Position Paper About Experimental Hypnosis

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mental images certainly exist

for the rest of the world
one must experiment

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Table 1. Acronyms Used in the Text.

ACC	anterior cingulate cortex
APA	American Psychological Association
ART	Artigianelli Echosystem Institute
BIS	bispectral index
c.c.	Codice Civile Italiano
CO	cardiac output
CSI	cerebral state index
D.L.	Decreto Legge
DA	dopamninergic
DACC	dorsal anterior cingulate cortex
DAT	dopamine active transporter
dlPFC	dorso-lateral prefrontal cortex
DOP	central opoid recepotor(s) δ
EC	European Community
ECG	electrocardiogram(s), electrocardiographic
EEG	elecroencephalogram(s), elecroencephalographic
EU	European Union
fMRI	functional magnetic resonance imaging
GABA	γ-gamma-hydroxybutyric acid
GDPR	General Data Protection Regulation
HF	high-frequency
HFA	hypnotic foused anesthesia
HGA	hypnotic general ansthesia
highs	subjects highly susceptible to hypnosis
HRV	heart rate variability
IASP	International Association for the Study of Pain
IFG	inferior frontal gyrus
IPL	inferior parietal lobule
L.	Legge
LF	low-frequency
lM1	left motor cortex
LORETA	low-resolution brain electromagnetic tomography
lows	subjects not very susceptible to hypnosis
MBZDP	modern benzodiazepine(s)
MCC	medial cingulate cortex
M.D.	medical doctor
MOP	central opoid recepotor(s) μ
mPCC	medial prefrontal cingulate cortex
mPFC	medial prefrontal cortex
NET	norepinephrine transporter
PAG	periaquaductal gray
PET	positron-emission tomography
Ph.D.	post-doctoral Doctor of Phylosophy
RC	responsabilità civile
rM1	right motor cortex
ROFC	right orbito-frontal cortex
R-R	interval between two peaks of the rapid ventricular complex on the ECG
SERP	somatosensory evoked potentials
USA	United States of America

Part 1
Theory and Premises

1.1 Who is Participating in this Position Paper

The authors of this *Position Paper* are scientists from diverse backgrounds who have experience in the field of experimental hypnosis and have been interested for years in the search for scientific truth, particularly in the fields of the *ψυχή* (psyche), consciousness, and hypnosis. They know each other, have already collaborated actively, and have come together because they share a shared interest in moving hypnosis from the realm of narrative disciplines to that of Galilean science.

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The 11 experts gathered here – representing 22 fields of expertise, 4 universities, 1 superior institute and 4 hospitals – have joined a nascent, grassroots movement that has struggled to gain traction. They have decided to produce this *Position Paper* in an effort to move hypnosis out of the realm of narrative disciplines and into the fold of Galilean scientific ones. While hypnotism originated in an era when little was known about the

[1] Al acronyms and abbreviations are listed and explained in Table 1.

mind and almost nothing about hypnosis itself, we now live in the *age of neuroscience*, possessing cultural approaches and technological tools unavailable to the founding fathers. The opinion of the experts gathered here is that hypnosis cannot exist in ignorance of its underlying scientific bases. Even those who employ it in a non-scientific manner will benefit from an understanding of the underlying mechanisms. Indeed, it is also important to know what is real in the physical world and what, conversely, is merely real as a subjective experience of the patient.
Furthermore, experimental research in the field of hypnosis has shed light on many aspects of the mind.

1.2 Why the Focus on Experimental Hypnosis?

Hypnosis – defined as a state[2] of focused attention, reduced contact with the environment, and a slight reduction in critical judgment [108] – occurs either spontaneously or in a dedicated setting, whether through the use of specific techniques [235] or even in their absence [31]. This definition, along with many others proposed over time [34,42,75,125,145,280] – too numerous to summarize here[3] – fails to clarify the nature of hypnosis or explain what the *trance state* actually entails. Even when definitions do address the nature of the phenomenon, they tend to limit themselves to describing the phenomenology of hypnosis without delving into the underlying mechanisms. While useful in some settings[4], this approach does not help bring hypnosis into the realm of science, thereby contributing to its status as a predominantly descriptive discipline.

1.3 Experimental Research and the Galilean Method

Galilean science is the modern model of scientific inquiry established by Galileo Galilei [138[5],139,255], combining empirical observation with mathematical analysis [268]. Under this approach, for a statement to be considered a scientific law, it must abandon dogmas based on authority, be grounded in criteria of objectivity, measurability and verifiability, and remain open to potential falsification (i.e., being contradicted by more compelling data). It therefore focuses on the primary, measurable properties of physics and uses subjective qualities merely as an initial guide. Every law of the naturalistic universe[6] must be expressible through equations ranging from simple to highly complex. Verifiability requires the formulated law to pass what Galilei [138] termed the *il vaglio del cimento*, i.e. a practical trial reproducible in the laboratory by other researchers. From this perspective, the truth value of a statement does not depend on who makes it, but solely on empirical facts. Furthermore, every statement must be predictive and causal; that is, it must identify the underlying biological or mechanical cause→effect relationships, thereby enabling the prediction of future phenomena under identical conditions [39]. Not all these characteristics of scientific research necessarily appear simultaneously; research is a dynamic process that unfolds over time, sometimes spanning generations, and certain assertions are considered true *pro tempore* until potentially falsified. Many laws once held to be true *pro tempore* have subsequently been disproven or refined. The two pillars of the Galilean method are *sensate experiences* rooted in an analytical/inductive phase (the careful, systematic observation of natural phenomena, aided by technological instruments to gather numerical data), and *necessary demonstrations* rooted in a synthetic/deductive phase (leading to the formulation of a logical-mathematical hypothesis, which is then tested *via* a thought experiment or a real-world experiment to confirm or refute the law). The Galilean process

[2] Here, the modern state theory is adopted rather than the non-state theory, in line with experimental evidence from imaging techniques [63,68,72,73] which affirms that *hypnosis is a state of consciousness spontaneously occurring or induced through various techniques* [182], and rejecting the view of it as a mere *skill metaphor* [83,181]. Other interpretations, not without merit, have been proposed in the past; artificial intelligence may well contribute to this area of inquiry.
[3] Some definitely wrong.
[4] In its September 2009 *Position Statement on Hypnosis*, the APA [8] established that the practice of hypnotism and the use of hypnosis do not, in themselves, constitute forms of psychotherapy. In a subsequent 2015 statement, the APA clarified that while hypnosis does not qualify as a therapy in its own right, it should be employed by psychiatrists or other healthcare professionals (in Italy, this category includes physicians, psychologists, and likely dentists) who have undergone appropriate training and certification – as is the case with hypnosis training schools – and who operate within the scope of their respective professional competencies. (https://www.psychiatry.org /File%20Library/About-APA/Organization-Documents-Policies/Policies/Position-Hypnosis.pdf).
[5] See also: https://www2.museogalileo.it/it/biblioteca-e-istituto-di-ricerca/progetti/incontra-galileo/31-opere/460-dialogo-sopra-i-due-massimi-sistemi-del-mondo-1632.html and in https://liberliber.it/autori/autori-g/galileo-galilei/dialogo-sopra-i-duemassi mi-sistemi-del-mondo-tolemaico-e-copernicano/ o anche in https://archive.org/details/dialogo sopraidue01gali.
[6] Everything extending from the initial singularity (often called the *Big Bang*) to the present, including the experimenter.

represents a continuous synthesis of these phases. Today, all scientific and technological discoveries across every field are achieved in this manner. An assertion not demonstrated according to the criteria outlined above is not necessarily false; it is simply indeterminate, i.e. potentially true or false. It does not constitute science; rather, it is a descriptive statement, an opinion that may be either useful or misleading, yet adds nothing to our understanding of the nature of the universe.

1.4 Tools for Experimental Research on Hypnosis

First of all, hypnosis is in itself a tool [55,62]. Furthermore, research employs a variety of technological instruments – ranging from the very simple to the highly complex – to render results objective, measurable, and reproducible. Unlike in clinical practice, where the hypnotist's conviction suffices [74], experimentally demonstrating that induction has occurred (§ 2.3) or that a hypnotic state has been achieved is a significant challenge that has only recently been resolved (§ 2.5). Although modern understanding acknowledges that the seat of the psyche, higher functions, and the Ego is not necessarily the brain [43], experimental studies of hypnosis tend to rely on electrical methods such as EEG[7] (Table 2) or on more refined, precise brain imaging techniques like fMRI or PET[8] [73,97].

Table 2. ECG waves and their meaning.

Sensorium	Waves	Frequency (Hz)	Realated to
HIGH ⇩	β	23-30	active wakefulness (but also stress, anxiety, arousal)
	γ	30-42	higher cognitive functions (learning, insight, arousal)
	α	8-13	closed-eye waking relaxation
	θ	4-8	drowsiness, light sleep (even in REM phase), imagination, *mind turned inward*
	δ	5-4	deep dreamless sleep (stage N3)

PET (the only technology enabling cerebral receptor neuroimaging) (§ 2.2), delivering a dose of 10-20 mSv due to the radioactive tracers ^{18}F or ^{15}O [77], entails a risk considered acceptable in routine medical practice but questionable in research[9]; conversely, fMRI is considered non-invasive, offers higher spatial resolution[10] than PET, and allows for the visualization of the temporal stages of brain activation [77]. Since both methods are complex, expensive, and time-consuming, the research protocol must be meticulously designed prior to commencement (§ 1.3-1.8).

Hypnotic *trance (hypnosis)* develops when *plastic monoideism* (§ 2.5) – a phenomenon whose demonstration has only recently become possible thanks to fMRI and a creative protocol [271] (§ 2.5) – is triggered. There is ongoing debate in many circles as to whether degrees of hypnotic depth exist or whether it is an *all-or-nothing* phenomenon [83] (§ 2.4). However, in specific tasks, hypnotic depth can be measured using indices that employ an alternative description of the EEG by quantifying its sinusoidal components[11] (§ 2.4). Sometimes a hypnotic effect that is not directly measurable can be quantified indirectly by assessing its secondary effects, such as cardiovascular, autonomic, or biohumoral changes. From a cardiovascular perspective, useful tools include the ECG, spectral analysis of the R-R interval, HRV, and devices for measuring total [57] and segmental [59] arterial flow; when arterial pressure values are known, these allow for the calculation of

[7] Pain-related EEG changes classically consist of an increase in β-HF and δ-LF components and a reduction in the α component [76, 77]. However, in hypnosis research, EEG analysis now routinely relies on processing systems and automated projection onto standard anatomical atlases, such as LORETA [62], to facilitate the interpretation and visualization of results.

[8] The positron is the antiparticle of the electron, possessing the same mass but a positive electric charge. In PET, positron-emitting radioactive substances injected into the body annihilate upon colliding with electrons, transforming into γ rays that are detected by sensors to create detailed images of organs and their metabolism.

[9] Activated brain areas require more oxygen and glucose. Cerebral blood flow in a given area or region is proportional to activation, which can be inferred by measuring β-decay using external sensors [77].

[10] A resolution, however, that does not go so far as to distinguish between adjacent or very closely spaced brain nuclei [77,78].

[11] Widely used by anesthesiologists particularly for assessing deep sedation, but also applicable to hypnosis [27,158,167,233,253].

arteriolar resistance, which are proportional to the level of adrenergic stimulation (§ 2.14). The same methods, when applied to veins, allow for the measurement of flow and resistance within the capacitance system (§ 2.15) [140].
Internal medicine ultrasound allows for the assessment of the diameter and flow of visceral vessels (mesenteric artery, renal arteries) and the portal vein (§ 2.15).
Experiments investigating mental performance and reactivity utilize cardiovascular methods and electronic measurement of reaction times (§ 2.14).
In short, any task in which an attempt is made to demonstrate an effect *via* hypnosis requires that the effect be quantified using instruments generally available only to major research institutions rather than individual practitioners, and that it be measured not arbitrarily or subjectively, but in standard units of human physiology. This enables the effect to be incorporated into an equation describing its behavior. Although standardized tests and visual-analogue scales [123] are frequently used in hypnosis research (§ 2.10), their results are not free from ambiguity and should be viewed merely as complementary: subjecting subjective data to statistical analysis does not render them objective, nor does it constitute a purely experimental method.
The following sections (Part 2) will provide a detailed description of various phenomena induced during hypnosis that have been scientifically demonstrated and accurately measured within an experimental setting.

1.5 Informed Consent and Other Ethical Aspects

In any healthcare activity, preventing adverse events is essential for both ethical and legal reasons. This rule is particularly relevant in healthcare research; when the research involves hypnosis, the need becomes even more pressing, as the practice of hypnotism is viewed with suspicion and finding qualified court-appointed expert is difficult.
Any experimental research, including studies involving hypnotism, must be approved by the competent Bioethics Committee. This presents the first major hurdle, as these committees (like people) often know little or nothing about hypnotism, frequently equating the hypnotic state with a loss of consciousness (which is not the case). Consequently, getting a study protocol accepted is generally more difficult than obtaining approval for other types of research involving human subjects. The recent GDPR regarding personal data protection, in force since May 25, 2018, and immediately binding for all EU member states, mandates that anyone processing data on EU residents must do so transparently and securely, and solely for legitimate purposes. It also grants individuals the right to know what data is being collected, to access and correct it, and to have it deleted (the *right to be forgotten*). While many of these rules existed previously, the GDPR has significantly tightened enforcement.
Bioethics Committees are composed of members from highly diverse backgrounds, and apply laws and regulations strictly, leaving little room for personal initiative. A crucial aspect is their intense focus on statistical details; they employ expert statisticians and often raise objections in this area (for instance, regarding practical feasibility, sample sizes, or proposed data analysis methods). Since weeks or months can elapse each time a protocol is returned to the Committee for modification, the protocol itself must be drafted with almost obsessive precision.
Some argue that failing to consult the Bioethics Committee does not constitute a serious legal offense; however, it is unwise to test the judiciary with such conduct, not least because, even if criminal liability were avoided, there would still be civil liability issues regarding damages (and it is not always certain that professional liability insurance covers the practice of hypnosis, especially for experimental purposes).
What everyone agrees upon is the absolute importance of *informed consent*[12], an aspect regarding which many researchers are not adequately trained. *No medical treatment may be initiated or continued without the patient's free and informed consent.* Informed consent is not merely a form signed by the participant (even though such a signature is required in clinical trials); rather, it is a procedure comprising a series of coordinated, sequential acts. Through this process, it must become evident that the participant has gradually reached the decision to take part in the research, exercising their right to dispose of their own body (e.g., Art. 5 of the Italian Civil Code), a right that is restricted when such acts cause a permanent impairment of physical integrity or otherwise violate the law, public order, or public morality. Consent must be free[13], informed, and revocable at any time

[12] e.g. in Italy: L. 22 dicembre 2017, n. 219, in particular Art. 1.
[13] For doubts regarding the freedom of human decisions, see references [39], [40], and [46].

