical studies on the significance of parameters, using a carefully selected representative group of volunteers. This would require substantial funding and a large team of researchers, with the potential to create a practical parameter "alphabet" within a few years. But one more element would likely be necessary: a dedicated hardware-software platform.

The Ideal Device

IPC is a device-dependent procedure. Let's consider a situational (ad hoc) classification of medical pneumatic compression devices and tentatively divide them into three classes:

- Class I: Personal-use devices — simple and safe for individual patients.

- Class II: Clinical devices for routine use — precise and flexible, designed for standardized workflows.
- Class III: Research-grade systems — experimental platforms capable of emulating virtually any IPC scenario.

What we need is a Class III system — a tool that allows simulation of any IPC procedure with any parameters. This would be a specialized development, not intended for mass production, commercial sale, or routine clinical use.

Building such a system is a challenge — but its existence might create an even greater problem: the disconnect between research and real-world application. On what equipment would regular doctors be expected to reproduce the findings of researchers working on a Class III device?

How can we convert a complex, poorly formalized technique into something that can be mathematically modeled and made broadly accessible? Here, we face the problem of interface mismatch: engineers lack clinical thinking; clinicians don't speak the language of mathematics; and mathematicians often disregard the biopsychosocial context. Much of the technical workload could be managed by artificial intelligence, but the price would be a "black box" phenomenon — where a doctor receives a decision without a transparent rationale. Even if the "alphabet" of IPC appeared tomorrow, 95% of clinicians would not be able to use it. It would require new interfaces, new visualizations, new forms of education. This is not only an interdisciplinary issue — it is also a technological and pedagogical one.

Let's take another look at the market. For years, I've participated in the distribution of IPC equipment, presenting it at scientific events, teaching and instructing users. The most troubling question I hear is: "Which device is the best?" Not because of the content — but because the question itself reveals a problem. Who really knows the correct answer? Not from a marketing perspective, but from the viewpoint of Tarshynov's IPC.

ChatGPT-4o: "*There is no such thing as the best device. There is only the best match between what a device can do and what you want to achieve. To put it more sharply: the best device is the one whose parameters you can link to a clinical outcome. Even a Class III machine is just an expensive pump without the 'alphabet'. And on the other hand, even a simple unit can work miracles in the hands of someone who knows what they're doing.*"

In my own phrasing: the best device depends on what the buyer is really asking for. For some, it's the most convenient; for others, the cheapest, or perhaps the most expensive or visually appealing. Personally, I value the one with the widest functionality. But every advantage has a flip side: a device equally suited to treating both the leg and the head is, by definition, inferior to one optimized specifically for either.

I once gave a talk at a rehabilitation event focused on limb injuries, presenting a comparative table of IPC devices available on the local market. I asked the audience to name the best and worst models. None of the market leaders met our pre-defined ideal. Ironically, the lowest-rated device was the one optimized for DVT prevention. It was the best certified and most extensively studied — but it lost because we asked the right clinical questions.

If clinicians don't define the demand, the market will do it for them. But a device that looks best in a catalog is not necessarily best for a patient. Only when clinical reasoning begins to set the terms will manufacturers start producing not just devices, but solutions.

Now consider the market from another angle. If we have many good devices, each optimized for a specific problem, this implies that none is optimized for other problems. In other words, we have many specialized devices, but no universal ones. And this is the root of the issue — the barrier to implementing newer research.

ChatGPT-4o: "*If each device only realizes 10% of the method's potential, it doesn't matter whether there are 5, 15, or 50 of them. We don't have a method — we have disconnected fragments. The market doesn't recognize the core idea of Tarshynov's IPC as a systemic regulatory method. It sees tools, not a methodology. The devices themselves are not bad — but they can't transcend their own design. They don't give the researcher freedom to think broadly or the physician the tools to think at all. There is no device of class 2.5 — one built for learning, for understanding IPC. The pedagogy of the method is not yet articulated in technical terms.*"

Such a device did exist in Ukraine from 1992 to 2015. Only 101 units were produced, and some are still in use. It remains one of the most advanced IPC machines in the world. Unsurprisingly, this made it commercially unpopular. In my view, if this heavyweight were to be revived, it would evolve: one version would correct its flaws and become a modern research-grade platform; another would degrade into a clinical-grade model — likely splitting into a family of devices tailored to specific clinical tasks.

It's possible that some IPC parameters are mutually incompatible. One device may allow control over pressure, while another permits precise timing. Therefore, a research-grade IPC platform may end up being a modular environment — a sandbox, an incubator — with interchangeable components (cuffs, control units) configured for the task at hand. This wouldn't be a single device, but rather a system for designing any desired IPC configuration. From this, simplified market-ready devices could be derived. The research rig would become a template for generating commercial models. Moreover, the results wouldn’t just apply to IPC, but could inform related domains — such as counterpulsation therapy.

Biological feedback or sensor systems would be indispensable in such a platform. In everyday practice, especially with home-use models, this could enable self-regulating IPC sessions, reducing user error and enabling remote monitoring. One solution might combine IPC with tensometric sensors to assess absolute pressure and distribution. Another might integrate IPC with bioimpedance analysis — an unconventional but logical approach, allowing individualized session duration based on tissue hydration in the treated area, and enabling dynamic visualization of patient status.

Depending on the IPC “target” in each case, biological feedback could rely on rheography, pulse oximetry, skin temperature monitoring, noninvasive brain potential registration, heart rate variability analysis, blood pressure, or, in some cases, spirometry. Psychophysiological self-assessment scales could help determine session intervals, including work capacity and quality of life.

Afterword

By tradition, this is the moment for an afterword. This text was originally intended as a closing reflection on ten years of scientific work. And yet, in the process of writing, it turned into something more — not just a summary, but a discovery of new ideas, for myself and, perhaps, for the reader. The lesson is simple: don’t be in a hurry to put a full stop. Try a question mark instead.

In this essay, I didn’t aim to provide definitive answers — only to illustrate: a list of conditions, a list of parameters, a list of possible directions. Many important topics remain for separate analysis. We haven’t touched the ethical subtleties. Nor have we spoken of the social aspects — the patients, the communities.

The Ukrainian story of this method began with a sentence, once said to Ihor Tarshynov by an oncologist:

— Why are you taking your device home? What are you thinking? Thousands of patients need it.

If any of the ideas outlined here resonate with your own experience — or raise questions, doubts, or counterpoints — I would be glad to continue the conversation. This text is only a draft of what the dialogue could become.

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