



Review Article

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Cardiac Rhythm Control & Measurement: New Perspectives

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Abstract

The heart is a complex self-regulated system in which mechanical, electrical, and neural processes interact continuously to maintain cardiac function. This study considers cardiac bioelectromagnetic activity from complementary theoretical and experimental perspectives, thereby treating it as an observable and controllable coupled system. Particular focus is placed on autonomic regulation and atrial vagal stimulation. Ionic and nerve currents in the cardiac system generate the electrical potentials recorded by Electrocardiography (ECG) and the associated magnetic fields detectable by Magnetocardiography (MCG). Based on Maxwell's equations and Plonsey-Heppner parameters, the quasi-static approach for cardiac electromagnetic modeling is validated. Retardation and magnetic induction effects are found to be negligible, whereas the high dielectric permittivity of cardiac tissue resulted in a borderline capacitive regime. As the Π_{BW} adimensional parameter - linked with the Quasi Static Approximation (QSA) - reaches 2/3 under standard conditions, the applicability of QSA is shown, while highlighting the need for tissue-specific corrections.

The BioWave1 experimental protocol evaluated the effects of targeted acupuncture on the auricular branch of the vagus nerve in a cohort of 14 healthy volunteers. Twelve-lead ECGs and infrared thermography of the thoracic region were performed before and after stimulation. The average reduction in heart rate was 6.4%, with individual reductions reaching up to 13% or 20%. In some participants, formal ECG findings were consistent with increased parasympathetic modulation. Infrared thermography showed a decrease in sternal skin temperature of up to 3.2°C, suggesting a potential influence of the autonomic nervous system on cutaneous blood flow. This is coherent with possible retro-controlled system models based on the four principles of thermodynamics applied to electrodynamical systems. These findings verify the pertinence of the conjectures that auricular vagal stimulation may modulate cardiac autonomic activity. However, the small sample size and some inter-individual variability limit definitive conclusions. The study establishes a theoretical and experimental framework for investigating cardiac bioelectromagnetic activity and electromagnetic energy flux, while supporting further research into selective autonomic modulation for cardiac rhythm control and tachycardia management. Future studies should determine whether this non-invasive protocol could be adapted as a cardiovascular countermeasure to support autonomic regulation and protect cardiac function during longduration spaceflight.

1. List of Acronyms: ABVN: Auricular Branch of the Vagus Nerve; AC: Alternating Current; ANS: Autonomic Nervous System; AV-Atrioventricular; BPM: Beats Per Minute; CEA-LETI: Commissariat à l'Énergie Atomique et aux Énergies Alternatives – Laboratory for Electronics and Information Technology; DC: Direct Current; ECG: Electrocardiogram/Electrocardiography; EEG: Electroencephalogram / Electroencephalography; HF: High-Frequency band of heart-rate variability; HR: Heart Rate; HRV: Heart-Rate Variability; IR: Infrared; IUT: Institut Universitaire de Technologie; LF: Low-Frequency band of heart-rate variability; MCG: Magnetocardiogram/Magnetocardiography; MEG: Magnetoencephalography; MHD: Magnetohydrodynamic; MRI: Magnetic Resonance Imaging; OPM: Optically Pumped Magnetometer; PR: PR interval of the electrocardiogram; QRS: Q–R–S complex of the electrocardiogram; QSA: Quasi-Static Approximation; QT: QT interval of the electrocardiogram; QTc: Rate-corrected QT interval; RR: Interval between successive R waves; SA: Sinoatrial; SDNN: Standard Deviation of normal-to-normal intervals; SQUID: Superconducting Quantum Interference Device; tACS: Transcranial Alternating-Current Stimulation; tDCS: Transcranial Direct-Current Stimulation; TRL: Technology Readiness Level

2. List of Symbols: E: Electric field (V/m); B: Magnetic flux density (T); H: Magnetic field strength (A/m); J: Current density (A/m²); ρ : Charge density (C/m³); σ : Electrical conductivity (S/m); $\sigma^*(\omega)$: Complex conductivity, $\sigma^*(\omega) = \sigma(\omega) + j\omega\epsilon(\omega)$; ϵ : Absolute permittivity (F/m); ϵ_0 : Vacuum permittivity (F/m); ϵ_r : Relative permittivity (dimensionless); μ_0 : Vacuum magnetic permeability (H/m); ω : Angular frequency (rad/s); f: Frequency (Hz); c: Speed of light in vacuum (m/s); L: Characteristic geometric length / source-sensor distance (m); r: Field-point position vector; r': Source-point position vector; t: Observation time (s); t_r: Retarded time, $t_r = t - |r - r'|/c$ (s); j: Imaginary unit, $j^2 = -1$; $P_1 = kL$: Retardation parameter; $kL = \omega L/c$; $P_2 = \omega\epsilon/\sigma$: Capacitive-to-ohmic current ratio; $P_3 = \mu_0\sigma\omega L^2$: Magnetic-induction parameter; Π_{BW} : Composite QSA parameter, $\Pi_{BW} = \max[kL, \omega\epsilon/\sigma, \mu_0\sigma\omega L^2]$; Π : Poynting vector, $\Pi = E \times H$ (W/m²); Δ : Difference or change between two measured values

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Background & Goals

The cardiorespiratory system is a complex architecture that integrates structural, fluid, and control elements, and exhibits its own behavior as well as input and output interactions with the external environment. The cardiac system, in particular, is of special interest, on the one hand due to the complexity of its mechanical interactions (non-Euclidean fluids, intrinsically nonlinear active structures) and, on the other hand, due to its innervation and the highly specific measurement and control system it contains. This complex, regulated system is capable of responding to an extremely wide variety of life-threatening situations and maintaining operability for more than a billion cycles, making it a unique system in engineering.

Modeling and observing this system has long been a fascinating scientific and technical challenge, bringing together the life sciences, materials science, physical measurement and modeling techniques, and control algorithms. In this context, the first part of this article provides a summary of current thinking on electromagnetic emissions from the heart, placing the electrostatic emissions measured by the conventional electrocardiogram within the broader context of Maxwell's equations, and considering the electrostatic field and a possible electromagnetic emission, at very

low amplitude and frequency. The idea here is to propose a physical observability that is inseparable from the commandability of the system.

In the second part, which precisely combines conventional observation with innovative control methods, this article presents the results of electrocardiographic measurements conducted on a cohort of about fifteen individuals who underwent a specific protocol. This protocol modifies cardiac control through acupuncture, an ancient technique used here in a new context. The experiments, conducted under rigorous medical and scientific supervision, show that the cardiac system can be regulated in a significant, repeatable, and predictable manner, and support a number of hypotheses related to the modeling and electromagnetic inferences of the heart.

The applications and implications of this study therefore open up two avenues. On the one hand, it enables, in the short term, the validation of new tachycardic protocols, or protocols designed to prevent cardiorespiratory stress in extreme situations (complex missions, astronauts during extravehicular activities, etc.). On the other hand, in the longer term, it opens up prospects for expanded electromagnetic modeling and measurement, enabling not only

the visualization of intracardiac physical phenomena but also the neurological regulation of the autonomic nervous system of the myocardium.

Control Principles

Auricular Acupuncture

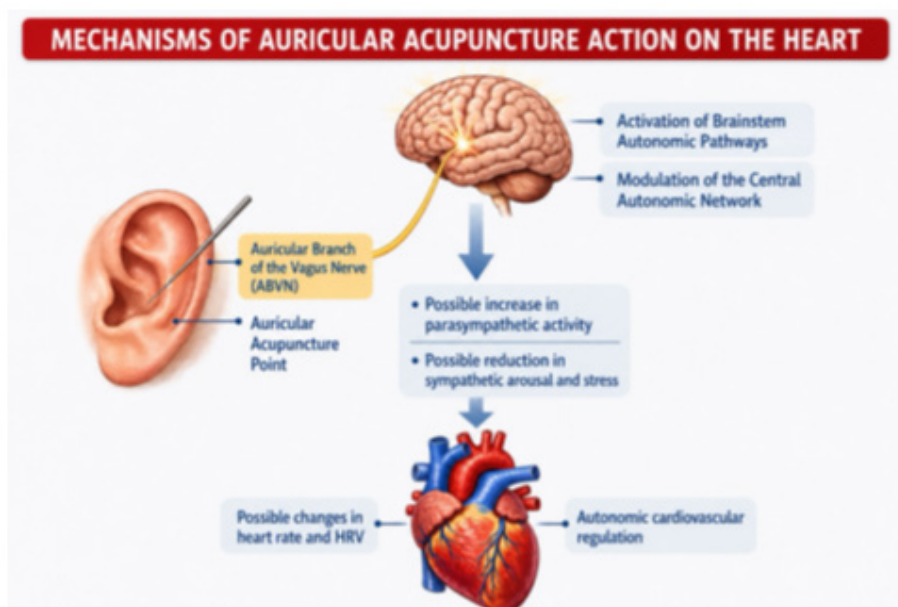
Auricular acupuncture is a therapeutic technique involving the stimulation of specific points on the external ear. Its roots can be traced back to ancient Chinese medicine, with references to the relationship between the ear and different organs, while the modern systematic approach to auricular acupuncture was later developed in France in the 1950s by Paul Nogier, who proposed a somatotopic representation of the body on the auricle [1,2]. Since then, auricular acupuncture has been investigated as a potential method of peripheral neuromodulation, particularly due to the innervation of the ear by the Auricular Branch of the Vagus Nerve (ABVN).