(even while the research is underway). It must comply with EU Regulation 536/2014 (specifically Art. 29 and 30), Good Clinical Practice Guidelines, and the Declaration of Helsinki, while ensuring a risk↔benefit balance favoring the individual or public health. Furthermore, consent must be unequivocally documented[14] (for instance, via entries in medical records or outpatient charts, repeated information sessions, or any other means that serve the participant's interests). Compliance is also required with D.L. 211/2003 (implementing the former Directive 2001/20/EC) and Art. 2050 of the Italian Civil Code regarding liability for dangerous activities; there will always be those who consider hypnotism to be a dangerous activity (§ 1.6). In other countries, the underlying principle is everywhere the same (informed consent plus a prohibition against permanent or disproportionate harm), though with varying nuances. In the EU, the framework is harmonized by the aforementioned Regulation 536/2014 and the Oviedo Convention; informed consent is mandatory, and ethics committees play a central protective role. In the USA, where the Common Rule and Institutional Review Boards apply, there is a strong emphasis on individual autonomy and the assessment of minimal risk, alongside specific rules for vulnerable populations. In the United Kingdom, the Human *Tissue Act* and *Research Ethics Committees* mandate a rigorous approach to consent and risk proportionality. France and other continental countries have detailed regulations within their Public Health Codes, featuring strict ethical oversight and explicit limits on non-therapeutic risks. In summary, while the willingness to participate in research as a living subject is recognized across almost all Western legal systems, it is invariably contingent upon informed consent and the prohibition of causing permanent or disproportionate harm to physical integrity. In cases of doubt, a judge makes the decision based on the uncertain instrument of expert testimony [240,272].
As outlined above, drafting a research protocol that incorporates hypnosis is no small undertaking; it is best entrusted to expert researchers in the field, of whom there are few, who have already grappled with the inherent challenges of such work. The *written informed consent form* (which, while not exhaustive, is nonetheless mandatory) must be drafted to clearly demonstrate a commitment to participant protection and to emphasize the participant's voluntary decision to take part in the research solely for the purposes of personal enrichment and the advancement of scientific knowledge.
Few people realize that even seemingly minor research projects, such as undergraduate, specialization, or diploma theses (including those at private institutions), must comply with the aforementioned obligations if they involve human experimentation; it is not the scale of the experiment that matters, but the protection of the participant.

1.6 Incidents in Experimental Hypnosis

Experimental hypnosis carries less risk than clinical hypnosis because participants are selected with extreme care; unlike in clinical practice, there is no requirement to accept or even test just about anyone [86]. Research in the field of hypnosis consists predominantly, if not exclusively, of physiological experiments; consequently, participants are generally selected to be free from major psychiatric disorders – including latent ones [276, 297-300] – as well as major medical conditions. Thus, working with individuals suffering from overt psychosis (acute or chronic) or uncompensated affective disorders (such as bipolar disorder) – scenarios that might occur or be chosen in clinical practice [1] – is ruled out. Furthermore, the likelihood of encountering subjects with severe cluster A or B personality disorders (such as *borderline personality*) is minimized, as these individuals are carefully screened by psychiatrists, psychotherapists, or both. However, baseline functioning is not assessed in the same way; the essential element here is its complete preservation and maintenance, regardless of specific personality traits. In cases where experimental work involves patients, the subjects typically present with neurotic-type personalities or neurotic-range borderline states[15] (e.g., *character neurosis*); in such instances, subjects – who are already aware of their condition – can only benefit from the hypnotic state and the subsequent, cautious suggestions[16]. Traumas are generally brought to conscious awareness prior to the experiment (in a clinical setting, separate from the research team) or are incorporated into the experiment itself

[14] According to Art. 1 of the aforementioned L. 219/2017, *"time dedicated to communication between physician and patient constitutes time spent on care"*.
[15] That is, according to the most recent views, from individuals who have experienced trauma or disturbing conditioning. As a rule, individuals with obsessive-compulsive disorder are excluded, as they appear to respond less well to hypnotic procedures.
[16] These experiments on patients are led by researchers who are also psychiatrists or psychotherapists, while the others provide support.

with all due precautions, at which point the procedure also takes on the nature of a treatment[17]. Actually, such experiments are infrequent. Secondly, hypnosis experiments are invariably conducted not by a lone researcher but by a large team, creating a critical mass that serves as a safeguard against mishaps. Thirdly, Bioethics Committees, which must grant prior approval for the experiment and are already highly cautious by nature, exercise particular scrutiny regarding hypnosis experiments; this is due to the aura of suspicion that still surrounds this type of research, particularly within academic circles. Consequently, it often takes many months and numerous adjustments before a hypnosis experiment receives approval.
Nevertheless, experimental hypnosis, like any healthcare practice, is not without risk. Much depends on how the experiment is conducted[18]. It is strongly recommended that experimental hypnosis not be performed by anyone lacking the specific expertise or working outside of a team within a qualified institution.

1.7 The Research Staff

Hypnosis research must be conducted by a team possessing specialized training beyond what is required for clinical hypnosis practice. While researchers must obviously be expert hypnotists, that alone is not enough. Such research goes beyond merely inducing a neutral hypnotic state; it delves into the specific mechanisms of applied hypnosis. Consequently, each task demands particular expertise. When investigating the effects of hypnosis described later in this paper, *the authors already possessed years of academic and professional experience* in fields such as internal medicine, neurology, psychiatry, neuroimaging, pharmacology, anesthesiology, cardiology, autonomic reflex studies, blood transfusion management, and internal medicine ultrasonography, not necessarily all at once, but always available when required. Without this foundation, a study involving hypnosis is not only less safe but risks being futile from the outset. This will become clear later on. Furthermore, the team was always prepared and trained to address any psychological or physical issue by planning psychiatric, psychotherapeutic, and resuscitation interventions. Building a comprehensive hypnosis research team requires years of selection, testing, and refinement.
Each individual researcher bears *personal responsibility* for the success or failure of the experiment and is never merely a passive assistant; however, it is important to note that there is a principal investigator who bears *overall responsibility*, including legal and financial liability [63].

1.8 The Research Subjects (Participants)

What is required of a participant in scientific research involving hypnosis is that they: 1) be capable of providing valid informed consent (and generally be of legal age, barring research in specific fields subject to special rules); 2) possess sufficient intellectual and cultural depth to understand the nature of hypnosis (which also enhances hypnotizability); 3) possess enough empathy to establish a connection with the hypnotist *(rapport)*; 4) be willing to volunteer their time for the research solely in the interest of scientific knowledge; 5) be free from psychological conditions that would make the induction and conduct of hypnosis difficult or risky; and 6) be free from physical conditions or malformations that would make recording the effects of hypnosis impossible or even merely difficult. In cases where the use of certain brain imaging techniques is planned, it is also required that one be free from claustrophobia and willing to remain inside a narrow scanner bore for several hours[19].
Participants are often selected from among those who frequent the scientific laboratory, or from their friends or relatives. Given the great interest in hypnosis, it is never difficult to find volunteers even for studies on

[17] According to some, the mere elimination of the symptom is no longer viewed entirely unfavorably [12]; indeed, some [290] argue that the hypnotist, should actually limit themselves to removing the symptom. However, prudence suggests avoiding risks in a research procedure where professional liability insurance might be invalidated, and instead adhering to principles of caution.
[18] For example, in a research laboratory, hypnosis can easily be used both to elicit and to resolve clinically relevant phenomena, in order to study their characteristics in a controlled environment [63,90,110,191,207,210,247,248]. Here, the symptoms are deliberately induced rather than accidental, but the example serves to illustrate that this is possible.
[19] Those who are frightened by magnetic fields are not suitable candidates, even though, in another wing of the hospital, they pay to undergo magnetotherapy.

pain[20]. Alternatively, participants are recruited *via* notices posted near the laboratory or in university and hospital bulletins.
In many respects, the requirements for participants do not differ significantly from those expected of the researcher.
One interesting topic is the participant's level of hypnotizability. When a paper undergoes peer review, reviewers almost invariably ask whether the subjects were evenly distributed between those who are *highs* and those who are *lows*, or they object if only *highs* were used. In reality, if the study aims to evaluate an effect of hypnosis, it is natural to select only *highs*, as the effect is more pronounced in them[21]. For physiological studies, *highs* are preferably recruited. The only instance requiring the study of both *highs* and *lows* (ideally matched for age and sex) would be a hypothetical epidemiological study on the prevalence of hypnotizability in the general population, a scenario that has never arisen and does not appear plausible[22].
The ideal participant is a healthy, curious individual aged 18 to 50, the age range when hypnotizability seems to be at its peak and uncritical suggestibility at its lowest.

1.9 Assumptions About Hypnosis: the Narrative Tradition

Since the days of Mesmer [111] and the early psychoanalysts [169,170,211], hundreds of theoretical claims regarding hypnosis have been put forward; unsupported by controlled research, these claims have often proven unfounded and lack any authority other than the subjective view of the author in question. In the absence of scientific data, any content can be attributed to a theory, content that often simply *reflects the spirit of the times* [274] or *projects the author's inner world*. Even today, some believe that hypnosis is a magnetic phenomenon or involves the transfer of something[23] from the operator to the subject, that the operator rather than the subject is the focal point of the action[24], or that it is possible to induce someone to perform actions against their will[25] while under hypnosis. All these theories have been proven false, yet they continue to circulate and appear in books and at conferences. Until recently, much effort went into teaching what hypnosis *is not*; today, it is far more consistent to teach what hypnosis *is* (§ 2.1 to start, and the entire Part 2 to continue).
In narrative terms, hypnosis can be defined as a social interaction in which one person (designated as *subject* or *participant*) responds to suggestions offered by another person (designated as *hypnotist* or *operator*) to elicit experiences involving alterations in perception, memory, and voluntary action [163]. This definition is ambiguous [166]. Hypnosis can also be defined more broadly as the administration, self-administration, or experience of primary suggestions, independent of a *trance* induction [44,165,286]. According to narrative theories, hypnosis is distinguished from other suggestive phenomena (such as sensory suggestions, the placebo effect, or the misinformation effect) because it induces a change independently of variations in stimulation conditions.
Nowadays, there is no longer any need to dwell on outdated definitions of hypnosis formulated in the past, definitions that were appropriate for the times in which they were conceived but have often been superseded, if not refuted, by subsequent experimental evidence.

[20] Except for studies involving dental pain [119-121,124,127]; in these cases, the number of volunteers drops drastically, partly due to the profound significance of interventions on or within the oral cavity [281].
[21] Choosing *lows* in this case would be tantamount to studying swimming aptitude in subjects who do not know how to swim, a concept difficult to convey to those who are not researchers.
[22] The authors of this *Position Paper* who are also epidemiologists (E.C., V.T.) have conducted and published numerous population studies in other fields and would know how to organize a population-based survey on hypnosis; however, it is impossible to secure the funding required for such a highly expensive population-level project. Some studies use the term *population* incorrectly and misleadingly and are not actually population-based.
[23] It seems that for some, the theory of the *subtle or luminiferous ether* which posited the existence of an extremely tenuous, impalpable, and pervasive *fluid or material medium* serving to fill space and explain the transmission of forces or light has not yet been superseded, as if the advances in physics during the 19th and 20th centuries had never occurred [104,105] and as if Michelson and Morley had not already disproven its existence back in 1887.
[24] As if Braid had never existed [24,25].
[25] As if subjective free will, even outside of hypnosis, had not been refuted by scientific studies, in addition to being incompatible with the determinism of the naturalistic universe [39,46,146,178-180,254].

1.10 Limited Utility of Hypnotizability Scales

Hypnotizability scales [151], while perhaps useful in other contexts for identifying in advance which subjects should undergo hypnoanalysis or hypnotherapy, are of little or no use in experimental research. Moreover, in most cases, they are scales of suggestibility rather than hypnotizability [165,285]. Even when they do genuinely test hypnotizability [258-262], *they are essentially induction procedures* that merely attempt and quantify a preliminary induction; this is a task that can easily be carried out during the experiment itself, thereby saving time and human resources. The authors of this *Position Paper* therefore seek to avoid using them whenever possible and will not discuss them here.

1.11 Mental Models Underlying Hypnosis

This chapter 1.13 lies at the boundary between hypnosis understood in a descriptive sense and in an experimental sense. The scientific study of hypnosis cannot overlook a reflection on its epistemological dimension, which is inextricably intertwined with the ways in which human beings learn about, process, and interpret the surrounding reality [44-46]. Physical reality *in and of itself* is intrinsically inaccessible, since knowledge is inevitably mediated by receptors (§ 2.2) and the neurophysiological processes (§ 2.2 *et seq*.) triggered by them [35,42,73,161]. What is commonly defined as *perceived reality* is, in fact, the outcome of a unified subjective representation [137]. Hypnosis thus ceases to be viewed merely as a peripheral illusion or an artifact of simulation, emerging instead as *a manifestation of the fundamental dynamics of the nervous system*. Hypnosis experimentally demonstrates that *the entire experience of the world is an internal process*. By narrowing the focus of attention through plastic monoideism [72] (§ 2.5), hypnosis allows for profound alterations in perceptual, bodily, visceral, and temporal representations, endowing mental images with the same phenomenological, physiological, and biochemical status accorded to physical stimuli from the external world [44,75].
Since the neural matrix and representational reality are structured within a single representational space, hypnosis offers an ideal methodological tool for scientifically investigating how a focused idea can translate directly into receptor, immune, endocrine, and hemodynamic responses [64,73,137]. Building on these epistemological premises, some lnes of research in modern cognitive neuroscience has progressively moved away from the model of the brain as a passive receiver of *bottom-up information*, proposing instead a view of the brain as probabilistic and the mind as a *Bayesian predictor* [80,252]. Perception is fundamentally a controlled hallucination [134,251]: the cerebral cortex does not passively await stimulation from sensory organs but constantly generates in a *top-down* manner an internal model that anticipates the external causes of incoming sensory data. Perceptual processing thus takes the form of a dynamic, ongoing *process of probabilistic inference*. The mind calculates the probability of the world's hidden causes (the posterior distribution) by combining prior beliefs – formed through past experience – with the likelihood of current sensory data [251].
What an individual consciously perceives at any given moment is not a direct measure of physical input, but rather the working hypothesis projected by the brain's attentional spotlight[26] that minimizes the discrepancy with peripheral data [80]. Under ordinary conditions, the construction of internal reality follows a strict Darwinian logic: the brain continuously generates predictive conjectures about the world, and hallucination remains in check because the error signals (prediction errors) conveyed by the *bottom-up* stream serve to constantly refute, correct, or update the internal model[27].
Within this context, the *relational interaction (rapport)* between hypnotist and subject becomes an integral part of the mind's predictive architecture, enabling the guided reconfiguration of perceptual and existential models [137]. In accordance with Karl Friston's free energy theory [213], to counteract the natural tendency toward entropy and maintain structural integrity, every biological system constantly minimizes so-called *surprise* or free energy, i.e. the discrepancy between expectations generated by the internal model and signals conveyed *via* the sensory veil [213]. Hypnosis appears to interfere with *precision weighting*, which represents the estimate of reliability or informational value assigned to a specific transmission channel. When the

[26] It is the *searchlight theory*.
[27] It is the *theory of the extended mind* [81]: the boundaries of the cognitive system extend to encompass cultural artifacts, social relationships, and linguistic interactions.

environment sends a clear and stable signal, the brain assigns high weight to the bottom-up sensory prediction error, forcing the top-down internal model to update itself through perceptual inference. Conversely, if a sensory channel is deemed uncertain, noisy, or irrelevant, the brain drastically lowers its precision, *ignoring its input and allowing perception to be guided almost exclusively by the internal model's predictions*.
Hypnosis acts upon this *precision weighting* mechanism [159]. Through the induction process and the attainment of plastic monoideism (§ 2.5), the hypnotized subject narrows their attention to a single representation; this deep focus reduces the precision assigned to the array of external and peripheral sensory signals. In this state, verbal or symbolic suggestions provided by the hypnotist are adopted by the brain as a *hyper-prior*, a prediction of *extremely high priority and certainty*. From the perspective of neurofunctional connectivity, this entails a transient decoupling of the reality-monitoring function associated with the mPFC and dlPFC [173]. By deactivating analytical criticism, internal simulation is promoted (unfiltered) to the status of perceived reality. PET and fMRI studies have shown that when a blind person explores Braille text with their fingertips or performs spatial processing tasks, their primary visual cortex (V1) becomes intensely and structurally active [242], and that applying transcranial magnetic stimulation to temporarily disrupt V1 activity impairs their tactile Braille reading ability. V1 is not merely a passive screen designed solely to receive signals from the retina, but a processor of top-down spatial maps and representations. In line with this view, perceived reality is a flexible construct generated by the brain to minimize free energy, regardless of the sensory modality of origin[28].

[28] A blind person develops an internal spatial representation and places total trust in it precisely because they lack the stream of retinal visual signals that might refute or contradict it. Under hypnosis, the sighted subject's brain is induced into the same dynamic: sensory signals are neutralized by a reduction in precision, allowing the predictive model, triggered by suggestion, to populate cortical sensory areas with mental images possessing absolute spatial and perceptual consistency. Blindness and hypnosis thus represent two sides of the same coin, demonstrating that the mechanism generating internal reality functions consistently even in the absence of retinal stimulation; hypnosis bypasses reality testing, highlighting the complete autonomy of the representational world [137].

Part 2
Experimental Research in the Field of Hypnosis

2.1 What is Known About Hypnosis: Experimental Research

In modern terms, hypnosis can be defined as a *modified consciousness*[29] in which, due to the establishment of self-induced or hetero-induced plastic monoideism [72] (§ 2.5) and a privileged, reciprocal relationship between the operator and the subject (commonly referred to by professionals as *rapport* [249]), significant behavioral and somato-visceral changes become possible [42].

Alternatively, hypnosis is viewed as a specific *state* of the organism [44-47,137] that arises whenever specific emotional and sensorimotor stimuli intervene; it is a *"physiological and dynamic modification of subjective consciousness resulting from a connection formed by the combination of relationship and communication"* [74] between the person inducing and managing the hypnosis (the operator, hypnologist, or hypnotist) and the *participant* (the subject). Logic remains present during hypnosis; the lowering (though not the total suspension) of critical judgment allows for the acceptance of the meaning conveyed by both verbal and non-verbal communication [74]. These definitions – which remain highly relevant today, even though their seeds (although with some imprecisions) were already present in the works of 20th-century masters – represent the most up-to-date understanding of hypnosis that can be concisely articulated. They rely heavily on scientific evidence regarding the nature of hypnosis accumulated in recent years, much of which is described in detail later in this text. Two prominent researchers recently published an article examining hypnosis from a neurophysiological perspective [98]. They highlight how hypnosis emerges as *a multifaceted process shaped by individual susceptibility, the induction method, and the structure of suggestions, involving various neural pathways.*

The study of hypnosis has taught and continues to teach us a great deal about the nature of the mind, and the Ego in particular [47]. Marked reductions in mPCC and dACC activation are observed during neutral hypnosis. Hypnotic attentional absorption is associated with reduced activity in the default mode network and increased activity in the prefrontal system (§ 2.8); the bilateral dlPFC (executive control network) and the ipsilateral insula (salience network) are also implicated [97]. This is accompanied by reduced intrinsic connectivity within default mode network regions, a factor contributing to alterations in the sense of agency and consciousness [152].

There are, therefore, differing views regarding where hypnosis takes place. For instance, it is taken for granted, though never experimentally proven, that hypnosis represents a *modification of consciousness*. Although hypnosis is likely not confined solely to the brain [43], current answers can only be derived from brain imaging techniques. In the view of the authors of this *Position Paper*, once the induction phase (§ 2.3) has passed, imaging results for neutral hypnosis are rather disappointing or contradictory. Various studies have focused on hypnotizability (which is easier to measure) rather than hypnosis itself; depending on the methodologies employed, those that have addressed the question of where neutral hypnosis occurs have identified such a wide array of brain areas and regions as to suggest a nearly ubiquitous distribution, ultimately confirming the non-locality hypothesis [99,187,228,230,231].

The locus[30] of hypnotic activity can be identified only during a task, whatever that task may be. Under these circumstances, in *highs* in deep hypnosis, the prefrontal areas (specifically, though not exclusively, Brodmann areas 9, 25, 32, and 47), the precuneus, and parts of the cerebellum are activated (Figure 1).

[29] For the difference between *consciousness* and the *sensorium*, see Table 2. Throughout the *Position Paper*, the term used is (egoic) *consciousness*, never (moral) *conscience*.

[30] As previously mentioned, it is inappropriate to speak of a specific location, given that hypnosis is likely a dynamic and non-local function, much like the Ego [43].

Table 3. Sensorium, consciousness and arousal [35,42].

Functions	Description
sensoriun	the ability to perceive and integrate events through the use of attention and memory; it arises from the coordinated activity of diverse and varied nervous functions. Typical of animals and relevant to humans as animals
consciousness	the ability to respond to external stimuli while having a qualitative internal experience of existence. It is acquired over time through culture
arousal	a temporary state of heightened alertness and alertness evoked by a prompt reaction to a stimulus; a concept of exclusive interest to psychologists; in animals, it pertains only to the sensorium; in humans, it pertains to both the sensorium and consciousness, thus also taking on cognitive connotations

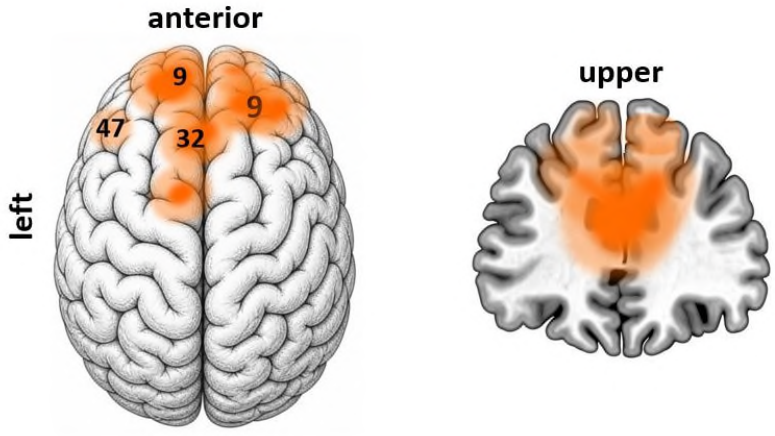


Figure 1. Some of the areas activated during hypnosis while performing a task, shown in two brain sections *via* 1.5 T fMRI [73].

2.2 The Neuropharmacological Bases of Hypnosis

The authors of this *Position Paper* – particularly those who are also cardiologists, internists, neurologists, pharmacologists, or anesthesiologists – are fully aware that any effect of hypnosis necessarily occurs through the action of ligands[31] on receptor structures [75]. Indeed, any state or change of state in an organism is inevitably due to a receptor-mediated effect; no other mechanisms of action are known. This is obviously true for hypnotic effects as well, whether physical or psychological.
Schools of thought maintaining that hypnotic effects (especially psychological ones) are merely *as if* phenomena express a narrative opinion that cannot be proven; science cannot explain the *as if* aspect, whereas it can attribute every known hypnotic phenomenon to the action of known ligands. In the case of hypnosis, where no pharmacological ligand is administered externally, the ligands involved are endogenous ones, produced within the body in response to external stimuli (fear, anger, anxiety, calmness, arousal, meditation, introspection, etc.) or, as studied in the field of psycho-neuro-endocrino-pharmacology, to mental imagery [75,112]. Mental images[32] are powerful stimuli that trigger biological consequences ranging from sensations and emotions to

[31] Receptors are glyco-proteic, nucleic, or lipid structures capable of specifically recognizing and binding a molecule known as a ligand; upon interacting with a ligand, they undergo a conformational change that triggers a biological or pharmacological response. Natural or synthetic ligands (which can act as agonists or antagonists) are molecules capable of binding to specific receptors and altering their structure, thereby producing specific effects [75]. This applies not only to higher animals but also to lower animals, plants, fungi, lichens, and molds. The presence in higher organisms, including *Homo sapiens*, of specific receptors that respond to animal and plant ligands [75] is attributable to the incorporation of receptors from lower organisms during the course of evolution.
[32] It is quite logical to assert that mental images are the only knowable aspect of reality [137], a topic that cannot be treated at length here.

thoughts and physical changes. By way of example, in daily life as well as in dreams [30], the mental image of a fanged beast raises heart rate and blood catecholamine levels, while an erotic mental image triggers an erection, and so on. This power of mental imagery holds true in all cases, but is vastly amplified when the mental image is generated or suggested under hypnosis.
Exogenous ligands each elicit one or more specific effects ranging from anxiolysis[33] (§ 2.7, 2.10), to sedation (§ 2.7, 2.10), to analgesia and anesthesia (§ 2.8-2.10) to hypermnesia [236] (§ 2.19), to revivification (§ 2.19), to amnesia (§ 2.10), to enhanced performance (§ 2.14), etc., yet they do so in a somewhat chaotic, haphazard manner, consistent with the traits selected by Darwinian evolution over millions of years. The same – likewise in a disorderly fashion – occurs through the action of thought involving the same ligands, though in this instance, they are endogenous. In hypnosis, the endogenous ligands released whether by the hypnotic state itself or by targeted suggestions produce the same effects, yet in a manner that is *more coordinated, purposeful, and focused*; this explains the high efficacy of hypnosis *per se*, and especially of suggestions administered during and after the hypnotic session. It is commonly said that the *rapport* resonates at a psychosomatic level, that is, it has physical effects which is tantamount to acknowledging that thought activity (now demonstrable *via* neuroimaging) activates endogenous receptor systems. These biological concepts are of great interest to the researcher who investigates them to gain insight into the *what*, *where*, *how*, and *who* regarding hypnotic phenomenology [36,37,42].
The endogenous ligands plausibly implicated in the specific effects of hypnosis will be discussed below.

2.3 Hypnotic Induction

The phase[34] that, within a structured setting, deliberately initiates the emergence of plastic monoideism (§ 2.5) – that is, hypnosis – is termed *induction* [273]. Since the set of practices implemented in a structured setting to achieve hetero-hypnosis is referred to as *hypnotism*, it can be stated that induction is synonymous with hypnotism.
The term *techniques* refers to the measures sometimes implemented to achieve hypnotic induction within a structured setting. In reality, these are unnecessary (except perhaps for novices or practitioners lacking empathy) and often result in a waste of time that could be more productively spent managing the hypnosis itself. While treatises and manuals rightly emphasize that hypnosis is fundamentally a *rapport*, they often go on to devote dozens of pages to techniques; some even classify hypnosis itself as a technique [28], which is a clear contradiction. Induction does not occur in self-hypnosis [44] and is not strictly necessary even in a structured setting [31], although explicitly applying it can sometimes help save time; the Erickson School has addressed this topic [113-115].
From an EEG perspective, slow induction is characterized by increased activity in the right hemisphere compared to the left, whereas rapid induction without relaxation involves hyperactivity in both hemispheres (particularly the right) and the precuneus (Figure 2). From a cardiovascular standpoint, rapid induction triggers a substantial and significant increase in peripheral resistance, a response absent in slow induction (Figure 3). Slow and rapid inductions are therefore vastly different procedures, each with a distinct phenomenological profile [62]. In the past, only slow inductions (procedures that were lengthy and frankly tedious) were used. Today, the trend favors rapid, and sometimes ultra-rapid or instantaneous, inductions [235].

2.4 Depth of Hypnosis

Some Schools of thought hold that the depth of hypnosis does not exist or cannot be measured, while others maintain there is no need to do so [74].
Obviously, the concept of *depth* is merely a metaphor, here. Nevertheless, the use of certain methods employed in pharmacological general anesthesia to assess the depth of narcosis, such as BIS and CSI, has proven useful

[33] Prehistoric humans were more familiar with fear; modern humans are more familiar with anxiety. In the Paleo-Neolithic era, upon encountering a predator, the hominid felt under attack. Today, that prehistoric predator has not vanished; it still lurks within our emotional system: this is the underlying reason why we describe ourselves as anxious. Fear (the cascade of physiological phenomena triggered by an actual attack) has migrated into the unconscious, transforming into the *premonition of an event that might occur*. Anxiety is a state of anticipation regarding a potential future fear, keeping the defense system constantly on alert [281].
[34] Which can also be carried out remotely [70].

[162,216,295]. However, it must be borne in mind that working with hypnosis does not involve the *narcotized vs. awake* dichotomy, as a person under hypnosis is never asleep: their sensory system remains alert and their consciousness remains present, albeit physiologically modified. Sensorium and consciousness are distinct functions (Table 3).

The authors of this *Position Paper* do not consider it useful to employ the aforementioned indices during experiments involving hypnotizability scales; nor do they deem it anything other than useless and misleading to ask participants to rate their own degree of hypnotizability, particularly when using visual-analogue scales [295] (§ 2.10).

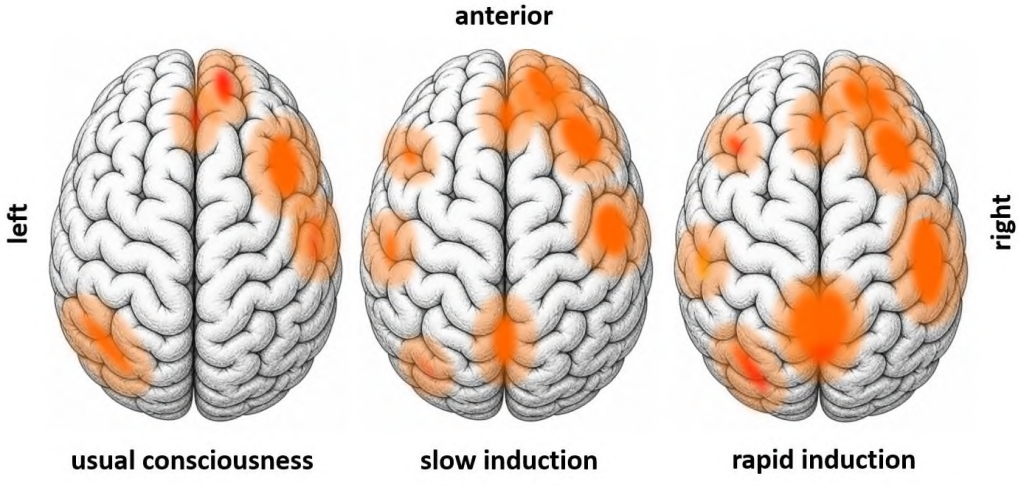


Figure 2. Brain imaging obtained during ordinary consciousness and during slow and rapid hypnotic induction using EEG-LORETA: during induction, particularly in *rapid induction*, the right hemisphere and the precuneus show significant activation [62].

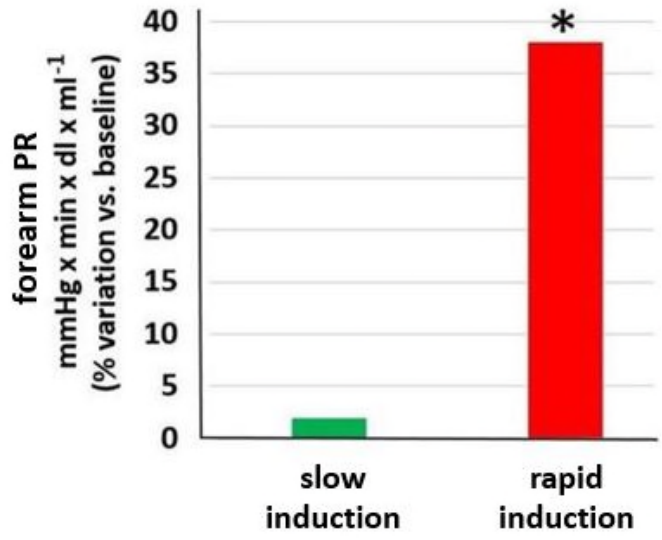


Figure 3. During rapid induction, PR (an index of stress) increases significantly (* $p<0.01$) compared to baseline, whereas this does not occur during slow induction.

2.5 Plastic Monoideism and its Experimental Demonstration

The authors of this *Position Paper* consider plastic (or ideo-plastic) monoideism to be the trigger for hypnosis, if not hypnosis itself [35].