Auricular stimulation provides a strong anatomical basis for autonomic modulation: while body acupuncture studies measure vagal effects solely through functional outcomes, auricular stimulation relies on the innervation of the outer ear by the auricular branch of the vagus nerve. This provides a direct neuroanatomical link to this topic and makes it particularly relevant for experimental models of vagal stimulation [3,4]. The most observed result is a

modest reduction in heart rate, along with a reduction in the LF/HF ratio, across randomized, controlled trials of auricular acupuncture, transcutaneous vagus nerve stimulation, acupressure and other similar treatments [3].

However, not all changes observed in HRV measures show equally consistent and correlated changes; in fact, both total HRV and HF alone are not uniformly altered across studies [3]. Thus, the more général autonomic pattern is more protocol-dependent, while the most evident auricular effect, expected with this type of treatment, is a slowing of the heart rate. In addition to the association of auricular acupuncture with a lower heart rate, thanks to much more specific experimental work targeting treatment applied to the auricular vagal area, it has also been associated with an increase in SDNN compared to placebo stimulation and further HRV indices have moved in a parasympathetic-dominant direction [4].

The observed pattern is therefore interesting for three main reasons: auricular vagal stimulation can influence cardiac autonomic regulation in humans (see Figure 1), the effect is detectable even when not every HRV parameter changes uniformly and heart rate and SDNN as we have seen appear among the most sensitive outcomes to monitor [4]. This means that a measurable reduction in heart rate can be expected in the results, as it would be consistent with the current literature, while more complex patterns in HRV would not be unexpected anyway.



Note*: Image source: original schematic prepared by the BioWave1 team.

Figure 1: Schematic of the proposed mechanism of action of auricular acupuncture on cardiac function: stimulation of the ABVN territory activates brainstem autonomic pathways, shifting the sympathovagal balance toward parasympathetic predominance and producing measurable changes in heart rate and HRV.

Furthermore, another very interesting aspect is observed in some studies that combined central nervous system monitoring with autonomic measures, through simultaneous EEG-HRV investigations. It was found that, with the application of acupuncture treatment, there was a shift in autonomic indices toward a parasympathetic pattern even when changes in background cortical activity did not directly indicate an increase in relaxation. This observed result leads us to consider that cardiac modulation through acupuncture is beyond even subjective or psychophysiological explanations; as in this case where it appears to be driven more by supraspinal reflex pathways than by generalized cortical relaxation alone [5].

Vagus Nerve Stimulation

The autonomic nervous system modulates the electrical and mechanical activity of the heart [6,7]. Autonomic control of the heart has both extrinsic and intrinsic components: extrinsic parasympathetic innervation originates from the vagus nerve, while sympathetic fibers reach the heart through the cervical, stellate and thoracic ganglia. Furthermore, the cardiac muscle integrates local information and can modulate cardiac activity, as well as transmit central commands, through an intrinsic neural network of intracardiac ganglionated plexuses and interconnected neurons [7,8].

The two branches, the sympathetic and parasympathetic, which constitute the ANS, have different functions but exert complementary effects. When activated, the sympathetic branch increases heart rate, conduction velocity and contractility, with catecholamine-mediated activation of beta-adrenergic pathways that promote calcium entry and calcium-dependent signaling [6,7,8]. Parasympathetic functions, on the other hand, are mediated primarily by acetylcholine and aim to slow sinoatrial transmission and atrioventricular conduction; through muscarinic receptors and the potassium current (sensitive to acetylcholine I_{KACH}), they modify atrial electrophysiology [6,8]. Thus, the autonomic system not only influences frequency, but directly alters membrane currents and conduction properties [8].

As demonstrated by modern literature on HRV and neurocardiology, autonomic regulation is not a simple linear opposition between sympathetic and parasympathetic tone. Another phenomenon to consider is the temporal asymmetry between the two branches: while parasympathetic effects typically develop within one or two cardiac cycles and therefore quite rapidly, sympathetic ones are more prolonged and slower; this is precisely one of the reasons why short-term beat-to-beat variability is particularly sensitive to vagal modulation [7,9,10]. Furthermore, there is a continuous exchange and transmission of information from the heart to the central nervous system, both through the vagus nerve and elsewhere, which contributes to reflex cardiovascular regulation, for example, but also to many heart-brain interactions [7].

In atrial fibrillation, both sympathetic and parasympathetic activity, albeit through different mechanisms, can facilitate arrhythmia, which is why autonomic regulation is very important: sympathetic activation increases excitability and promotes calcium trigger-related activity, while vagal, i.e. parasympathetic, stimulation shortens the duration of the atrial action potential and refractoriness in a heterogeneous manner, thus favoring re-entry [8].

For this reason, we have provided a framework to demonstrate that the ANS is one of the main determinants of cardiac electrical behavior and rhythm stability [6,7,8].

Cardiovascular Impact

The heart is not only a mechanical pump but also an electrically active organ whose rhythm is continuously shaped by the autonomic nervous system. The available literature describes that, through measurable changes in electrical and neurocardiac markers (especially heart rate and heart rate variability), cardiac function can be strongly influenced by acupuncture. Most of the current and ongoing studies have attempted to measure not whether acupuncture causes a direct spatial modification of the cardiac electrical field itself, but rather whether this is associated with changes in ECG timing and beat-by-beat variability, which are precisely the factors indirectly linked to autonomic regulation [11,12-16]. HRV has emerged as the most widely used analytical tool within this body of current work, as it provides a non-invasive and highly practical view of the interaction between autonomic control and cardiac rhythm [11,12].

The reported effects of acupuncture on HRV, summarized in previous reviews by experimental evidence, were often inconsistent across studies, especially when analyzing the frequency domain with measurements of HF, LF and the LF/HF ratio. In fact, some reports, especially when analyzing unhealthy subjects, associated acupuncture with a reduction in LF or LF/HF; in others, no clear effect was shown, and in other cases, significant results did not even favor the verum compared to the comparison conditions. All these inconsistencies were not considered as evidence against autonomic modulation, but only as an index of method heterogeneity for example in the selection of subjects, choice of acupoints (large fundamental variable), stimulation method, study design and interpretation of HRV [12].

Conversely, some more recent meta-analytic studies, although they also confirmed that the magnitude and direction of measured changes are significantly influenced by methodological choices, have nevertheless corroborated the thesis of a measurable modulatory effect [11]. Therefore, the current state of knowledge, that has been reached with certainty, confirms the existence of a physiologically plausible and repeatedly observed modulation, but still strongly influenced by methodological and interpretative variability [11,12]. Clearer examples demonstrating a more acute modulation during treatment have been observed in some experimental studies under

controlled conditions; acupuncture is associated with a momentary decrease in heart rate along with changes in HRV, linked to an increase in parasympathetic activity and therefore also with an increase in the HF contribution and a decrease in the LF/HF ratio, with the modulation occurring during the stimulation itself rather than only in a delayed recovery period [13].

However, it is confirmed that both the point where the puncture occurs and the stimulation technique are all influencing factors, in fact when these parameters are compared both the profile and the persistence of the autonomic response change, observing for example stronger or sometimes more sustained effects [14]. From here we can therefore conclude and deduce that the cardiac modulation induced by acupuncture is a phenomenon sensitive to the physiological variations of the decided protocol [14]. Clinical studies have also investigated whether repeated sessions can generate observable and gradual changes over time; observations in hypertensive patients have demonstrated a significant reduction in LF/HF not only during single sessions but also when observed long-term over weeks or months [15].

More generally, beyond the specific case of hypertensive patients, repeated sessions of acupuncture have also been associated with gradual changes in HRV parameters and with a reduction in cardiovascular reactivity to physiological stressors, suggesting that the effect on autonomic regulation is not confined to single treatment but consolidates over time [3].

Another advance that has been made is the leap from a purely theoretical and descriptive analysis of ECG changes, to a mathematical representation of the signal itself. Lee et al. modeled the averaged ECG changes in the signal before, during and after acupuncture with generalized transfer functions, demonstrating

that the ECG changes caused by acupuncture can be sufficiently regular to be modeled quantitatively [16]. The literature on the electrical manifestations of acupuncture is therefore moving from observation towards the formal representation and prediction of signal changes.

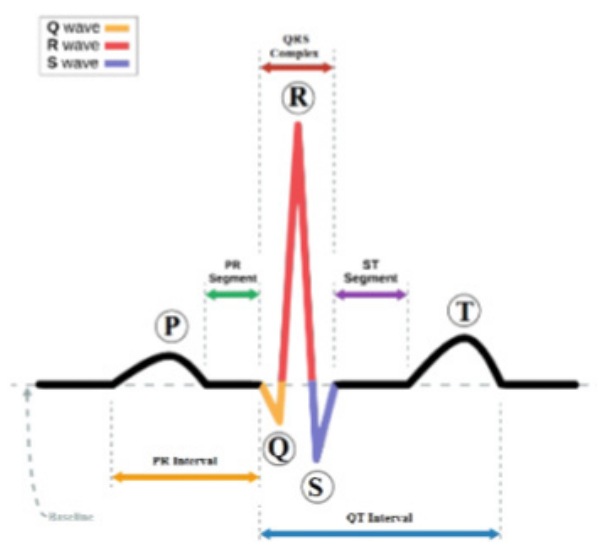
Cardiac Electrophysics

Electric Field E

During the synchronous contraction of the myocardial fibers, a precise and controlled electrophysiological process occurs that depends on the movement of charged particles across membranes within cardiac tissue cells [6,9,17]. After the propagation of the electrical activity through the myocardium, a measurable electric field is generated and detected on the body surface.

The cardiac electrical activity is influenced by the ANS in various aspects and parameters, such as impulse generation, conduction, refractoriness, repolarization and susceptibility to arrhythmias; these effects are mediated by neurotransmitters, receptor signaling and direct modulation of ionic currents. Activation of the sympathetic branch for example, increases calcium influx and calcium-dependent signaling, increasing automaticity, conduction and contractility [6,8,9].

While parasympathetic activation shortens the duration of the action potential and refractoriness, through the activation of I_{KAcH} sinoatrial discharge and atrioventricular conduction are slowed, also modifying atrial membrane currents [8]. Therefore, the autonomic effects do not simply change the frequency from outside the system, but are actually electrophysiological, as they alter the ionic determinants of cardiac electrical behavior [8,9,17].



Note*: Image source: Wikimedia Commons, public domain.

Figure 2: Schematic representation of a normal sinus-rhythm ECG complex with its main waveforms (P, QRS, T) and intervals (PR, QRS, QT, ST).

The duration of the potential, the conduction velocity and the electrical stability can be modified even with only modest alterations in the magnitude or kinetics of sodium, calcium and potassium currents [17,18]. Therefore, now, autonomic control must be included in the modern interpretation of cardiac electrophysiology as it is a determining parameter; autonomic stimulation directly or indirectly modulates these currents and generates influences detectable through ECG, shown in Figure 2, based on the shape of the wave and also through HRV [8,10].

To provide a complete cardiac bioelectrical description, we connect ionic currents at the microscopic level with electric fields, arriving at the magnetic fields detectable outside the human body. The transmembrane currents we have already mentioned, calcium, sodium and potassium, generate the action potential of cardiac cells at the cellular level, while at the tissue level, electrical sources are generated through the diffusion of these potentials in the myocardium [6,17,18,19,]. Finally, at the torso level, these sources give rise to the surface potentials detectable on the body and recorded by the ECG; the same underlying current systems give rise to the magnetic fields detectable outside the body by magnetocardiography, MCG [19-23].

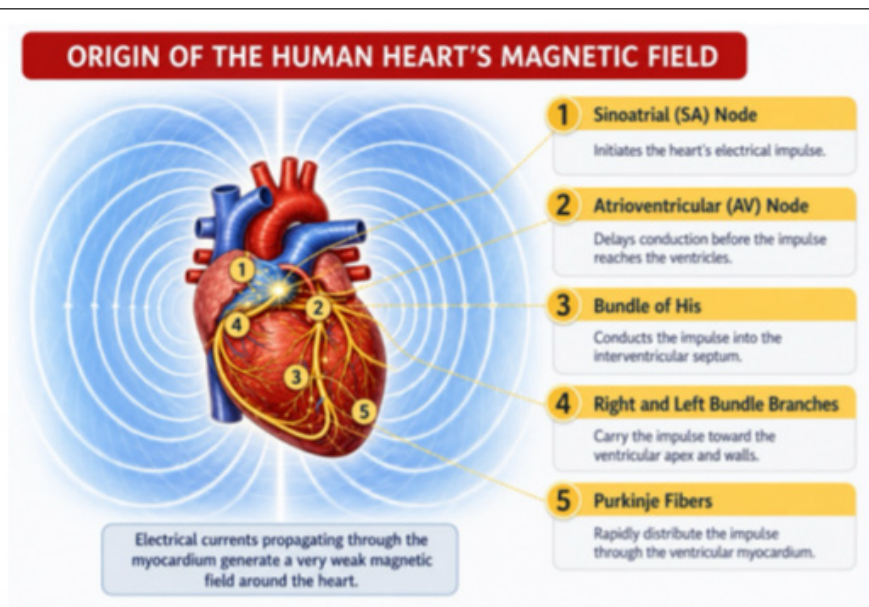
This sequence was formalized in classical biomagnetism: Geselowitz, with his theory of the electrocardiogram in 1970, was one of the first to describe the origin of cardiac bioelectric sources as impressed currents embedded in a volume conductor,

demonstrating that the reading of the body surface ECG is influenced by the resolution of the volume conductor problem rather than by the direct reading of intracellular voltages. Furthermore, it was also demonstrated that the source term is strictly linked not to the action potential of a single cell in isolation, but rather to the spatial gradient of the transmembrane potential [19].

Another very important thing that Geselowitz demonstrated is that cardiac magnetic fields measured outside a non-homogeneous volume conductor are shaped both by the distribution of the heart's electrical sources and by the geometry of the surrounding tissues: we have the contribution of the heart's internal current sources themselves which is added to the contribution of the conductivity discontinuities at the edges of the surrounding tissues [20]. The different influence given by the volume conductor is in fact one of the reasons why magnetic and electrical measurements provide complementary information of the same activity [19-22].

As a direct practical consequence, the ECG and the MCG are not two redundant readings of the same cardiac source: the surface electrocardiographic potential is in fact modeled by the heterogeneous conductivity of the thoracic volume conductor, while the magnetic field crosses the torso with magnetic permeability essentially equal to that of vacuum and therefore preserves a more faithful encoding of the underlying myocardial sources [20-22,24]. The first parameter that was investigated during the experimental campaign in BioWave1 is therefore the electric field, E , of the heart.

Electromagnetic fields B and P



Note*: Image source: original schematic prepared by the BioWave team.

Figure 3: Origin of the cardiac magnetic field: the impulse propagates along the conduction system (SA node, AV node, bundle of His, bundle branches, Purkinje fibres) and the resulting ionic currents through the myocardium generate the weak magnetic field measurable around the thorax.

Due to the movement of the same ionic charges that generate the electric field previously mentioned, a magnetic field can also be measured immediately near the chest [19,20]. As can be seen in Figure 3, the movement of these sodium, potassium, and calcium charges generates electrical currents that propagate throughout the cardiac tissue following a precise and organized sequence: the impulse originates in the sinoatrial node, is briefly delayed in the atrioventricular node, descends through the bundle of His, divides into the right and left bundle branches, and reaches the Purkinje fibers, which rapidly distribute it throughout the ventricular myocardium and trigger coordinated contraction [6]. The main point to keep in mind is the fact that every electrical current generates a magnetic field and as the wavefront of cardiac depolarization propagates through the myocardium, the associated ionic currents produce a very weak magnetic field around the chest [19,20].

The ECG and the Magnetocardiogram (MCG) are thus two different manifestations of the same single underlying source: the ECG measures the electrical potential on the body surface, while the MCG measures the magnetic field associated with the cardiac currents themselves [19,21,22]. The first experimental detection of this extremely weak field dates back to Baule and McFee in 1963, subsequently Geselowitz developed the theoretical framework relating the internal cardiac current sources to the measurable magnetic field outside the thorax [19,20,25].

Therefore, the elementary sources of currents, whose superposition produces the measurable magnetic field outside the chest, are precisely the same transmembrane ionic currents that depolarize and repolarize the myocardium and generate the ECG. Each impressed current contributes a vector field to the sensor, according to the Biot-Savart law, which depends on its direction and distance; while the volume (return) currents model the body surface electric potential but contribute comparatively less to the external magnetic field for axially symmetric geometries [20,21,24,26]. The cardiac magnetic field is on the order of tens of picoteslas at the QRS peak, typically between 50 and 100 pT a few centimeters above the chest wall, then drops into the femtoTesla range for the P wave and late repolarization components and is six to eight orders of magnitude below the geomagnetic field [21,22,24].