For decades, plastic monoideism was merely hypothesized and treated as *dogma* by the Schools that adopted it, or taken for granted (as implicit in the therapeutic relationship) by other schools, or deemed either unprovable or unnecessary to prove [74]. The ontological existence of plastic monoideism as a distinct phenomenon has recently been demonstrated by showing that, during a task, fMRI reveals the activation of brain areas characteristic of the hypnotic state (anterior regions, prefrontal cortex, precuneus, cerebellum) alongside the deactivation of any other regions previously engaged in the task (Figure 4). The plasticity of monoideism is evidenced by the fact that the hypnotic task, regardless of its nature, is executed precisely as suggested [72].

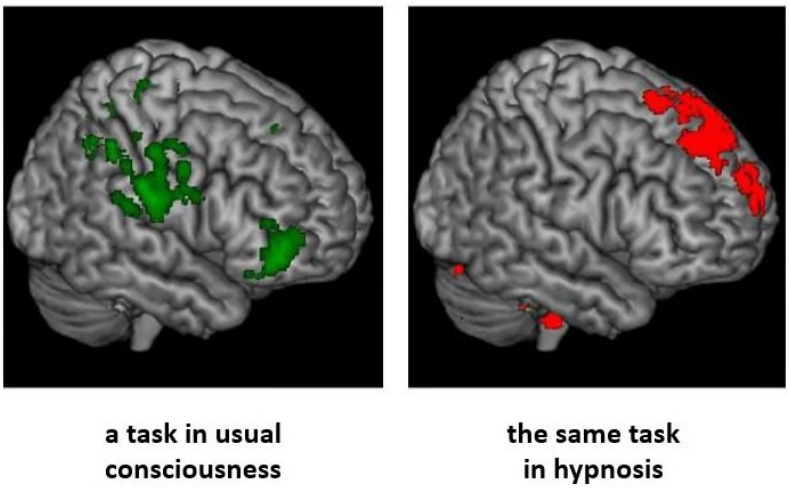


Figure 4. Demonstration of plastic monoideism using 1.5 T fMRI. Green indicates areas activated during a task in 20 highly hypnotizable volunteers. Red indicates areas activated during the same task in the same subjects following an appropriate hypnotic command (prefrontal and posterior regions, precuneus, and cerebellum). The areas do not overlap, as plastic monoideism excluded the areas not involved in the command [72].

2.6 The Deliberate Acceptance of Hypnosis

Every manual maintains that hypnotic *trance* differs from *suggestion*[36] in that, the former entails the critical acceptance of another person's ideas, whereas the latter calls for uncritical acceptance. But is this claim true? Or is it based merely on anecdotal evidence? [217]. Or is it, in fact, nothing more than a wish bordering on illusion? Evidence that hypnosis is deliberately accepted by the subject has recently come from neuroimaging. Indeed, during hypnosis, brain areas closely linked to what is popularly termed *conscious freedom*[37] become hyper-activated (Figure 5), while other areas are rendered silent [68]. It must be noted that this demonstration

[35] Various authors have gone to great lengths to describe and classify the unconscious in a multitude of ways—an undertaking that would be both impossible and pointless to summarize here. The reader is referred to the original sources [20,92,93,130,131,149,160].

[36] Then, once the hypnotic state has been established, it is possible (and indeed useful in clinical, therapeutic, educational, or experimental settings) to administer suggestions often termed *commands*. These are, in fact, forms of conditioning [275] capable of operating even in the post-hypnotic phase. While the subject accepts these suggestions if they align with their principles and become an integral or even fundamental part of the hypnotic session, they do not constitute hypnosis itself.

[37] Previously, in publications and lectures from years past, the authors of this *Position Paper* maintained that hypnosis is *accepted consciously and voluntarily* [68]; today, they prefer to state that it is *accepted deliberately*, given that the concept of conscious freedom has been seriously challenged not only in hypnosis but in everyday life [39,40].

is possible only during the performance of a task, not during neutral hypnosis: it is the task that, by activating the choice model, makes it possible to highlight the deliberate acceptance of the suggestion (or hypnotic command).

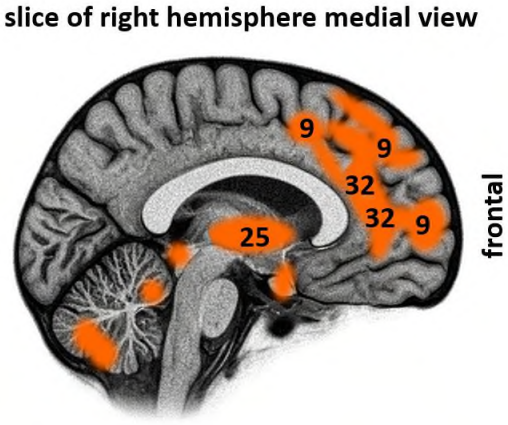


Figure 5. fMRI image. The areas affected by hypnosis are those considered to be involved in conscious will.

2.7 Anxiolysis and Sedation

The anxiolysis and sedation accompanying neutral hypnosis – and to an even greater extent those achieved through appropriate commands – are entirely receptor-mediated phenomena.
Apart from ethanol[38], the first truly effective substances used as anxiolytics were likely opiates, which are primarily agonists of central μ (MOP) and δ (DOP) opioid receptors [15,96,132,176,225,279]. Due to their strong tendency to induce tolerance, dependence, and even fatal respiratory arrest, starting in 1903 opiates were partially replaced in human Medicine by $GABA_A$ receptor agonists known as barbiturates[39] [17] which act by binding to central $GABA_A$ ionophore receptors, thereby opening chloride channels [116]. In 1957, chlordiazepoxide [284] was introduced into clinical practice; from 1963 onwards, it paved the way for other, much more convenient $GABA_A$-ergic agonists (MBZDP[40]) much easier to handle and safer[41].
The μ-receptor agonism is anxiolytic and sedative, and GABA is the primary inhibitor of glutamate's excitatory effects. Agonism at μ, δ, and $GABA_A$ receptors produces fairly similar effects *in vivo*; consequently, opioids, barbitura-tes, and MBDZP share potent anxiolytic and sedative actions at low doses, and narcotic effects at higher doses. Receptor studies indicate that anxiolysis (a $GABA_{A\text{-}\alpha2}$ effect predominant in the hippocampus) [202] and more intense sedation ($GABA_{A\text{-}\alpha1}$ agonism predominant in the thalamus and cerebellum), while often treated as synonyms by the general public, are biochemical[42] and hypnotic distinct phenomena [18,129].
It is now evident that the endorphinergic, endopioid, and endozepine actions triggered by stimuli originating in the cerebral cortex entail measurable phenomena: in self-hypnosis, and to an even greater extent in hetero-induced hypnosis, the anxiolytic and sedative effects of these endogenous systems are simply far more intense,

[38] A large central depression, the use of which dates back to at least 9000 BC.
[39] Not exempt, although to a lesser extent, from the same drawbacks.
[40] In addition to their use in surgical anesthesia and conscious sedation [89] – discussed later – benzodiazepines have been used for over half a century as sedatives, anxiolytics, and sleep aids in the home setting, including as a form of tolerated self-medication; given the issues of generalized anxiety and insomnia affecting a large portion of humanity [150], they are likely the best-selling drugs worldwide for enhancing one's sense of tranquility, now that opioids and barbiturates are strictly controlled.
[41] MBDZP [202] also induce tolerance and dependence, but lethal dose is vastly higher; consequently, respiratory arrest caused by MBDZP, at least *via* the oral route, is unlikely (though not impossible), even in cases of error or attempted suicide [48].
[42] Endogenous activation of μ and $GABA_{A\alpha1/\alpha2}$ receptors explains anxiolysis and sedation characteristic of prayer [6,190,264], yoga [265], autogenic training [172], massage [227], and meditation, practices that involve a form of unintentional hypnosis or hetero-hypnosis accompanied by the recruitment of the aforementioned receptors.

thanks to the extremely high level of concentration on the mental image and the plastic monoideism[43] [96,127]. The mental images that activate these endogenous systems and the associated ligand↔receptor interactions during hypnosis consist of simple suggestions such as *be relaxed*, *let go of your anxieties*, etc. Due to the combined agonism at μ, δ, and $GABA_A$ receptors, a significant degree of anxiolysis invariably occurs during any hypnosis session – even in the absence of chemical agonists – as a spontaneous effect that can be readily amplified through appropriate hypnotic and post-hypnotic suggestions [37].
The anxiolytic and sedative nature of hypnosis stems from this agonism at receptors [36]; since no exogenous drugs are administered, this effect is attributable to the activation (well-documented in the literature) of endogenous opioid (endorphin/endopioid), $GABA_A$-ergic (endozepine), and adenosine systems [193,194,246]. Conseque-ntly, research into the sedative and anxiolytic properties of hypnosis will focus primarily on μ, $GABA_A$, and A_2 receptors and their corresponding endogenous agonists, although it is currently unknown which of these play the most significant role. Just as chemical receptor-mediated effects are governed by specific pharmacodynamics and pharmacokinetics [50], the same applies to the receptor-mediated effects of hypnosis; however, in the latter case, both the *pseudo-dynamics* and *pseudo-kinetics* of the effect are largely independent of the organism itself and are more amenable to control.
Furthermore, just as there are specific antagonists for opioids and MBDZP[44] that terminate the effects of those drugs, there exists the possibility (indeed, the certainty) of halting endorphin, endopioid, and endozepine effects at any time through hypnotic suggestions; this represents another receptor-mediated effect of hypnosis, with the distinction that naloxone and flumazenil have short elimination half-lives (shorter than those of their agonists) and often require repeated administration, whereas hypnotic commands remain effective for the duration determined by the practitioner.

2.8 Local Analgesia Using the *Be Analgesic* Command

Hypnotic analgesia for acute pain is one of the most frequently discussed topics, particularly in the form of HFA. For anyone practicing clinical hypnosis at a high level, it has long been beyond doubt that HFA develops following a simple hypnotic command for analgesia. Until recently, it remained to be determined whether HFA was due to mere dissociation[45] or to an actual blockade of the pain signal's progression from the periphery to the brain. Both hypotheses were plausible in a narrative context, but in the scientific realm, the definitive answer could only come from brain imaging. Even defining pain as an objective event was difficult[46].
The issue was recently resolved using fMRI [3,73]. Volunteer participants were subjected to a painful stimulus while inside the fMRI scanner – both during ordinary consciousness and after receiving a hypnotic command not to feel pain – using a Latin square design in which each participant served as their own control. As shown in Figure 6 (left), the painful stimulus during ordinary consciousness triggers activation in the sensory areas (S1, S2, S3), as predicted by physiology; this demonstrates that the pain signal reached the brain and was consequently perceived as pain. Conversely, when the painful stimulus was administered under HFA conditions (Figure 6, right), resulting in a reduction of subjective pain perception by 68% ($p<0.01$) and 220% ($p<0.0001$) [51-53,56,58,59,63,119-121,124], the sensory areas remained silent. Instead, the brain activity pattern was dominated by the massive prefrontal hyper-activation characteristic of hypnosis during any given task [68]. The HFA hypnotic command persisting into the post-hypnotic state is therefore attributable not to dissociation, but to an actual blockade of the pain stimulus at some level of the ascending sensory pathway.
Cardiovascular methods were used to determine whether this blockade was cortical or subcortical. The authors of this *Position Paper* were well aware – based on direct knowledge of arterial circulatory physiology in various situations [36,51-54,58,59,63] – that systemic pain triggers an autonomic response characterized by increased total PR (calculated from cardiac output) and regional PR (calculated from segmental flow, e.g., in the forearm or leg). This reflex is involuntary and originates in lower subcortical structures [153]. By assessing

[43] Although they never lead to sleep, which, after all, would interrupt the hypnotic trance.
[44] Respectively, naloxone and flumazenil [48].
[45] There is an Ego that feels pain and another Ego that, when asked, declares it does not feel it.
[46] The official definition of pain [218, 219] formulated by the IASP comprises three elements: 1) it is associated with actual or potential tissue damage, 2) it is an unpleasant emotional experience, and 3) it is subjective. It was once said that *"since pain is purely a subjective experience, it poses great difficulty for scientific investigation"* [77]. But, as this section shows, that view was mistaken.

peripheral resistance during painful stimulation in both the normal conscious state and under HFA (Figure 7, left), it was observed that the sympathetic cardiovascular response was markedly attenuated, and in many cases abolished, when HFA was active (§ 2.8, 2.9, 2.10).

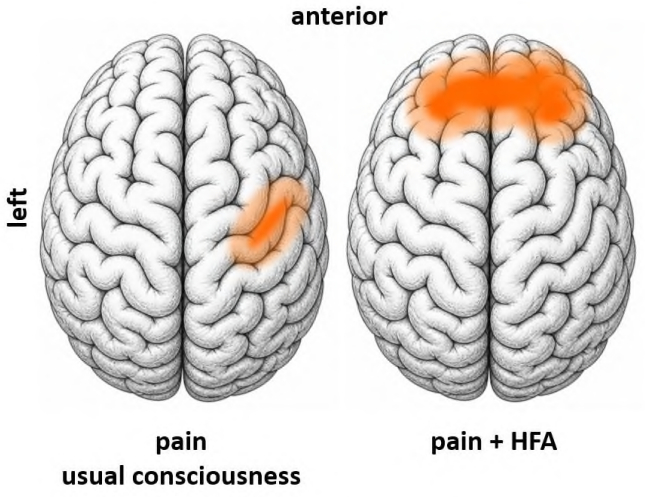


Figure 6. Administration of a painful stimulus to the left hand in 20 highly hypnotizable volunteers. Under conditions of normal consciousness (without hypnosis), primary sensory areas are activated. If the same stimulus is administered to the same subjects during an HFA command, these areas are not activated, and the pattern is dominated by massive activation of the prefrontal areas [73].

The situation differs for trigeminal pain (specifically pain involving the maxillofacial complex: teeth, gums, jaws, nose, paranasal sinuses, palate, tongue, oral mucosa, and facial bones): trigeminal pain entails a reduction in peripheral resistance due to vagal activation, rather than an increase due to sympathetic activation. Controlled studies have demonstrated that the drop in peripheral resistance resulting from trigeminal pain is attenuated and, in many cases, abolished by the hypnotic analgesic command (Figure 7, right) [119-121,125]. This further demonstrates that the sensory blockade abolished the autonomic response regardless of the nature of the pain and operated at a subcortical level.

The reduction in the autonomic response to pain is real and prevents damage caused by increased PR in systemic pain (acute hypertension, cardiovascular damage, etc.) and by decreased PR in trigeminal pain (acute hypotension, lipothymia, syncope, etc.). How is the blockade of the centripetal progression of the pain stimulus achieved in HFA? From a neurophysiological perspective, studies have highlighted the existence of *top-down* fibers with inhibitory activity originating in the pregenual cingulate, dorsolateral prefrontal, anterior insular, and anterior cingulate cortices, as well as in the hypothalamus and amygdala, capable of inhibiting nociceptive conduction in the spinal cord [193,194,212]. This explains the functional uncoupling between peripheral nociceptors and the cerebral cortex. Indeed, since these fibers originate in the brain, they respond to hypnotic suggestions just as they do to ongoing thoughts or distraction techniques; however, the response is far more effective under hypnosis. De Pascalis and De Benedittis [98] note that effects on SERP vary depending on the experimental paradigm, whereas EEG time-frequency analyses and studies on γ-band oscillations serve as markers capable of distinguishing hypnosis from distraction [98]. According to these authors, HFA involves distributed brain networks (ACC and MCC, thalamus, primary sensory cortex, dorsolateral prefrontal cortex, insula, and PAG). Functional connectivity analyses reveal enhanced interactions between the ACC, PAG, and insula during preparatory suggestion phases compared to a control condition. At the receptor level, HFA acts in every respect like local anesthetics, suggesting that it blocks sodium channels in the same way as lidocaine.