Magnetic recording of the heart is not redundant with electrical recording, but it instead offers a related observation. First, magnetic permeability is uniform throughout body tissue and for this reason, the magnetic field is less distorted due to the non-homogeneous conductivity that distributes the body surface potentials uniformly [20,22,24]. Furthermore, tangential current components (vortexes) are partially invisible to the surface ECG but are instead directly encoded in the magnetic signal [22,24]. Finally, MCG measurement is contactless and stable with respect to the baseline because there is no electrode polarization, no skin-electrode drift and no need to directly prepare the subject; all reasons that make this acquisition suitable for example for long-term and repeatable measurements

[22,24,27,28].

For the second part of this project, Biowave2 will investigate the induced magnetic field B [T] of the human heart; it was considered to measure it using high-sensitivity Zeeman Magnetometers, Optically Pumped Magnetometers (OPM), or Superconducting Quantum Interference Device (SQUID), in a dedicated shielded environment.

For this second phase of the program, still to be defined and whose feasibility needs to be evaluated, the MAG4Health / CEA-LETI sensor was considered, which reaches a sensitivity of the order of $\sim 10^{-15}$ T; this is consistent with the recent generation of OPM magnetometers, already demonstrated on adult and fetal cardiac magnetic signals in the laboratory and in lightly shielded environments [27,29,30,31,32]. Up until now, within BioWave1, no acquisition of the B field has yet been performed, the B axis is therefore at TRL 2 [33].

The third parameter that has been considered is the Poynting vector $\Pi = E \times H$ [W/m²], characterizing the electromagnetic energy flux that would be associated with the cardiac source. To the best of our knowledge and research, there is currently no experimental implementation and no peer-reviewed estimate of the cardiac Poynting vector has been published in any literature on magnetocardiography or biomagnetism; the most recent and comprehensive reviews on cardiac biomagnetism that have been analysed do not in fact contain analyses of the energy flux in the cardiac context, for these reasons Π is therefore at TRL 1 [21,22,33]. The project will in fact attempt to understand what a significant analysis of the cardiac Poynting vector would require and what its expected order of magnitude would be, considering the typical amplitudes of E and B at the chest wall [21,22,34,35].

Characteristic Numbers

To quantitatively analyse the cardiac electromagnetic field, we must start from Maxwell's equations, which couple the electric field E and the magnetic field B to their sources $\rho(r,t)$ and $J(r,t)$; in particular we can consider as a starting point Faraday's law ($\nabla \times E = -\partial B/\partial t$) and Ampère-Maxwell's law ($\nabla \times B = \mu_0 J + \mu_0 \epsilon_0 \partial E/\partial t$). The retarded-source solution of Maxwell's equations provides $E(r,t)$ and $B(r,t)$ at any point in the field in terms of integrals over the source densities ρ and J evaluated at the retarded time $t_r = t - |r - r'|/c$ where c is the speed of light. We now see that this solution admits two equivalent forms in the classical literature [36-38]. One form is Jefimenko's, introduced in 1966, which expresses E and B as explicit volumetric integrals over the retarded ρ and J , which reduces to Coulomb's law for E and Biot-Savart's law for B in the case of negligible retardation [34, 36]. The second form is the Panofsky-Phillips one, in which the radiative part of the field is isolated through integration by parts and provides a different but equivalent expression [38]. In 1997, McDonald demonstrated that the two forms are algebraically equivalent, so the choice between them is a matter of convenience rather than physics [37].

Whether or not the explicit delayed-source formalism, and therefore the use of the complete equations without simplifications, is necessary for a given problem, is determined by the three small, dimensionless Plonsey-Heppner parameters. If they are much smaller than one, then the quasi-static reduction can be used [34].

$$P1 = kL = \omega L/c \text{ (retardation parameter)}$$

$$P2 = \omega\epsilon/\sigma = \omega\epsilon_0\epsilon_r/\sigma \text{ (capacitive current vs ohmic current)}$$

$$P3 = \mu_0\sigma\omega L^2 \text{ (magnetic-induction parameter)}$$

Therefore, if P1, P2, and P3 are together much smaller than unity, the Jefimenko and Panofsky-Phillips integrals collapse into Coulomb's law for the electric field and into the Biot-Savart law for the magnetic field, and with the two fields decoupled; it is precisely within this framework that all modern problems on MCG and MEG are formulated [19,20,22]. In this regime, the conclusion is that the retarded form, although mathematically more general, is not numerically necessary. This is precisely what needs to be verified for BioWave1: whether the QSA is quantitatively justified for the cardiac geometry of the design.

Numerical Verification of the QSA for the Cardiac Case

Table 1: Plonsey-Heppner small parameters at $f = 100$ Hz, $L = 10$ cm, for the six BioWave1 relevant tissues.

Tissue	kL	$\omega\epsilon/\sigma$	$\mu_0\sigma\omega L^2$
Heart muscle	2.1×10^{-7}	0.66	6.5×10^{-6}
Blood	2.1×10^{-7}	4.1×10^{-5}	5.5×10^{-6}
Lung (inflated)	2.1×10^{-7}	0.36	3.2×10^{-7}
Lung (deflated)	2.1×10^{-7}	0.051	1.7×10^{-6}
Skin (wet)	2.1×10^{-7}	0.018	2.7×10^{-9}
Fat (subcutaneous)	2.1×10^{-7}	0.25	1.6×10^{-7}
Worst-case threshold	$\ll 1$	$\ll 1$	$\ll 1$

Analysing the data in the table, we can state that, first of all, kL is of the order of 10^{-7} for all tissues, so the delay parameter is perfectly negligible. Secondly, we note that the magnetic flux density parameter $\mu_0\sigma\omega L^2$ is also negligible because it is of the order of 10^{-6} everywhere. As for the ratio between capacitive current and ohmic current, $\omega\epsilon/\sigma$, it is small for blood, lung, skin, and fat (all ≤ 0.36 at 100 Hz) but reaches 0.66 in cardiac muscle; this number should be considered quite borderline and therefore the point at which the standard quasi-static reduction is not so trivial.

The explanation for the value of $\omega\epsilon/\sigma$ within cardiac muscle is due to a phenomenon called α -dispersion; counter-ions polarization occurs around the cell membranes of cardiomyocytes, causing a large increase in apparent relative permittivity up to $\epsilon_r \approx 10^7$ at 100 Hz [34,41]. Plonsey and Heppner established a standard solution that consists of using complex conductivity [34]. The real

As we anticipated, the objective of this section is to verify that kL , $\omega\epsilon/\sigma$, and $\mu_0\sigma\omega L^2$ are sufficiently small. The method used was to first evaluate the three parameters for the six tissues (cardiac muscle, blood, lung in the inflated phase, lung in the deflated phase, moist skin, subcutaneous fat) located on the source-magnetic/sensor line in BioWave1's thoracic geometry.

Since we are dealing with biological tissues, the parameters needed to calculate these values are variable, so we obtained the tissue conductivity σ and the relative permittivity ϵ_r from the Gabriel & Gabriel dielectric measurements [39], accessed through the IT'IS Foundation Tissue Properties Database [40] and sampled them at four canonical frequencies covering the cardiac signal bandwidth (1, 10, 100, and 1000 Hz). The geometric scale L is assumed to have three representative values: 5 cm (sensor near the chest), 10 cm (typical position of the MCG sensor, used as a reference geometry) and finally 30 cm (upper limit of the magnetic measurement array).

The three parameters were evaluated for the six tissues at the four sampled frequencies, for three source-sensor distances representative of the general ECG/MCG measurements ($L = 5, 10$, and 30 cm). The key numbers at the BioWave1 operating point $f = 100$ Hz, $L = 10$ cm, are shown in Table 1 below.

σ is then replaced by $\sigma^*(\omega) = \sigma(\omega) + j\omega\epsilon(\omega)$, which depends on the frequency and thus the quasi-static reduction remains valid. The numerical evaluation of this correction will be assessed with further investigations in BioWave2.

We conclude by stating that for BioWave1, the reductions in delay and magnetic flux density of the Jefimenko/Panofsky-Phillips's formulation are justified, while the reduction in displacement current remains valid with a specific reserve of cardiac tissue.

Definition and Calibration of Π_{BW}

The three Plonsey-Heppner parameters, analysed in the previous section, are condensed into a single dimensionless parameter Π_{BW} , which summarizes the regime in which BioWave1 operates and is defined as: $\Pi_{BW}(\omega, L, \text{tissue}) = \max[kL, \omega\epsilon/\sigma, \mu_0\sigma\omega L^2]$; $\Pi_{BW} \ll 1$ means that the QSA holds with respect

to the largest, and therefore most restrictive, of the three small parameters.

Three thresholds are defined for Π_{BW} : $\Pi_{BW} < 10^{-2} \rightarrow$ QSA OK (all three reductions are safe), $10^{-2} \leq \Pi_{BW} < 1 \rightarrow$ QSA borderline (typically α -dispersion regime), $\Pi_{BW} \geq 1 \rightarrow$ QSA invalid (full Maxwell solution with delay required).

For BioWave1, at the reference operating point ($f = 100$ Hz, $L = 10$ cm), $\Pi_{BW} = 0.66$, the value is dominated by the $\omega\epsilon/\sigma$ term for cardiac muscle reported in Table 1, $kL = 2 \times 10^{-7}$ and $\mu_0\sigma\omega L^2 < 5 \times 10^{-6}$ do not contribute. BioWave1 therefore belongs to the QSA-borderline class, in the α -dispersion regime; the behavior of Π_{BW} across the operational heart rate band of the project is shown in Figure 4.

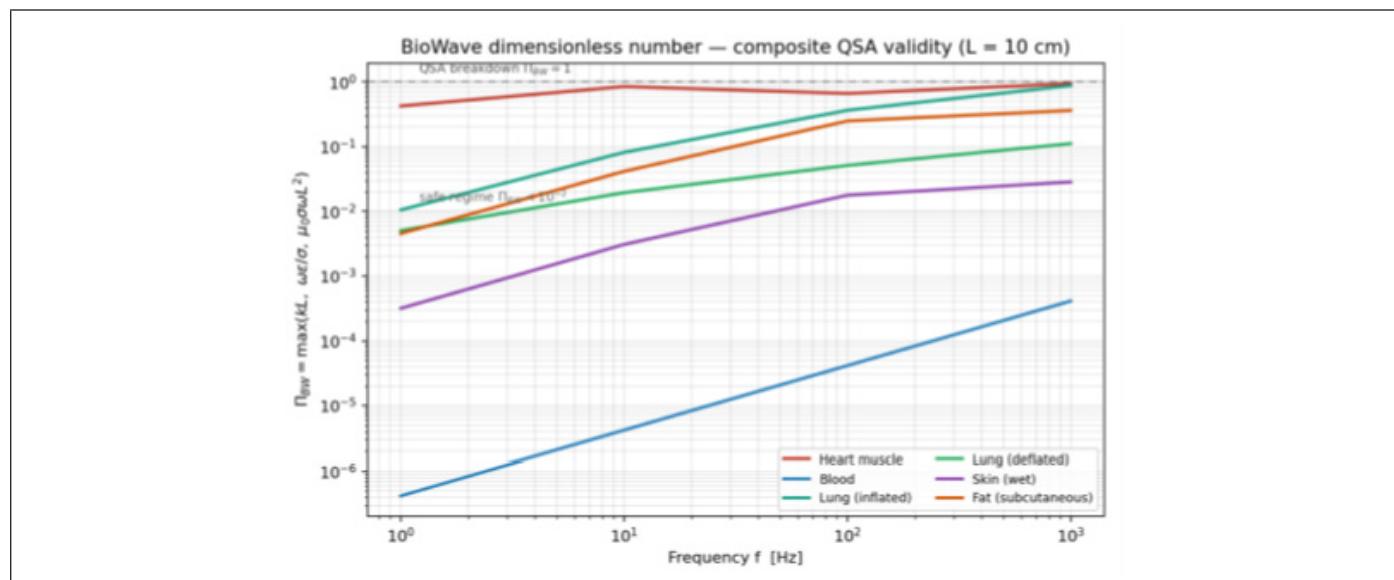


Figure 4: Composite $\Pi_{BW} = \max(kL, \omega\epsilon/\sigma, \mu_0\sigma\omega L^2)$ for the six primary BioWave1 tissues at $L = 10$ cm. Across the cardiac band, $\Pi_{BW} \leq 0.94$, dominated by the cardiac muscle entry.

To further interpret $\Pi_{BW} = 0.66$, the same numerical procedure was applied to eight reference bioelectromagnetic systems including: DC neural stimulation (tDCS), AC neural stimulation (tACS), magnetoencephalography, fetal magnetocardiography, surface ECG/EEG, MRI-induced MHD artifacts, radiofrequency MRI excitation and microwave hyperthermia. From this analysis, we can deduce that BioWave1 ($\Pi_{BW} = 0.66$) belongs to the same family as transcranial AC stimulation (0.19), fetal MCG (0.14) and surface ECG/EEG (0.014); all biological systems investigated at frequencies ≤ 100 Hz, where the α -dispersion of biological tissue determines the regime. MEG and tDCS, for example, fit comfortably in the QSA-OK region because cortical tissue has a lower ϵ_r than cardiac muscle at the same frequency. While radiofrequency MRI excitation and microwave hyperthermia are correctly classified as QSA-INVALID, the full Maxwell delayed solution is mandatory and the Jefimenko/Panofsky-Phillips formulation must be retained in its full numerical implementation.

Π_{BW} therefore places BioWave1 in a well-defined and widely populated region of the bioelectromagnetic landscape; indeed, the borderline α -dispersion class is a known regime in which a clear methodological solution exists and BioWave1's numerical value of 0.66 represents, to the best of our knowledge, the first formal inclusion of cardiac biomagnetism in this type of cross-domain

comparison.

Biowave1 Test Protocol

Electrocardiographic Measurements

For BioWave1 the goal was to conduct an experimental protocol, under controlled conditions, capable of recording, during vagal stimulation via auricular acupuncture, the cardiac electrical activity response in terms of heart rate, HRV, time and frequency-domain indices and also selected ECG morphological characteristics [6,8,19,21,22]. The expected result was a modest reduction in heart rate along with a shift in some HRV indices toward a parasympathetic predominance; this is what is supported by recent meta-analyses and experimental studies specifically targeting the auricular branch of the vagus nerve [3,4]. The experimental campaign was conducted on 14 healthy volunteers, 8 males and 6 females with a mean age of 21 years, free of cardiovascular disease and not taking psychotropic medications, conditions that could alter autonomic regulation. Intra-subject differential measurement is essential, so each volunteer serves as their own control, and the pre-stimulation, stimulation and post-stimulation phases are compared on the same individual; this isolates the response to stimulation and avoids the inter-individual variability known to be high in the literature on autonomic effects [3,4,11]. Furthermore,

the cardiac response is simultaneously observed on the electrical axis via a standard 12-lead surface ECG and on the thermal-skin axis via infrared thermography, while the magnetic axis is planned for the next phase of the program. The protocol involves the insertion of three needles at the auricular sites described below, using the PAK protocol selected by Leïla Kriaa. Its cardiovascular outcomes are examined in the context of the auriculotherapy literature [3,4].

The needles used are sterile, silicone-coated, stainless-steel needles with a twisted copper handle. The needle shaft is 15 mm in

length, with a diameter of 0.16 mm. Needle insertion is performed at three clearly defined anatomical sites on the auricle: the Shen Men point, the Zero Point, and the Sympathetic Point. Their anatomical locations are described below. The Shen Men point is located in the upper portion of the triangular fossa of the auricle, within a small depression near the bifurcation of the antihelix. The Zero Point is located in the concha, close to the center of the auricle, at the junction between the root of the helix and the concha. The Sympathetic Point is located on the root of the helix, at its junction with the concha, in the deeper portion of the auricle (Figure 5,6).



Note*: Image source: original schematic prepared by the BioWave team.

Figure 5: PAK: Acupuncture protocol selected by Leïla Kriaa for conducting the clinical trial.



Note*: Image source: illustration supplied with the BioWave additional details.

Figure 6: Pyonex. Adhesive press-needle device illustrated for the proposed future adaptation of PAK. This device is distinct from the 15 mm needles used in the BioWave1 experimental campaign.

Measurements are performed in the anechoic chamber at IUT Toulouse Blagnac, as shown in Figure 7.a. A 12-lead ECG will be acquired on volunteers lying in the supine position and a single session lasts approximately one hour; after a brief period of acclimatization with the volunteer at rest the pre-stimulation ECG is acquired. Subsequently, as can be seen in Figure 7.b, the three

needles are inserted and the stimulation phase lasts approximately twenty minutes. The post-stimulation ECG is acquired immediately after the needles are removed. The typical interval between pre- and post-stimulation recordings is between twenty and thirty minutes, consistent with the literature on acupuncture stimulation [4,13].



(7.a) Anechoic chamber at IUT Toulouse Blagnac. needles in The pyramidal RF-absorbing foam attenuates ambient electromagnetic interference and the supine bed provides a reproducible subject position across sessions.



(7.b) Insertion of the three acupuncture needles in the three selected sites of the external ear, as defined by the PAK protocol and illustrated in Figure 5.

Note*: Image source: photographs taken during the experimental campaign.

Figure 7: Experimental setup of the BioWave1 protocol: the controlled electromagnetic environment used for the recordings (7.a) and the auricular acupuncture intervention at the three PAK sites (7.b).