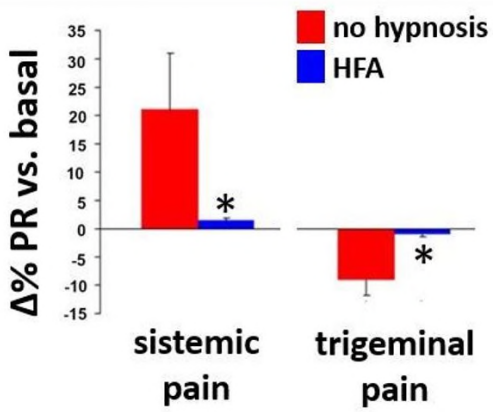


Figure 7. Percent change relative to baseline in forearm PRs during non-trigeminal systemic pain in the hand (systemic pain induced by ice-water immersion) and during trigeminal dental pain (trigeminal pain induced by trains of square-wave electrical pulses from a pulp tester). Non-trigeminal pain produces a >20% increase in PRs, which is reduced to ~2% under HFA conditions [63,64,69,71]. Trigeminal pain produces a ~9% reduction in PR, which decreases to ~1% in the presence of HFA [119,120,121,125]. HFA thus prevents the autonomic PR response to pain regardless of the pain type demonstrating that the blockade of the nociceptive stimulus occurs at a subcortical level [153].

This is not surprising, given that local anesthetics are known to act centrally as antagonists of nACh receptors, which are also implicated in analgesia induced by volatile anesthetics[47]. Cocaine[48] [11,48] is also a potent local anesthetic and was, for a long time, the only one available. Cocaine acts primarily as a DAT and NET antagonist [36]. The μ-endorphin system exerts an analgesic effect and is also spontaneously activated whenever necessary (for instance, in cases of intense acute pain), producing physiological effects identical to those of exogenous opioids used therapeutically. Various experimental findings indicate that the administration of naloxone (a competitive antagonist of the μ-mediated effects of opioids) prevents or abolishes the morphine-like analgesic effects of hypnosis[49] [131,133,294]. Hypnosis-induced μ-receptor activation is far more effective and targeted than spontaneous activation [42,75].
It is interesting to note that, despite this incontrovertible scientific evidence, the dissociative theory remains the most widely cited and accepted, particularly among psychologists. Dissociation is certainly present in hypnosis [277], but it is not the cause of the analgesia. In 2010, Piché et al. stated that *"determining the functional role(s) of pain-activated brain regions remains challenging"*; today, thanks in part to HFA, neuroimaging and hemodynamic studies, much more is known about this.

2.9 Local Analgesia Using Body Dysmorphism

Producing body dysmorphism is extremely easy during deep hypnosis, for instance using commands such as *your right hand is detached from your arm; I am taking it with me to the laboratory to work on it*. The same applies to trigeminal areas, though this requires great precision (e.g., *your mouth, tongue, palate, teeth, gums, etc., are detached from your body; I am taking them with me to the laboratory to work on them*). Consequently, any painful stimulus applied to these dysmorphic areas will fail to activate S1, S2, S3; it is neither perceived as painful nor does it elicit an autonomic response (Figure 8) [69].

[47] Which was once erroneously considered non-specific, that is, non-receptor-mediated.
[48] From this, lidocaine was obtained in 1943 and subsequently marketed in 1948. Subsequent anesthetic alkaloids, even though not of the tropane class, retained the suffix *-caine* by analogy [75].
[49] This finding is not shared by some researchers who unsuccessfully attempted to reduce only hypnotic analgesia using naloxone (see below); according to Barber, this is because hypnotic pain control is more complex and widespread than commonly believed [15]. In particular, pharmacogenetic studies have shown that G-carrier subjects for the *Asn40Asp* polymorphism (A118G, rs1799971) of the OPRM1 gene (a splice variant of the μ-receptor gene encoding μ_1 opioid receptors) are less responsive to exogenous opiates/opioids and endogenous opioids [225], thereby defining a subpopulation that responds less effectively not only to endorphins but also to the opioid-mediated effects of hypnosis.

Dysmorphism has been employed to induce HFA under the general label of *depersonalization* [Röder et al., 2007], a much broader phenomenon that invariably accompanies hypnosis. The analgesic mechanism underlying depersonalization is identical to the one described here as *dysmorphism* to explain the lack of activation in somatosensory areas; however, Röder et al. link this condition to the neural mechanisms driving OBE [2,22,126], an interpretation that seems less than objective when applied to HFA and is, in any case, unnecessary to explain the phenomenon.
The efficacy of dysmorphism-based analgesia is extremely high [69] (absolute in *highs*); for years, the authors of this *Position Paper* have used this method exclusively in clinical and experimental settings, reserving HFA solely for educational contexts[50].

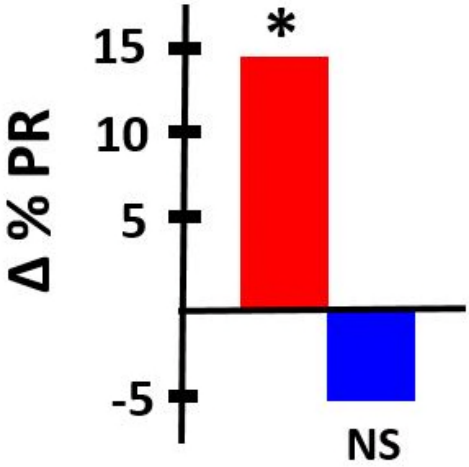


Figure 8. Percentage changes in PR during systemic pain (hand immersed for 1 min in melting ice at 0°C) under normal conscious conditions (no hypnosis ■) and during body dysmorphism (■). In the absence of dysmorphism, a significant increase in PR is observed; conversely, during dysmorphism (the hand is detached from the body), PR decreases due to the absence of an autonomic response and hypnotic relaxation. *p<0.01 vs. baseline values [69].

2.10 Hypnotic General Anesthesia

HGA[51] can be easily obtained by extending HFA to the whole body (§. 2.8, 2.9), which can be done in a single step, that is, with a single suggestion [64]. However, this is merely the first phase. In pharmacological general anesthesia for surgical purposes, the prevention of pain is one of the required outcomes. Also necessary are pre-operative anxiolysis and sedation (achieved with MBDZP), narcosis (achieved with barbiturates), flaccid paralysis (achieved with curare-like agents and necessitating intubation, as the respiratory muscles are also paralyzed), and retrograde amnesia (achieved with MBDZP) (§ 2.7). These measures aim not only to prevent surgical suffering but also to ensure immobility and protect target organs from physical and psychological stress.
HGA is obtained without drugs: anxiolysis stems from endogenous agonism at hippocampal $GABA_{A\alpha 2}$ receptors, analgesia is pursued through the extension of HFA (§ 2.10), pseudo-narcosis is induced by creating a state of indifference that is sometimes erroneously termed *hypnotic sleep*[52], flaccid paralysis *via* specific commands (§. 2.13), and retrograde amnesia simply by suggesting it[53]. The effects, including those on target organs, are exactly the same as those of pharmacological anesthesia (Figure 9) [64,237,238,241].

[50] The technique known as *hypnopraxia* is not discussed here, as it is not of specific experimental interest [103].
[51] HGA is always useful, but particularly so in burn therapy and in painful or uncomfortable diagnostic procedures, such as biopsies and endoscopies [85]. Furthermore, it offers the advantage of a conscious, communicative patient during the procedure, a highly beneficial factor when removing neoplasms from eloquent areas [88].
[52] Obviously, the EEG is not that of sleep.
[53] Many individuals in a high-hypnotic state exhibit amnesia even without suggestion (they were once referred to as *somnambulistic*).

Regarding hypnotic pseudo-narcosis with a preserved sensorium (Table 3) which may also be accompanied by profound sedation and an altered or absent perception of subjective time, it presents a physical phenomenology [282] entirely similar to the narcosis observed in pharmacology following the administration of barbiturates or opiates (μ, $GABA_A$, and δ agonists) [75]. The mental imagery administered during the hypnotic *trance* in this instance follows the pattern: *you will now fall into a very deep, pleasant sleep and will not emerge from it until I ask you to do so*. This is sufficient to produce a pseudo-sleep mediated by endogenous factors (characterized by EEG waves that differ from those of natural sleep) analogous to pharmacological narcosis. Some effectively regard this form of hypnotic anesthesia as equivalent to *deep conscious sedation* (§ 2.7) [197] and limit it to minor procedures, usually performed on an outpatient basis[54]. Others, distrusting hypnosis, also administer drugs, though these are unnecessary. While there are no controlled comparative studies, it is generally assumed that poor chemical anesthesia – involving anxiety, pain, nightmares, premature or disturbed awakening, etc. – is more likely to occur (given the ever-present possibility of error) than poor hypnotic anesthesia.

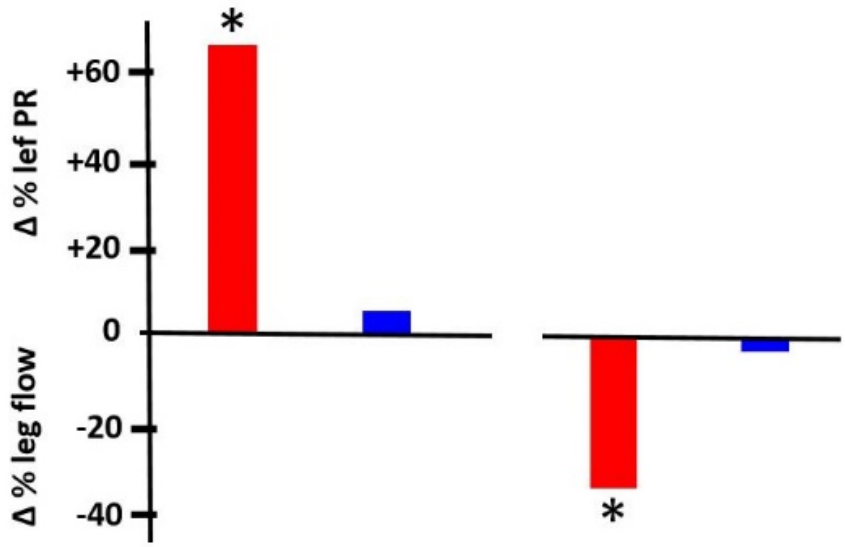


Figure 9. Hemodynamic parameters recorded in usual consciousness (no hypnosis (■) and during HGA (■); % variation maximal pain vs. basal conditions are shown.

2.11 Painful or Uncomfortable Procedures

This topic is only briefly touched upon here because this Position Paper focuses on experimental rather than clinical hypnosis[55], and very few studies have addressed this subject with experimental rigor, for instance by strictly avoiding the confounding of hypnotic and pharmacological measures [122,127] and by refraining from treating hypnosis merely as a complementary measure.

Since they can readily produce anxiolysis, sedation (§ 2.7), HFA (§ 2.8, 2.9), and HGA (§ 2.10), appropriate hypnotic commands can be employed during painful or uncomfortable diagnostic or therapeutic procedures such as burn treatment [95], digestive endoscopies (including operative ones) (§ 2.16) [198-201], colonoscopies [107,198], biopsies [7,16,177,243,250], etc. in both inpatient settings (where they are far less costly than pharmacological methods) and outpatient settings (where they are better tolerated and allow for immediate return home by any mode of transportation, free from residual drug-induced impairments on sensory perception and alertness) (Table 3).

[54] This is particularly the case for dentists, who do not go beyond conscious sedation [89,292].
[55] Clinical hypnosis has been treated elsewhere [36,37,42].

Surgical procedures both minor [122,127] and major [204,205,220-222] can be performed using only HFA or HGA, with the added benefits of anxiolysis and sedation. However, the hypnotists assisting surgeons (or surgeon-hypnotists) usually prefer to supplement this with pharmacological sedation or low doses of anesthetic [291]. In the opinion of the authors of this *Position Paper* – based on experimental results (§ 2.8-2.10) – this is not necessary, although in a clinical setting (where absolute research-level rigor is not required), hybrid procedures may sometimes make sense; for example, to save time and reassure the patient, and often the operator as well.

2.12 Resolution of Migraine

This is a topic that has received little experimental attention. A recent review [33] examined controlled trials regarding migraine treatment using hypnosis (though not always as the sole approach). The review's methodological rigor is evidenced by the selection of only 9 studies out of the 1,082 identified *via* search engines, involving a total of 247 patients. Nevertheless the interventions were highly heterogeneous (ranging from 2 to 24 weeks in duration, 3 to 21 sessions, 1 to 7 sessions per week, and 30 to 60 minutes per session, etc.), yet the review represents the most robust experimental evidence currently available. Following the initial session, three studies encouraged the use of self-hypnosis [44]. Results were heterogeneous too. Three of the examined studies showed no beneficial effects on migraine attack frequency, whereas others did, particularly in *highs*. Two studies observed a reduction in attack intensity, while one reported remission. Medication use sometimes decreased. These differences, along with others noted in the paper, may stem from the varying hypnotic suggestions employed; these ranged from enhancing internal resources, relaxation, conflict management, and ego strengthening to reducing anxiety and stress, boosting motivation, and – at times – direct symptom control.
The authors of this *Position Paper* have consistently achieved immediate resolution of all forms of migraine or headache in *highs* through direct suggestions of analgesia.

2.13 Hypnotic Paralysis

As any hypnotist knows, it is very easy to induce flaccid paralysis in a hypnotized subject. The paralysis has been experimentally confirmed and measured (in μV) using concentric bipolar single-needle electromyography, during hypnotic suggestions of paralysis of the striated muscles [49,154] with commands such as *this part of your body is paralyzed and limp*.
The resulting paralytic effect is, in most cases, total [49]. Experimental demonstration of the procedure's efficacy was obtained by hypnotically paralyzing the *corrugator supercilii* muscle of the forehead[56] and measuring its contractile capacity *via* electromyography under conditions of ordinary consciousness and during a hypnotic command of paralysis; the hypnotic paralysis proved as effective as the injection of 20 units of botulinum toxin per muscle (Figure 10) [49].
It is known that hypnosis triggers certain actions through mental images or internal representations mediated by the precuneus and reconfigured, in terms of executive control, by the frontal lobes [19]. The effective generation of the mental image of hypnotic paralysis is indeed attributable to the activation of the precuneus [282] (an effect also observed in various contexts [62,63]) and attentional control systems, as well as to the hypoactivation of the ipsilateral ACC and frontal gyrus [29].
The mechanism underlying flaccid paralysis has been studied primarily in the context of *conversion disorder*[57] (which is analogous to hypnosis); fMRI studies have demonstrated that during left-hand paralysis there was no activation of the corresponding motor neurons [84]. In conversion disorder, there was increased activity in

[56] For the simple reason that this muscle is often chemically paralyzed with botulinum toxin in aesthetic medicine settings, and thus serves as a good point of comparison.
[57] Once termed hysteria by Hippocrates (from *ὑστέρα*, *hystera* = uterus), it was subsequently classified as a *conversion reaction* (DSM-I, within *psychoconvulsive reactions*, from 1952), then *hysterical neurosis* (DSM-II, from 1968), then *conversion disorder* (DSM-III, from 1980, within *somatoform disorders*), and finally established as a distinct *conversion disorder* (DSM-IV, from 1994; DSM-V, from 2013).

the right hemisphere, including the IFG and IPL. During the conversion state, there was also a significant asymmetry in the connectivity of the rM1 compared to the lM1: the rM1 showed markedly higher connectivity with the precuneus and the vmPFC, a pattern not observed under ordinary conditions or during feigned paralysis [84]. It is interesting to note that in conversion paralysis, the internal representation of motor intentions was not abolished (as occurs, for instance, in neglect (§ 2.17) or motor anosognosia [128,143,174]). In other words, participants engaged in preparing the movement of the left hand and recruited the rM1 during the programming phase, even though the initiation of the movement was blocked during the execution phase [84].