Data acquisitions were performed between the end of May and the beginning of June 2026. The objective of this first phase was therefore the curated within-subject dataset of ECG recordings obtained from the fourteen subjects, which now forms the empirical basis on which the multimodal magnetic analysis of the second phase of the program will be interpreted.

Thermal measurements

At the same time as the electrical measurements were performed, an infrared thermal imaging camera will be positioned on the anterior thoracic region to record the skin thermal pattern before and after stimulation. Infrared (IR) thermography was incorporated into the BioWave1 protocol as a non-invasive observable, complementary to the electrocardiographic recording of the electric axis; the technique passively captures the thermal radiation emitted by the skin surface. Medical infrared thermography has been established as a reproducible functional-

imaging modality provided that the acquisition is performed under standardised environmental conditions, a stable ambient temperature, minimised air convection, a defined patient-to-detector distance and adequate subject acclimatisation [42].

In the BioWave1 protocol, thermograms of the anterior thoracic surface were acquired immediately before and immediately after each auricular acupuncture session using a FLUKE thermal camera positioned at a fixed working distance, with the ambient temperature of the anechoic chamber kept constant. The physiological rationale for this measurement rests on the well-documented coupling between autonomic tone and cutaneous vasomotor activity: the human skin is one of the principal effectors of the sympathetic branch of the autonomic nervous system and any experimentally induced shift in the sympathovagal balance may produce a measurable change in local skin perfusion, and therefore in surface temperature, through vasoconstriction or vasodilation [43]. In the specific case of vagal activation via the auricular branch

of the vagus nerve, the modulation of the autonomic balance was expected to produce a transient thermal modulation of the localized sternal region over the timescale of the intervention. Infrared thermography therefore contributes an independent physiological signature that can either corroborate or contrast the electrocardiographic evidence acquired in parallel; an important cross-modality consistency check within the BioWave1 design.

Bio Wave1 Results

Electrical Analysis

Beta Test (May 7, 2026): The beta test was used to validate the acquisition chain and operationally define the pacing protocol. The main parameter reported was heart rate (HR), which decreased from 71 bpm before stimulation to 57 bpm after stimulation, a reduction of 14 bpm ($\approx -20\%$).

The QT interval increased from 376 ms to 412 ms (+36 ms), explained by the reduction in heart rate, without any independent change in ventricular repolarization duration. Bazett's rate correction ($QT_c = QT / \sqrt{RR}$), evaluated with $RR_{pre} = 60/71 = 0.845$ s and $RR_{post} = 60/57 = 1.053$ s, yields $QT_{c_pre} = 409$ ms and $QT_{c_post} = 402$ ms; a residual variation of -7 ms, which is within the expected measurement variability for a single recording [6,23].

Case 1 of the Main Campaign (May 27, 2026): Case 1 was acquired from a healthy 24-year-old male volunteer. The pre- and post-stimulation ECG recordings see Figure 8), separated by approximately 25 minutes and encompassing the entire acupuncture sequence, were interpreted as sinus rhythm without morphological abnormalities and with a normal electrical axis between 30° and 60° . Quantitative changes between the pre-stimulation and post-stimulation acquisitions are summarized below:



(8.a) Pre-stimulation (09:54:49): average HR 87/min over the first 5 s window.



(8.b) Post-stimulation (10:19:49): average HR 79/min over the same 5s window.

Note*: Image source: photographs taken during the experimental campaign.

Figure 8: Twelve-lead electrocardiograms of Case 1 (27 May 2026), before (8.a) and after (8.b) auricular acupuncture. Acquired at 25 mm/s and 10mm/mV with the Digital Medical MM9852 system.

Mean heart rate: 90 $\rightarrow$ 78 bpm ($\Delta = -12$ bpm, -13%). Heart rate range: 82–97 $\rightarrow$ 74–81 bpm (narrower after stimulation, consistent with rhythm stabilization under increased vagal modulation

[7,8,10]).

PR interval: 128 $\rightarrow$ 148 ms ($\Delta = +20$ ms).

QRS duration: 84 ms, unchanged.
 QT interval: 360 → 348 ms ($\Delta = -12$ ms).
 Bazett QTc: 428 → 399 ms ($\Delta = -29$ ms).
 Rhythm and axis remained unchanged.

It is confirmed that heart rate decreased by approximately 13% (slightly less than in the beta tests, and this may be due to the subject being subjected to journalistic stress in this session, as the experiment was being recorded for an article). This is close to the beta test observed and to the effect sizes reported in the literature on auricular vagal stimulation [3,4]. The narrowing of the heart rate window after stimulation (82–97 → 74–81 bpm) is consistent with the stabilization of cardiac rhythm typical of increased vagal modulation [7,8,10].

The PR interval prolonged from 128 to 148 ms, a change of +20 ms, while QRS duration remained unchanged at 84 ms. More mechanistically, the PR prolongation in the absence of QRS widening is the textbook fingerprint of acetylcholine release at the muscarinic receptors of the atrioventricular node, which slows nodal conduction without affecting His–Purkinje propagation [6,8]. It is the clearest direct electrocardiographic signature of the vagal

mechanism postulated by the auricular vagal literature and was not observable in the beta test, where the PR was not reported [3,4].

Furthermore, the rate-corrected QTc shortened by 29 ms (from 428 to 399 ms). Unlike the beta test, where the QTc was essentially stable, here the change in QTc is not explained by the reduction in rate alone. The Bazett-corrected values were manually verified ($QTc_{pre} = 360/\sqrt{0.706} = 428$ ms; $QTc_{post} = 348/\sqrt{0.759} = 399$ ms), confirming the device output. This is because after vagal stimulation we have more parasympathetic and less sympathetic behavior; since the latter generally keeps ventricular cells active for longer, if it decreases then ventricular electrical duration also decreases and this is manifested by a shorter QTc on the ECG.

Main Campaign Cohort Summary (n = 14): The complete results of the main electrocardiographic campaign are summarised in Table 2. Across the whole cohort, the mean heart rate decreased by 4 bpm (–6.38%) between the pre- and post-stimulation recordings, with individual responses ranging from a minimum impact of –4 bpm (–6.07%) to a maximum of –6 bpm (–8.05%). When stratified by sex, men show a slightly stronger mean HR reduction (–7.11%) than women (–5.42%), although the limited sample size does not allow statistically meaningful inference between the two subgroups.

Table 2: Summary of the BioWave1 main campaign cohort. Heart-rate and QT-interval changes between pre- and post-stimulation acquisitions for the 14 healthy young volunteers of the main campaign, with sex-stratified subgroups. All HR values are expressed in beats per minute (bpm); QT values in milliseconds (ms). Negative values denote reductions.

Group	Age (yrs)	Min HR change		Max HR change		Mean HR change		QT change	
		bpm	%	bpm	%	bpm	%	ms	%
Total (F+M, n=14)	21	-4	-6.07	-6	-8.05	-4	-6.38	+2	+0.58
Women (n=6)	20	-3	-4.9	-7	-8.56	-4	-5.42	+5	+1.36
Men (n=8)	21	-5	-6.95	-6	-7.67	-5	-7.11	-0.25	-0.01

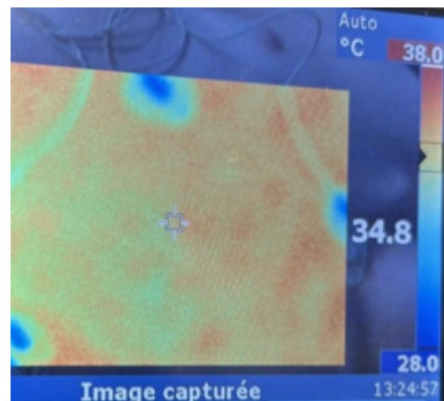
The QT interval increased by an average of 2.00 ms (+0.58%), a small change consistent with the rate-dependent prolongation expected when heart rate decreases, rather than with an intrinsic shift in ventricular repolarization. The slightly more pronounced QT response in women (+5.00 ms, +1.36%) compared with men (–0.25 ms, –0.01%) is best interpreted, in the absence of a cohort-level Bazett correction, as a rate-dependent variation within the statistical noise of small subgroups.

Overall, the cohort confirms the reproducibility and the direction of the heart-rate response already observed in the beta test and in Case 1: a measurable and consistent reduction in heart rate after auricular stimulation, with magnitudes in agreement with the auricular vagal-stimulation literature [3,4,11]. The cohort-average amplitude is more modest than the Case 1 outcome (–12 bpm, –13%), consistent with the well-documented inter-individual variability of vagal modulation in healthy young adults [7,10].

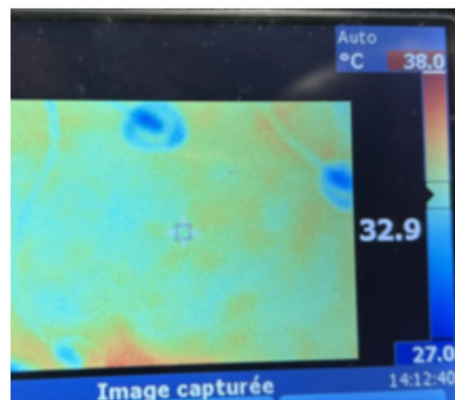
Thermal Images

During acupuncture, thermographic measurements were also taken on the anterior chest at the level of the sternum and a temperature reduction of –1.9°C was reported in the beta test (see Figure 9), from 34.8°C to 32.9°C.

During the main campaign a cooling of –3.2°C was observed (see Figure 10), from 35.4°C pre-session to 32.2°C post-session. This change reflects a localized cooling of the central thoracic surface following stimulation, already observed in beta tests. This phenomenon could occur due to a redistribution of cutaneous blood flow with localized vasoconstriction at the level of the sternum, or a reduction in metabolic and inflammatory heat production in the skin and subcutaneous tissue, consistent with meta-analytic evidence on the cardiovascular and inflammatory effects of auricular stimulation as mentioned before [3].



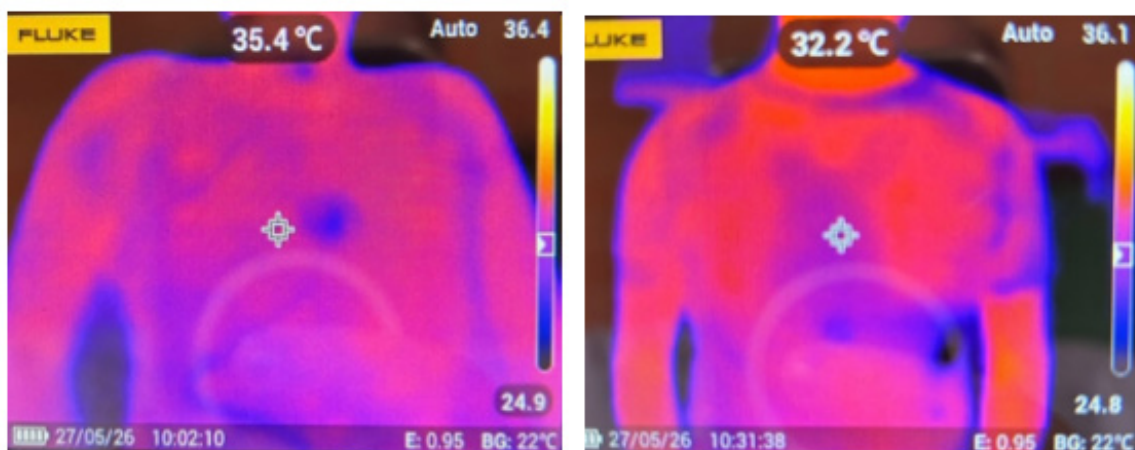
(9.a) Pre-stimulation acquisition: central thermal reading of 34.8°C across the sternal area.



(9.b) Post-stimulation acquisition: central thermal reading of 32.9°C, indicating a qualitative reduction of approximately 1.9°C.

Note*: Image source: photographs taken during the experimental campaign.

Figure 9: Infrared thermography of the anterior thoracic region during the beta test of May 7, 2026. The qualitative cooling of the sternal area between the pre-stimulation (9.a) and post-stimulation (9.b) acquisitions is consistent with vagally-mediated cutaneous vasomotor redistribution following auricular stimulation, and anticipates the larger and quantified -3.2°C effect observed in Case 1.



Note*: Image source: photographs taken during the experimental campaign.

Figure 10: Infrared thermography of the anterior thoracic region in Case 1 of the main campaign: pre-stimulation and post-stimulation images, with reported temperatures of 35.4°C and 32.2°C, respectively.

Discussion

Overall, the beta test, Case 1 and the full main-campaign cohort are consistent with and supported by the current literature on acupuncture stimulation of the auricular vagus nerve. Indeed, a reduction in heart rate was measured after treatment, along with ECG and thermographic changes, all consistent with behavior linked to the parasympathetic pathway. The reduction in heart rate observed across all three layers of evidence are: -14 bpm in the beta test, -12 bpm in Case 1 and a cohort-average of -4 bpm (-6.38%) across the 14 subjects of the main campaign.

These results are quantitatively consistent with meta-analytic findings in the literature that have identified a reduction in heart rate as the most reproducible cardiovascular outcome through randomized controlled trials of auricular stimulation, as well as with experimental findings of an increase in SDNN and a reduction in heart rate after stimulation of the auricular branch of the vagus nerve [3,4]. The narrower heart rate window after stimulation in Case 1 is similarly consistent with the increased vagal modulation also reported in the literature [11,13]. The temporal profile observed in Case 1, a measurable autonomic shift 25 minutes after the onset of pacing, is also consistent with the time course documented in the literature, in which the autonomic effect develops during pacing rather than only in a delayed recovery phase [5,13]. The selective slowing of atrioventricular nodal conduction in the absence of QRS widening is the direct electrocardiographic translation of vagal acetylcholine action on the AV node and provides a clear signature of the vagal mechanism [3,4,6,8].

Infrared skin cooling at the sternal midline is certainly the most innovative result of the campaign but the least directly supported by the existing literature. However, in support of this result, we can consider: reproducibility across two independent subjects, constancy of environmental temperatures, which excludes thermal drift of the room or sensor, and consistency in the direction of change (cooling, not heating).

Furthermore, another important information was found: The subject reported a subjective sensation of immediate calm and a perception of cardiac slowing from the moment of needle insertion. Although subjective reports cannot be treated as objective findings, the alignment of subjective experience with measured ECG and IR changes is consistent with autonomic effects related to relaxation and alertness, and also supports the interpretation of the post-stimulation state as parasympathetic predominance [5].

Conclusion & Perspectives

The electrical emissions of the cardiac system can be quantified, as a first approximation, using a quasi-stationary analysis. A dimensionless number can be proposed to evaluate the dynamics of cardiac activity through the electric field, as measured by the electrocardiogram. This approach makes it possible, on the one hand, to normalize the signal and, on the other hand, to pave the way for future magnetostatic and electromagnetic observations.

The expected orders of magnitude will differ significantly from static amplitudes but will also enable the measurement of more subtle phenomena. Indeed, these emissions are due to physiological factors intrinsic to the tissues, as well as to the couplings and regulatory mechanisms of the cardiac nervous system, which is highly specific and autonomous relative to the central nervous system.

The tests conducted using auricular specific acupuncture have demonstrated the protocol's effectiveness, both in terms of heart rate (correlated evidence) and thermal emission (more nuanced evidence). The measurements associated with these protocols open up new possibilities for moderating heart rate and managing tachycardia. We therefore propose a strategy of expanded observability and selective control, enabling a controlled cardiac rhythm management approach.

Finally, this study also provides an opportunity to examine combined systems consisting of living solids, non-Newtonian fluids, and integrated control systems through their electrical emissions in a general sense, with a focus on very low intensities and very low frequencies in self-regulated mode.

Potential application to Astronaut Health

Long-duration human spaceflight alters cardiovascular loading and autonomic regulation, providing a rationale to investigate adjunctive countermeasures [44]. Volf's parabolic-flight report involving Jean-François Clervoy offers a historical precedent for auricular acupuncture in aerospace conditions, but concerned motion sickness rather than sustained cardiovascular protection [45,46,47].

Building on the BioWave findings, a possible next step would be to investigate three Pyonex adhesive press needles placed at the PAK sites: Shen Men, Zero Point and Sympathetic Point. The compact format of Pyonex devices makes this proposal attractive for exploration in operational environments where simplicity and portability matter. Their cardiovascular effects, tolerability and appropriate application schedule would first need to be evaluated in controlled terrestrial and space-analogue studies. The BioWave observations and the available clinical precedent therefore suggest a concrete research direction: assessing whether the PAK approach could become a practical adjunct for supporting astronauts' cardiovascular regulation. Its feasibility deserves investigation, with a realistic prospect of contributing to future spaceflight countermeasures.