At a pathological level, conversion disorder is the closest imaginable equivalent to hypnosis (which is, conversely, a completely physiological state) [267]. Confirming this, Halligan et al. [148] demonstrated, following a hypnotic command for flaccid paralysis, selective activation of the ACC and ROFC without activation of rM1, and they considered this pattern analogous to that observed in patients with conversion disorder [188].

From a receptor perspective, hypnotic flaccid paralysis is comparable to that induced pharmacologically by botulinum toxin (which binds to SV_2 to block acetylcholine release and neuromuscular transmission), curare-like agents, and succinylcholine [100,301]; however, the mechanism potentially linking hypnotic and hysterical flaccid paralysis to these receptors has not yet been identified.

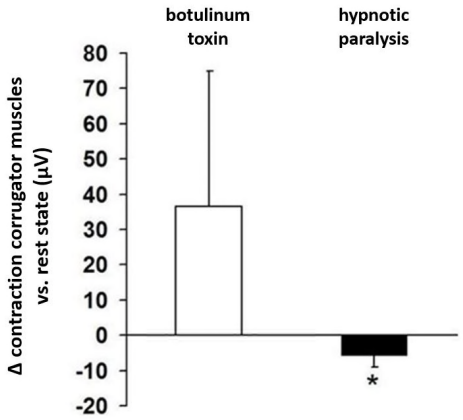


Figure 10. Effect of the injection of 20 IU of botulinum toxin per corrugator muscle (□) and of hypnotic suggestion of flaccid paralysis (■) on the tone of a striated muscle, measured via electromyography. Hypnotic suggestion proves more effective than the toxin; indeed, it is known to induce total paralysis—a phenomenon that, as demonstrated by the experiment described here, is objective rather than subjective [49].

2.14 Hallucination with Enhanced Psychophysical Performance

Physical performance can be enhanced through appropriate hypnotic suggestions. Major sports teams and elite athletes typically employ a hypnotist to address this aspect. Here, *performance* encompasses muscle strength, resistance to fatigue and lactate accumulation, as well as precision and speed of execution.

Using hypnotic and post-hypnotic commands such as *you will be stronger* and *you will feel less fatigue* it is easy to achieve the results shown in Figure 11 in controlled studies and measure them using standard human physiological units. In the cited experiment [269], the suggestion involved the hallucinatory consumption of a highly energizing drink (which did not exist in physical reality but only in the participants' mental imagery). As shown, this resulted in an increase in both muscle strength (measured *via* dynamometer) and endurance, yielding a 71% boost in overall performance. The command *you will perform exceptionally well in your task* also led to a 20.5% increase in speed and precision when solving a jigsaw puzzle [18], an effect attributable to

DAT-mediated dopaminergic agonism, NET-mediated noradrenergic agonism, σ_1-mediated opioid-neurosteroid agonism, and A_{2A}-mediated adenosine antagonism [75].
From a receptor-level perspective, these results resemble those obtained with cocaine [5,32,82,101,186,296], a DAT and NET antagonist with psychostimulant and ergogenic pharmacodynamic effects [10,32,101]. Dissociation was also observed[58]. The difference compared to cocaine lay in the absence of addiction and an elimination half-life of zero, rather than the several hours characteristic of chemical cocaine. Caffeine is also energizing, albeit less so than cocaine. Its action relies on antagonism of the A_{2A} adenosine receptor; it functions as a central stimulant because A_{2A} receptor activation in the olfactory tubercle and striatum exerts depressant effects [155,196,239], effects that are nullified by caffeine's antagonism, thereby producing the energizing effect. Furthermore, through the inhibition of cardiac A_1 adenosine receptors, caffeine also exhibits – albeit with lower intensity – the cardiac effects associated with cocaine [9,53,102].
Here, too, endogenous A-receptor antagonism can be observed in daily life in individuals who feel energized, either spontaneously or immediately[59] after consuming coffee (an Italian-style espresso contains 80 mg [53]) or other caffeinated beverages. Clearly, endogenous A_{2A} mechanisms elicited by hypnosis are at play here. An endogenous adenosine system does indeed exist [246] and can be activated by cognitive stimuli during hypnosis; in fact, adenosine activity is often normally inhibited by the body in response to various stimuli through an A-receptor-mediated anti-adenosine effect, simply more intense during hypnosis.

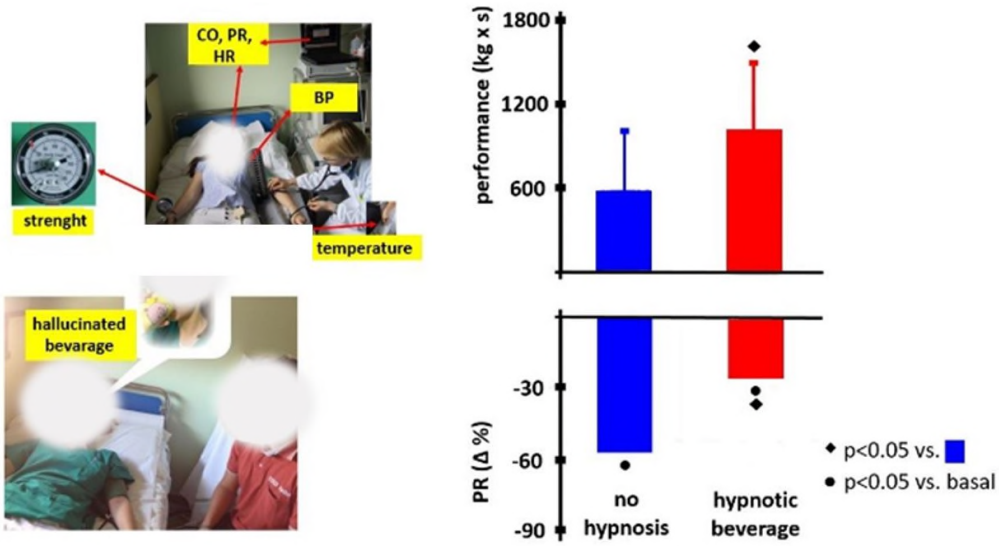


Figure 11. Isometric effort measured *via* dynamometer, involving the hallucination of consuming an energy drink. The increase in performance (in kg × s) is accompanied by an increase in heart rate (due to increased cardiac output) that is smaller than the increase observed during effort under normal waking conditions outside of hypnosis [269].

In summary, the endogenous energizing effect with positive inotropism achieved during hypnosis may stem from: 1) hypnosis-induced inhibition of DAT and NET (a cocaine-like stimulant effect); 2) hypnosis-induced inhibition of cardiac A_1 receptors and cerebral/systemic A_{2A} receptors (a caffeine-like stimulant effect); 3) a positive inotropic effect resulting from the activation of cardiac dopaminergic DA_1 and adrenergic β_1 receptors; and 4) an effect mediated by an endogenous digitalis-like system demonstrated by other authors [Bogrov et al., 2002] to act on digitalis glycoside receptors [23,48]. Distinguishing between these various effects requires specific, receptor-by-receptor studies, each necessitating approval from a different ethics committee; consequently, research in this area is likely to continue for years. Nevertheless, in light of the various effects

[58] Cocaine also acts peripherally as a local anesthetic and centrally (especially in the mid-cingulate cortex) as a psychic dissociative agent [32].
[59] This is pharmacologically impossible [70], given that the full effect of orally ingested caffeine, corresponding to the steady-state concentration, is not reached until after 3.3 half-lives, a period amounting to several hours.

observed, it can be stated that hypnotic recruitment engages specific receptors among those mentioned, depending on the type of suggestion administered [129].
These clearly receptor-mediated effects of hypnosis on performance – while not pharmacodynamic, given that they rely solely on cortical action (i.e., a mental image) – are not surprising. Indeed, even outside of hypnosis, individuals facing unexpected demands can enhance their psychophysical capabilities without the use of exogenous substances, managing under emergency conditions to perform tasks that would otherwise seem impossible, such as lifting heavy weights or solving problems that exceed their usual aptitudes. These additional self-hypnotic abilities (which are far more intense in hetero-induced hypnosis) are achieved by recruiting, through a cortically initiated action, the activity of receptors that are normally unrecruited, inhibited, or underutilized; this leads to increased physical strength and psychomotor capacity, as well as a reduction in the sensation of fatigue, effectively eliminating the subjective limit [269] that manifests in daily life as thoughts such as *I can't go on* or *it's too much for me*.

2.15 Heat-Induced Hallucination and Effects on Vascular Smooth Muscle

A sensation of warmth is almost always perceived, even during neutral hypnosis, and can be greatly intensified through appropriate commands to the point of becoming barely tolerable. It is interesting to determine whether this sensation of warmth is purely subjective (hallucinatory) or objective, that is whether it is measurable and reproducible in an experimental setting.
Attempting to measure temperature differences directly is unrealistic, given that they are extremely small, well below the method's margin of error[60]. The experimental challenge was overcome by measuring parameters proportional to temperature.
The fundamental point of this research is that, in the absence of an external heat source, as is the case with hypnosis, the only way to raise the temperature of a specific body part is to increase arterial blood flow to that area. Therefore, if a subject under hypnosis perceives a rise in temperature in a specific body segment, whether spontaneously or in response to an appropriate suggestion, it is easy to determine whether that increase is physically real and measurable or merely subjective. It suffices to measure local arterial flow[61] before and during the suggestion of heat: if it increases, the rise in temperature is real and measurable; if it does not, it is merely hallucinatory and subjective. Highly sensitive methods typical of cardiovascular physiology [51-56,58,59,140] make it possible to detect even minute thermal variations.
A suitable experimental model involves administering a command to a group of highly hypnotizable volunteers, first that their forearm is immersed in hot water, and then that their entire body is immersed in hot water (Figure 12) [60]. This generates a *hallucination of heat* (initially local, then systemic), the effect of which can be measured in terms of arterial flow (ml x min^{-1}). The experimental design is shown in Figure 12, and the setup is illustrated in Figure 13.
When the forearm is halluncinated to be immersed in hot water, the subject perceives heat in the forearm; arteriolar resistance in the forearm decreases, while arterial flow increases correspondingly. When the heat hallucination involves the entire body, the subject perceives systemic heat; total resistance decreases while CO increases correspondingly, exactly as occurs during an actual hot bath [91] (Figure 15). Since no external heat is applied, these cardiovascular effects stem entirely from the hallucination induced by the hypnotic command; the mind thus acts upon the body, much like when facial flushing occurs due to embarrassment. In this case, however, the vasodilatory effect is more intense and, crucially, can be modulated at the operator's discretion; it persists for the duration of the hypnotic phenomenon and then ceases. It is a targeted, purposeful, and controllable effect.
Those familiar with vasomotor responses know that an increase in CO entails a compensatory increase in inotropism. Myocardial contractility, expressed by SV, indeed increases during whole-body heating (§ 2.20, 2.21), but not during isolated forearm heating, such as occurs during a hot bath [91]. Whole-body heating also

[60] It should be noted that the temperature defined as normal body temperature [293] varies within a very narrow range (between 36.93 and 37.73°C), and that skin temperature (35.01–36.93°C) is lower than core temperature (rectal 36.3–37.8, tympanic 35.8–37, urinary 35.6–37.6, oral 35.7–37.4), which decreases further in the elderly. Normothermia is defined as temperatures up to 36.8°C.
[61] The first measurement of this kind and for this purpose was conducted under hypnosis in the mid-1990s [55] using complex equipment (a venous-occlusion indium-gallium-in-silicone strain-gauge plethysmograph) [51-56,58,59,140]. The main challenge lies in the fact that one must already be familiar with such instruments, knowing how to use them and having employed them hundreds of times in a hemodynamics laboratory.

affects the smooth muscle of visceral vessels (Figure 15). Imagining being in a hot bath produces a 16% increase in mesenteric artery diameter and a 43% increase in flow velocity [270]. Conversely, portal vein diameter decreases[62] by 25% in response to the mental imagery of body heating, counteracting arteriolar vasodilation and the systemic rise in CO. This may be due to the release of adenosine (§ 2.2) from the splanchnic region [155,156,157,196]. Ultimately, the result is the maintenance of pressure within the hepatic circulation (the so-called hepatic arterial buffer response) [106].

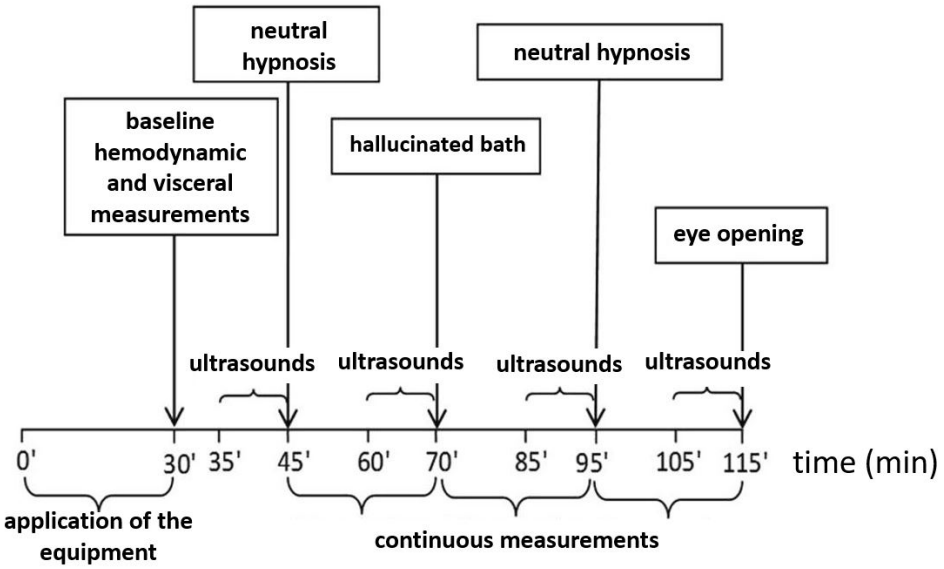


Figura 12. Hypnotic warm-up study plan including measurement of the diameter of deep vessels (mesenteric artery, portal vein) [270].



Figura 13. Experimental setting for heat hallucination. The participant is induced to hallucinate immersion in a tub of hot water, which is not physically present. Cardiovascular parameters are continuously monitored [60].

[62] Crandall et al. [91] have indeed demonstrated a reduction in liver volume during actual (non-hypnotic) body heating.

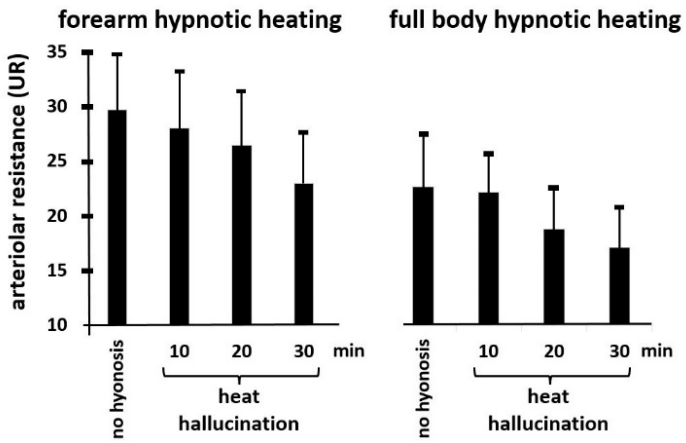


Figura 14. PR in the normal conscious state (no hypnosis) and after 10, 20, and 30 minutes of hallucinated immersion of the forearm and the entire body in warm water. UR: resistance units (mmHg x ml^{-1} x min x dl tissue) [60].