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References

1. Hou PW, Hsin Cheng Hsu, Yi Wen Lin, Nou Ying Tang, Chin Yi Cheng, et al. (2015) The History, Mechanism, and Clinical Application of Auricular

- Therapy in Traditional Chinese Medicine. *Evid Based Complement Alternat Med* 2015: 495684.
2. He W, Xiaoyu Wang, Hong Shi, Hongyan Shang, Liang Li, et al. (2012) Auricular Acupuncture and Vagal Regulation. *Evid Based Complement Alternat Med* 2012: 786839.
 3. K Hua, Mike Cummings, Miriam Bernatik, Benno Brinkhaus, Taras Usichenko, et al. (2023) Cardiovascular effects of auricular stimulation: a systematic review and meta-analysis of randomized controlled clinical trials. *Front Neurosci* 17: 1227858.
 4. Boehmer A, S Georgopoulos, J Nagel, Thomas Rostock, Axel Bauer, et al. (2020) Acupuncture at the auricular branch of the vagus nerve enhances heart rate variability in humans: an exploratory study. *Heart Rhythm* 17(3): 215-221.
 5. Chikako Uchida, Hideaki Waki, Yoichi Minakawa, Hideaki Tamai, Shogo Miyazaki, et al. (2020) Acupuncture Relaxation, Vigilance Stage, and Autonomic Nervous System function: a comparative study of their interrelationships. *Med Acupunct* 32(4): 218-228.
 6. M Sampson and A McGrath (2015) Understanding the ECG. Part 1: Anatomy and physiology. *British Journal of Cardiac Nursing* 10(11): 548-554.
 7. Shaffer F, R McCraty, and C L Zerr (2014) A healthy heart is not a metronome: an integrative review of the heart's anatomy and heart rate variability. *Front Psychol* 5: 1040.
 8. A Khan, GYH Lip, A Shantsila (2019) Heart rate variability in atrial fibrillation: the balance between sympathetic and parasympathetic nervous system. *Eur J Clin Invest* 49(11): e13174.
 9. DM Bers (2002) Cardiac excitation-contraction coupling. *Nature* 415(6868): 198-205.
 10. F Shaffer, JP Ginsberg (2017) An overview of heart rate variability metrics and norms. *Front Public Health* 5: 258.
 11. SHamvas, P Hegyi, SKiss, Sz Lohner, D McQueen, et al. (2023) Acupuncture increases parasympathetic tone, modulating HRV- systematic review and meta-analysis. *Complement Ther Med* 72: 102905.
 12. WY Chung, VC M Yan, H Zhang (2014) Effect of acupuncture on heart rate variability: a systematic review. *Evid Based Complement Alternat Med* 2014: 819871.
 13. C Uchida, Hideaki Waki, Yoichi Minakawa, Hideaki Tamai, Tatsuya Hisajima, et al. (2018) Evaluation of autonomic nervous system function using heart rate variability analysis during transient heart rate reduction caused by acupuncture. *Med Acupunct* 30(2): 89-95.
 14. Li L, S Liang, J Bai, Yun Zeng, Mengzhen Zhang, et al. (2025) Regulation of autonomic nervous system by acupuncture: a heart rate variability study on physical stress. *Front Hum Neurosci* 19: 1676863.
 15. K Sparrow, B Golianu (2014) Does acupuncture reduce stress over time? A clinical heart rate variability study in hypertensive patients. *Med Acupunct* 26(5): 286-294.
 16. H Lee, Hyunho Kim, Jungkuk Kim, Hwan Sup Oh, Young Jae Park, et al. (2017) Feasibility study of transfer function model on electrocardiogram change caused by acupuncture. *BMC Complement Altern Med* 17(1): 101.
 17. Y Rudy, J R Silva (2006) Computational biology in the study of cardiac ion channels and cell electrophysiology. *Q Rev Biophys* 39(1): 57-116.
 18. JM Nerbonne, RS Kass (2005) Molecular physiology of cardiac repolarization. *Physiol Rev* 85(4): 1205-1253.
 19. DB Geselowitz (1989) On the theory of the electrocardiogram. *Proceedings of the IEEE* 77(6): 857-876.
 20. DB Geselowitz (1970) On the magnetic field generated outside an inhomogeneous volume conductor by internal current sources. *IEEE Transactions in Biomagnetism* 6(2): 346-347.
 21. B J Roth (2024) The magnetocardiogram. *Biophys Rev (Melville)* 5(2): 021305.
 22. D Brisinda, P Fenici, R Fenici (2023) Clinical magnetocardiography: the unshielded bet — past, present, and future. *Front Cardiovasc Med* 10: 1232882.
 23. P Kligfield, LS Gettes, JJ Bailey, Rory Childers, Barbara J Deal, et al. (2007) Recommendations for the standardization and interpretation of the electrocardiogram. Part I: the electrocardiogram and its technology. *Circulation* 115(10): 1306-1324.
 24. K Zhu, A Kiourti (2022) A review of magnetic field emissions from the human body: sources, sensors, and uses. *IEEE Open Journal of Antennas and Propagation* 3: 732-744.
 25. G Baule, R McFee (1963) Detection of the magnetic field of the heart. *American Heart Journal* 66(1): 95-96.
 26. H Koch (2004) Recent advances in magnetocardiography. *J Electrocardiol* 37 Suppl: 117-122.
 27. S Strand, William Lutter, Janette F Strasburger, Vishal Shah, Oswaldo Baffa, et al. (2019) Low-cost fetal magnetocardiography: a comparison of superconducting quantum interference device and optically pumped magnetometers. *J Am Heart Assoc* 8(16): e013436.
 28. Tavarozzi I, S Comani, C Del Gratta, Silvano Di Luzio, Gian Luca Romani, et al. (2002) Magnetocardiography: current status and perspectives. Part II: clinical applications. *Ital Heart J* 3(3): 151-165.
 29. G Bison, N Castagna, A Hofer, P Knowles, JL Schenker, et al (2009) A room-temperature 19-channel magnetic field mapping device for cardiac signals. *Applied Physics Letters* 95(17): 173701.
 30. Jensen MA, Skarsfeldt H, Stærkind Hans, Jens Arnbak, Mikhail V Balabas, et al. (2018) Magnetocardiography on an isolated animal heart with a room-temperature optically pumped magnetometer. *Scientific Reports* 8: 16218.
 31. ME Limes, E L Foley, T W Kornack, S Caliga, S McBride, et al. (2020) Portable magnetometry for detection of biomagnetism in ambient environments. *Physical Review Applied* 14: 011002.
 32. N Aslam, H Zhou, E K Urbach, Matthew J Turner, Ronald L Walsworth, et al. (2023) Quantum sensors for biomedical applications. *Nat Rev Phys* 5(3): 157-169.
 33. John C Mankins (2004) Technology Readiness Levels: A White Paper. Tech. rep. Originally issued April 6, 1995. Edited version: 22 December 2004. Advanced Concepts Office, Office of Space Access and Technology, NASA.
 34. R Plonsey, DB Heppner (1967) Considerations of quasi-stationarity in electrophysiological systems. *Bull Math Biophys* 29(4): 657-664.
 35. P Wikswo, J P Barach, JA Freeman (1980) Magnetic field of a nerve impulse: first measurements. *Science* 208(4439): 53-55.
 36. O D Jefimenko (1989) Electricity and Magnetism: An Introduction to the Theory of Electric and Magnetic Fields, and of Electromagnetic Radiation. 2nd. First edition: Appleton-Century-Crofts, New York, 1966. Star City, WV: Electret Scientific Company.
 37. T McDonald (1997) The relation between expressions for time-dependent electromagnetic fields given by Jefimenko and by Panofsky and Phillips. *American Journal of Physics* 65(11): 1074-1076.
 38. WKH Panofsky, M Phillips (1962) Classical Electricity and Magnetism. 2nd Dover reprint 2005. Addison-Wesley.

39. S Gabriel, R W Lau, C Gabriel (1996) The dielectric properties of biological tissues: II. Measurements in the frequency range 10 Hz to 20 GHz. *Phys Med Biol* 41(11): 2251-2269.
40. PA Hasgall (2022) IT'IS Database for thermal and electromagnetic parameters of biological tissues, Version 4.1. IT'IS Foundation, Zürich.
41. J Malmivuo, R Plonsey (1995) *Bioelectromagnetism: Principles and Applications of Bioelectric and Biomagnetic Fields*. Oxford University Press.
42. Ring EFJ, Ammer K (2012) Infrared thermal imaging in medicine. *Physiol Meas* 33(3): R33-R46.
43. Charkoudian N (2010) Mechanisms and modifiers of reflex induced cutaneous vasodilation and vasoconstriction in humans. *J Appl Physiol* (1985) 109(4): 1221-1228.
44. KT Mandsager, D Robertson, A Diedrich (2015) The function of the autonomic nervous system during spaceflight. *Clin Auton Res* 25(3): 141-151.
45. Nadia Volf (2009) Auriculothérapie et acupuncture dans le mal de l'espace. *Acupuncture & Moxibustion* 8(3). Auricular acupuncture protocol applied during 31 parabolas on the A300 Zero-G (Novespace/CNES), Bordeaux-Mérignac, with French astronaut Jean-François Clervoy as project lead: 174-175.
46. RC Niemtzw (2023) Aerospace acupuncture and space flight: a realm not too distant. *Med Acupunct* 35(2): 61-62.
47. Gourinat Y, Christon L, Lachaud F (2021) Structural Dynamics in Biology. A bridge Given by Implicit Vibratory Crossed Models. *Engineering* 13: 5.