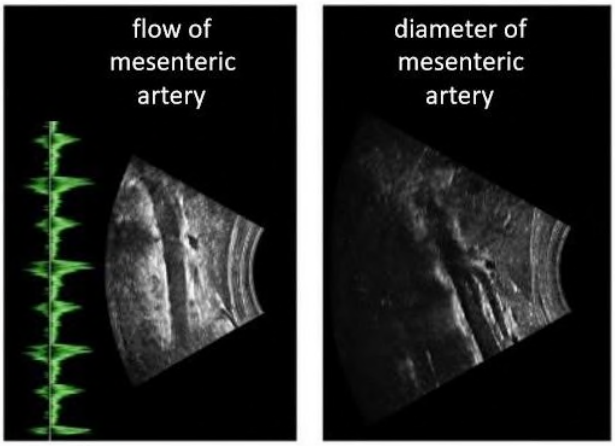


Figura 15. Mesenteric artery flow and diameter of a participant in the hypnotic body-warming experiment [270].

2.16 Effects on Visceral Smooth Muscle

Endoscopists practicing hypnosis had long observed that patients in a hypnotic *trance* appeared capable of controlling gastroduodenal peristalsis [147] and that, during the procedure, gastric peristalsis appeared reduced, an effect logically attributed, at the time, to hypnotic relaxation of the smooth muscle. After all, as previously noted, hypnotic suggestions can act powerfully and readily upon voluntary striated muscle (§ 2.14), involuntary striated muscle (§ 2.15), striated muscle subject to unconscious nocturnal contraction (§ 2.13), and the smooth muscle of blood vessels (§ 2.15). The first experimental demonstration took place at an international conference, where a specific hypnotic command delivered in real time experimentally demonstrated for the first time that it was possible to both induce and resolve antro-pyloric spasms through targeted instructions[63] [198-201].

[63] The mental image employed, which is now known not to be strictly necessary, as a direct command can be used instead, was that of *the petals of a flower opening in the morning*. Since the volunteer had previously undergone a digestive endoscopy, the technique of

The gastrointestinal tract possesses its own nervous system that regulates the basic motor and secretory functions of the stomach and intestine. The sympathetic and parasympathetic systems act merely to modulate the enteric nervous system (the former in an inhibitory manner and the latter in a stimulatory one). Under normal conscious conditions (outside of hypnosis), visceral afferents transmit information from mechano-ceptors, chemo-ceptors, and noci-ceptors to the central nervous system, where they elicit perception or trigger autonomic reflexes. The primary neurotransmitters released by enteric nerve endings include acetylcholine (which has excitatory effects, such as membrane potential depolarization leading to increased excitability), adrenaline and noradrenaline (which have inhibitory effects on intestinal smooth muscle), serotonin, dopamine, *vasoactive intestinal polypeptide*, somatostatin, leuenkephalin, metenkephalin, and bombesin. The receptor mechanisms associated with these transmitters have already been outlined (§ 2.2); it is now known that the ligand↔receptor interaction responds to cortical stimuli [75], thereby providing the basis for the response to hypnotic commands. In a controlled study on the effects of hypnosis on gastroduodenal motility [201], participants (who were also given suggestions regarding fasting and satiety) placed their hands on their abdomens; the instruction was: *your hand, guided by your brain, is capable of making you perceive your intestinal movements...* This was followed by: *"Your stomach is like a fist that contracts and closes pleasantly, and then your stomach relaxes, expands, and opens like the petals of a flower.* Continuous manometric monitoring demonstrated that, under hypnosis, the patient can perfectly simulate fasting, satiety, and digestion, and contract and expand the stomach with an almost immediate response. When asked to contract the stomach, peristalsis begins at the antro-pyloric level, approximately 5–6 cm from the pylorus (where the peristaltic impulse physiologically originates), and propagates in an oro-aboral direction (Figure 16). Separately, it has also been observed that gastric motility can be modified *via* hypnotic commands, targeting specific regions such as the *fundus*, body, *antrum*, and *pylorus* which the patient in hypnosis is able to control (Figure 17). Experimental results thus confirm that gastroduodenal motility can be modified and controlled when the subject is in a hypnotic state. PET studies have demonstrated that the prefrontal cortex and the ACC serve as the anatomical source of specific visceral symptoms arising from ideas or mental images [234]. The prefrontal cortex acts as a personal memory repository, combining the meaning and emotional weight of events with personal experience, while the ACC responds to emotionally charged ideas, memories, and various stimuli by generating a visceral response. Hypnosis acts on the ACC without altering somatosensory cortex activity [229]; this explains how a suggestion administered during hypnosis can, through mental imagery, elicit an immediate and precise visceral motility response. Today, hypnosis involving specific commands is frequently used in digestive endoscopy, either alone or in combination with mild sedation [203].

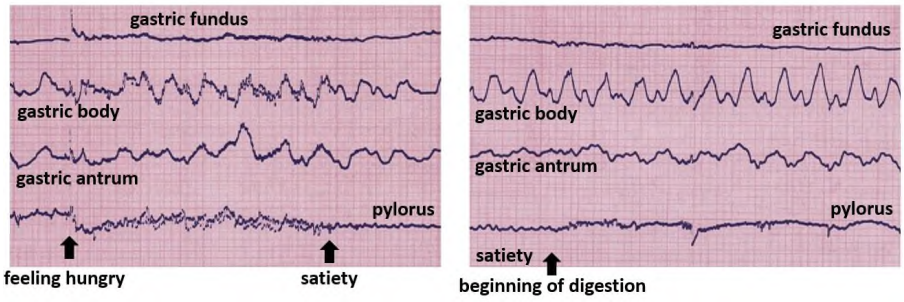


Figure 16. Left: endoscopic manometry with hypnotic simulation of fasting in 20 participants (phase II: 1–2 cycles/min with unsynchronized antral movements and pyloric contractions) and simulation of satiety (antral phase III: 3 cycles/min with synchronized mixing waves). Right: simulation of digestion showing the classic downward-directed peaks. Arrows indicate hypnotic commands [201].

revivification (§ 2.19) regarding the conscious sedation and local anesthesia (§ 2.10) was also used to reduce the discomfort associated with the procedure.

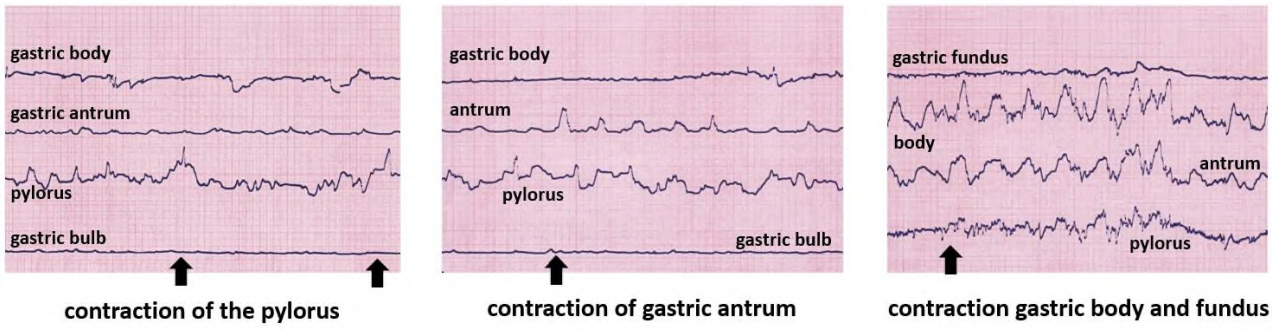


Figure 17. Peristaltic pulses following contraction commands at the level of the pylorus, extending to the gastric antrum, the body, and finally the fundus of the stomach. The arrows indicate the hypnotic commands [201].

2.17 Neglects

All hypnotists with some experience are capable of producing[64] what are loosely referred to in the field as *negative hallucinations* [37,42,65], i.e. situations in which an element of the world goes unperceived (a body part, an object, one or more people – even selectively, such as for instance only those dressed in red – or an entire sector of the visual field). The same applies to auditory stimuli. These phenomena are, in reality, instances of *selective neglect*. Demonstrating their objective existence experimentally is no easy task.

A particularly striking example of negative hallucination is *spatial hemineglect* [21,65], in which one side of the physical world (such as *everything to the left of the nose*) ceases to be of interest to the subject[65]; it is obviously perceived visually, yet it does not enter consciousness. If asked, the subject will state in perfect good faith that they do not see it. The premise is that spatial attention can be directed by specific endogenous cues [223,224]. By inducing, for instance, left-sided neglect, the hypnotist can *appear and disappear* from the participant's field of attention simply by changing sides in what is known as the *now you see me, now you don't* experiment (the participant sees the hypnotist, of course, but takes no interest in them). To demonstrate hemineglect, one can use a system consisting of a screen where an algorithm causes a light point to appear at unpredictable intervals to the right or left of a central cross; a button available to the participant (who keeps their gaze fixed on the central cross) is used to signal perception in the right or left periphery[66]. If, for instance, a command such as *the left side of the world does not interest you*[67] is given during hypnosis or in post-hypnotic state, light points originating from the left are perceived a few ms later than those from the right; this indicates that directing visuospatial attention toward the neglected side results in a slower reaction [226]. The same phenomenon occurs in reverse with right-sided hemineglect. Hemineglect can thus be experimentally demonstrated and measured in terms of reaction-time delays (145 vs. 79 ms, $p<0.02$) (Figure 18). In cases of complete hemineglect (easily induced in *highs*), the failure to perceive one side of the world is absolute – as if that side did not exist – because the lack of interest driving the neglect is total. This confirms that participants allocate their visuospatial attention along a gradient [164]. It is essential that these results, obtained at the University of Padua's Laboratory of Experimental Hypnosis, be replicated using neuroimaging techniques, something that, to date, has not occurred. Consequently, to the best of the present *Position Paper* authors' knowledge, the mechanisms underlying hypnotic hemineglect remain unknown. An experiment from the late 1980s involving carotid injection of barbiturates (the Wada test [283] conducted, moreover, on epileptic patients) appears to point toward an extinction-type response [192], but in reality, nothing more is known.

[64] And they usually do so in an educational or demonstrative context.
[65] This is a well-known condition in neurology, usually caused by a neoplasm, ischemia, or physical damage to the brain—typically the right hemisphere, resulting in left hemineglect [189].
[66] The protocol is somewhat more complex; for details, please refer to the original source [226].
[67] Left hemineglect faithfully reproduces the pathological condition described above, but the same procedure can be repeated with similar results to induce right hemineglect.

Another interesting form of neglect concerns written language *(hypnotic alexia)*; the Stroop effect[68] has been used to demonstrate this. Under the hypnotic command *you have lost the metaphor of language*, words are stripped of their linguistic meaning and appear as non-verbal symbols; consequently, they produce little to no word-color incongruity. The existence of a neglect for the written word is demonstrated by a reduction in the Stroop effect, measured as the change (Δ) in response time (in ms) to the visual stimulus (-34%, p=0.03) [61]. Using a similar procedure and the command *you have lost your sense of rhythm*, researchers demonstrated a form of neglect known as *hypnotic amusia*[69] [125]. In this experiment, the participant hears a series of *beeps* through headphones; when a different signal (a prolonged *beeeeep*) is introduced, their EEG records a discrepancy known as *mismatch negativity* (an auditory event-related potential widely distributed across the scalp, with greater amplitude in the right frontal areas [4,209]), measured as a change (Δ) in signal amplitude (in μV). Mismatch negativity decreases on average from -8.2 ± 2.2 μV to -5.4 ± 1.1 μV (−20%, $p<0.05$) when the sense of rhythm has been abolished by the hypnotic command.
While these experiments designed to demonstrate and measure neglect may seem simple when described here, they are actually highly complex and require a large, trained team. Even creating the conceptual model is challenging; Figure 19 shows the experimental model for alexia as an example. Furthermore, reaction time measurements require specialized equipment – often custom-built – are difficult and prone to error, and must be repeated several times.

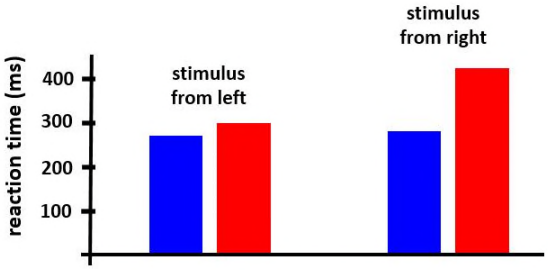


Figura 18. Right-sided hemineglect induced by hypnotic suggestion. When a visual stimulus originates from the right, its perception is slower under hypnosis (■) compared to usual consciouness (■). When the stimulus originates from the left, there is no difference between the two conditions [226].

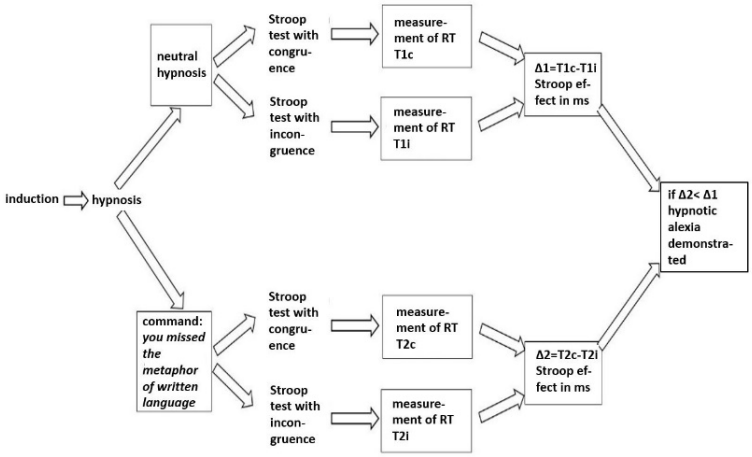


Figure 19. Mental model of the experiment demonstrating the neglect known as *hypnotic alexia*.

[68] In the Stroop test, colored words denoting colors (RED, GREEN, YELLOW, BLUE) are presented to the participant, who must select the button corresponding to the color; if the word is written in its own color (e.g., **RED**), it is termed *congruent*, whereas if it is written in a different color (e.g., **GREEN**), it is termed *incongruent*. The time (measured in ms) required to select the color is longer in cases of incongruence, and the difference compared to the congruent condition is known as the *Stroop effect*.
[69] A pathological form of amusia, commonly known as *temporal amusia* or popularly referred to as *beat deafness*, does indeed exist and affects 1.5% of people to varying degrees of severity [214,215].

2.18 Phantom Limb

This topic would have been of purely narrative interest and would not have found a place in this *Position Paper* on Experimental Hypnosis, but a recent review [244] re-examined it by introducing an analysis of experimental methods. The authors state that the therapeutic modality most frequently used to treat phantom limb pain is *mirror therapy*[70] [232] followed by *virtual reality* and *augmented reality*. Auditory feedback, neurofeedback, thermal biofeedback, and illusory touch have also proven useful; these are procedures closely linked to medical technology that border on hypnosis.
The authors conclude that phantom limb pain *"is a complex condition involving physiological, psychological, and social factors"* in addition to pharmacological ones. It is plausible that a hypnotic hallucination would render Ramachandran's mirror box setup useless, even though such an experiment has never been attempted.

2.19 Regression and Revivification

It has long been known that *age revivification* is possible [141,256,257], whether spontaneously [135] or within a therapeutic context [289]. In an educational or demonstrative setting, it is relatively easy to lead a participant to relive events, experiences, thoughts, and sensations from their personal life, even from the distant past [287]. In short, it is possible to induce age revivification during deep hypnosis and directly observe the associated phenomena (for instance, if regressed to childhood, the subject adopts childlike facial expressions, asks for milk, looks for their mother, cries, etc.). This applies to the narrative level.
However, demonstrating that this actually occurs – that the revivification is real (rather than a mere subjective experience) and that the subject feels and actually *is there and then*, rather than *here and now* – is a different matter. In age regression, a subject is led through hypnotic suggestion to relive moments from their autobiographical memory, as well as moments that had been removed[71] from consciousness and relegated to the Unconscious [160].
Regression and revivification are sometimes confused. While hypnotic revivification (age assumption adopting the experiences, behaviors, and personality of the past) does indeed involve age regression (recalling the past while retaining one's present personality), it also requires hypermnesia [236] with enhanced powers of recall and memory fixation an increased, fluid, and orderly flow of recalled experiences, and a certain degree (potentially an intense one) of dissociation [42,277]. For instance, a person undergoing hypnotic revivification of his/her childhood thinks and behaves like a child, while also being convinced (in a state of dissociation) that he/she *is a child* [142].
A child's personality differs from an adult's, and projective tests yield different responses depending on the subject's age: adults tend to provide more *global responses*, whereas children provide more *detail responses*. Consequently, a projective test can serve as a valid objective measure in age-revivification studies [195], provided that the frequency of global vs. detail responses is compared using an intra-subject Latin square design and quantified by measuring both response types. Under these conditions, the test can be used with subjects unfamiliar with the instrument to assess baseline personality outside of hypnosis (based on chronological age) and to observe whether during age revivification (e.g., to age 6) there is a shift toward more frequent detail responses and less frequent global responses (Figure 20). If this shift occurs, it demonstrates that adult subjects undergoing childhood revivification exhibit a childlike personality, thereby confirming the reality of the revivification process.
Following pioneering experimental attempts [13,14,135,136,168,263], this approach was implemented in a study conducted at the University of Padua; participants undergoing revivification actually showed an increase in detail responses and a decrease in global responses, i.e. a pattern consistent with childhood (Figure 21, left panels) [142,245].
Further confirmation came from intelligence tests. Under identical conditions – and conditions are identical when each participant serves as their own control – a child revivifying participant possesses lower intelligence

[70] A method that employs a mirror device capable of making the patient believe that their amputated limb still exists and can be moved. The technique is based on the theory that phantom limb pain stems from an altered body schema and the futile attempt to contract muscles that are no longer present. The *mirror device* creates the illusion that the patient can contract these muscles, whereupon the pain caused by the impossible contraction disappears [232]. Although this has never been attempted, the mirror device could be replaced by a hypnotic hallucination (§ 2.14, 2.15, 2.20) or by a simple body dysmorphism (§ 2.9).
[71] At the narrative level, there also exists an unsuppressed unconscious [93].

than an adult. Measuring intelligence *via* the Raven test for adults at both the chronological age and the revivified age revealed lower intelligence during revivification (Figure 21, right-hand panels). The adult subjects undergoing revivification were thus *truly children* in this respect as well [142], thereby confirming the reality of the revivification[72] [142].

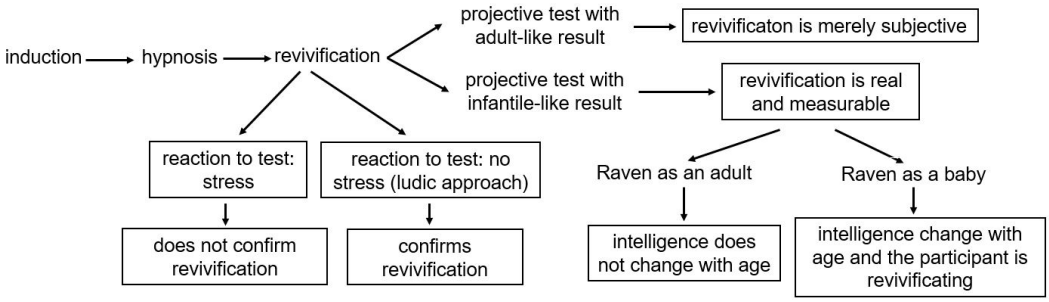


Figura 20. Study protocol implemented for the experimental demonstration of hypnotic revivification [213]. The childhood personality was assessed using a projective test and Raven's matrices. The cardiovascular reflex response to the Raven test, acting as a stressor, was also investigated [142].

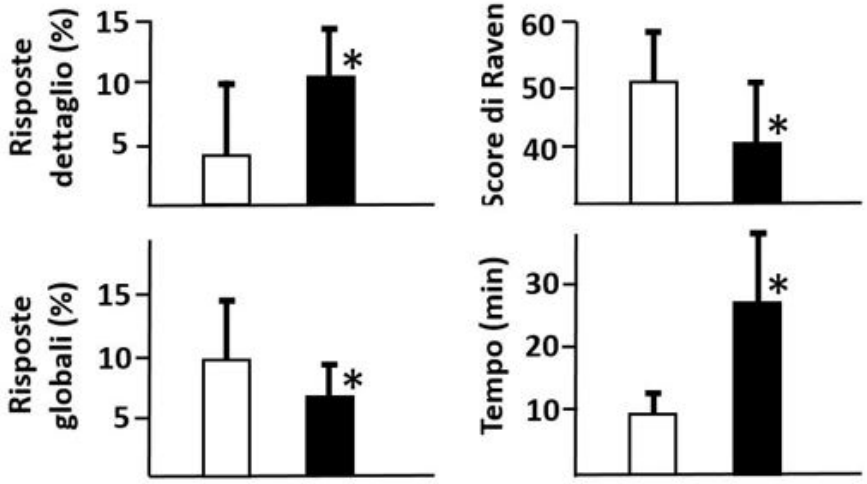


Figura 21. Demonstration and measurement of hypnotic revivification [142]. Compared to ordinary waking states (□), the subject undergoing hypnotic revivification (■) shows a higher frequency of detail responses and a lower frequency of global responses on the projective test (left panels) indicative of a childlike personality. Furthermore, on the Raven's Progressive Matrices test for adults (right panels), the subject achieves a lower score and takes longer to solve the problems. *$p<0.01$ vs. □.

Even the alleged reliving of past lives (now more accurately termed *regression to an unreal time*) has been the subject of some experimental study. Curiously, the two studies that came closest to meeting scientific standards

[72] Revivification can serve for instance to rapidly retrieve repressed memories and traumas. Demonstrating that it is a genuine, measurable phenomenon grounded in science has paved the way for its application in other areas, such as experimental research, forensic practice (with all the associated complexities [272]), and criminal investigation [41]; it also offers a practical method for recovering memories that have slipped into the unconscious [87] or locating lost objects whose whereabouts have been forgotten.

were commissioned by national television broadcasters (one Korean, one Italian), and video recordings of them exist. It goes without saying that these rigorously conducted investigations led to the conclusion that it is easy to manipulate a participant into adopting a past personality [171]; indeed, often no specific prompting is required, as the subject is capable of spontaneously constructing a past identity (usually one of some distinction). Gonella [144] notes that during hypnotic *trance* the imagination can break free and give rise to the most extraordinary fantasies; consequently, a subject's creativity may shift into a retrograde or anterograde *unreal time* [288], even extending to alleged memories of encounters with extraterrestrials [144]. Unfortunately, the internet is rife with accounts of such unreal regressions, that people tend to believe, and the topic is often treated superficially; for instance, it is rarely specified whether these memories are articulated during a state of *waking trance* or a so-called s*omnambulistic trance*[73]. The formation of false memories and false beliefs under hypnosis is well documented [208] and is clearly mediated by the k-opioid receptor. Indeed, experiences of revivification occur (albeit in a far less coordinated and much more bizarre manner than in hypnosis) even outside of a hypnotic context following the administration of KOP agonists, typically psychedelics such as salvinorin A or ibogaine[74] [94,183].
Hypnotic age revivification (real or to unreal) is therefore a KOP phenomenon suggesting the existence of a cortically controlled endoKOPergic system, one that has never been experimentally demonstrated given the practical impossibility of conducting research with psychedelics due to current legal restrictions [75].

2.20 The Experimental Demonstration of Hallucinations

High-level hypnotists know how easy it is to induce hallucinations (§ 2.4) that, for the subject, possess the quality of absolute reality. It is more difficult to demonstrate that these hallucinations are real – that they are not merely subjective experiences – and that they have consequences at an experimental level [42].
Evidence that hallucinations are real is already implicit in several preceding sections of this *Position Paper*. The hypnotic sensation of heat (§ 2.15), enhanced psychophysical performance (§ 2.14), dysmorphism (§ 2.9), the digestion of a non-existent meal (§ 2.16), and revivification (§ 2.19) are hallucinations that not only hold the quality of reality for the participant but also produce physical effects that can be demonstrated and measured in units typical of human physiology.
To round out this topic, a more complex situational hypnotic hallucination of paradigmatic significance is presented here. In one experiment, a bloodletting procedure was hallucinated [55,57] to determine whether the hemodynamic changes observed during blood donation (which serves as an experimental model of acute hemorrhage) should be attributed to the actual physical withdrawal of blood or to the experience of the donation itself.[75].
Can a hallucination be used to resolve an outstanding question in physiology? When subjects lose 300 ml of blood, they exhibit reflex sympathetic reactions (specifically tachycardia and vasoconstriction) that override the drop in blood pressure that would otherwise result from the blood loss. For decades, the question remained whether this sympathetic *alarm response* stemmed purely from hemodynamic factors (the loss of blood itself) or from the experience of the blood draw (which is inherently stressful).
Individuals who had never donated blood first underwent an actual 300 ml of blood draw as a voluntary donation (Figure 22, left). On a separate occasion, the same individuals underwent a hallucinated blood draw under hypnosis, during which they experienced the sensation of donating blood, but suffered no actual blood

[73] An individual entering a *waking trance* maintains a strong connection to the here and now; consequently, any fantasizing occurs while retaining awareness of the narrative, even when accompanied by vivid mental imagery, and the person remembers the events and contexts they memorized, imagined, or interpreted after emerging from the trance. In contrast, a subject in a so-called *somnambulistic* hypnotic state drifts in a condition detached from time and space, where critical judgment is moderated and serves the new context emerging within their mental representation. In an ordinary state of consciousness, the somnambulistic subject is invariably unable to recall the hypnotic experience they underwent. The amnesia associated with somnambulistic hypnosis is always a failure of recall rather than of encoding; thus, all the stored memory material would be available and retrievable if the patient were returned to a somnambulistic hypnotic trance [144].
[74] The k-stimulation also elicits a combination of ego expansion [81], OBE (including autoscopic ones) and a sense of divination; these experiences can be induced at will by the operator during hypnosis, whereas with the chemical ligand, they occur unpredictably, depending on the context and the subject's personality [36].
[75] This physiological question remained unresolved until the experiment described here, and, frankly, it is difficult to see how it could have been clarified other than through experimental hypnosis.

loss. In reality, there was no needle, collection set, or blood bag, except in the subjects' minds (Figure 22, right). Hemodynamic parameters were continuously monitored.

Thus, actual *bloodletting involving blood loss* and the *hallucinatory version without blood loss* elicited the same hemodynamic response, with a reflex vasoconstriction that was virtually identical in both experimental settings (Figure 23). These results align with the observation that when a normal subject moves from a lying to a standing position there is no increase in peripheral resistance (even though 300-500 ml blood shifts abruptly due to gravity from the central circulation to the peripheral capacitance vessels) because the experience of changing posture involves no stress. Consequently, this answers a long-standing question among physiologists: the response to blood loss is neither physical (hydraulic) nor chemical, but entirely psychological, so much so that it can be triggered by a mere hallucination in the absence of any actual reduction in circulating blood volume. At the same time, the hallucination of a complex situation was demonstrated to be real and measurable.



Figura 22. Experimental setting for demonstrating the hypnotic hallucination of bloodletting. On the left, the subject undergoes the withdrawal of 300 ml of blood; on the right, the subject hallucinates the bloodletting while under deep hypnosis. Hemodynamic changes are monitored in both conditions. The minimum critical mass for this experiment is three researchers [55,57].

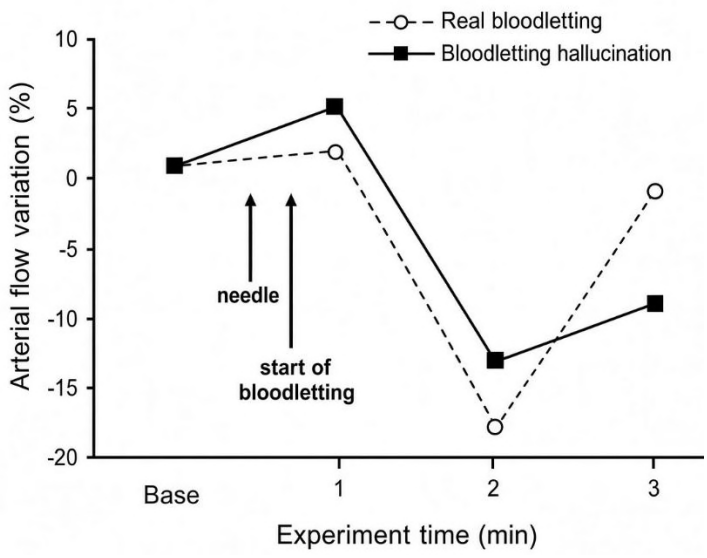


Figure 23. Hemodynamic effects of actual bloodletting (■) and a hallucination of bloodletting (○), which produce equal vasoconstriction in the leg.

2.21 The Experimental Demonstration of the Unconscious

An Unconscious[76] (i.e., a part of the mind inaccessible to consciousness) was first vaguely hypothesized by Arthur Schopenhauer (1788-1860), subsequently formalized by psychoanalysis [130,131,160], and later summarized in various historical reviews [109,266]. However, no one had ever proven its existence. The Unconscious as an ontological reality [61,66] has recently been experimentally demonstrated at the University of Padua using indirect methods (that is, through its effects) employing hypnosis as a tool [55,63]. The chapters discussed above have highlighted various responses that can only be described as unconscious.

During hypnotic body warming induced by hallucinating hot tub bathing (§ 2.15), the participant is unaware that physical warming [91] increases SV (+28%); even if they were aware of this, they would be unable to increase it voluntarily and consciously. The increase in SV during actual warming (Figure 24) is an autonomic event driven by the temperature effect; conversely, the increase observed during hallucinatory warming in the absence of external heat application (§ 2.15) [60] is a phenomenon driven solely by the mental image [44-47] of warming and is, therefore, a completely unconscious phenomenon. The existence of the Unconscious is thus demonstrated [66].

Also in the context of body warming simulating warm tub bathing [91], a 59% increase in the diameter of the visceral arteries was observed, along with a mirroring trend in the portal vein (§ 2.15) [270]. The setup is that outlined in Figure 25. Once again, the participants are unaware that physical warming causes dilation of the mesenteric artery; even if they were aware of this, they would be unable to induce it voluntarily and consciously, whereas a mental image can achieve it. The existence of the Unconscious is thus demonstrated once more [66]. Furthermore, the drop in portal vein blood flow during warming is a phenomenon virtually unknown even to physiologists, yet the hallucinatory mental image of heat succeeded in producing it; it is, therefore, a completely unconscious phenomenon. Not only that, but, being independent of the consciousness of both the participant and the experimenter, it constitutes a sort of *double-blind unconscious experiment* [270].

Through bloodletting – an experimental model of acute hemorrhage feasible in the laboratory (§ 2.15) – it has been demonstrated that an acute loss of blood that is merely hallucinated (though real to the subject within their subjective world) is also real in the physical world, insofar as it produces reflex vasoconstriction resulting from the formation of a mental image from the plasticity of a monoideism.

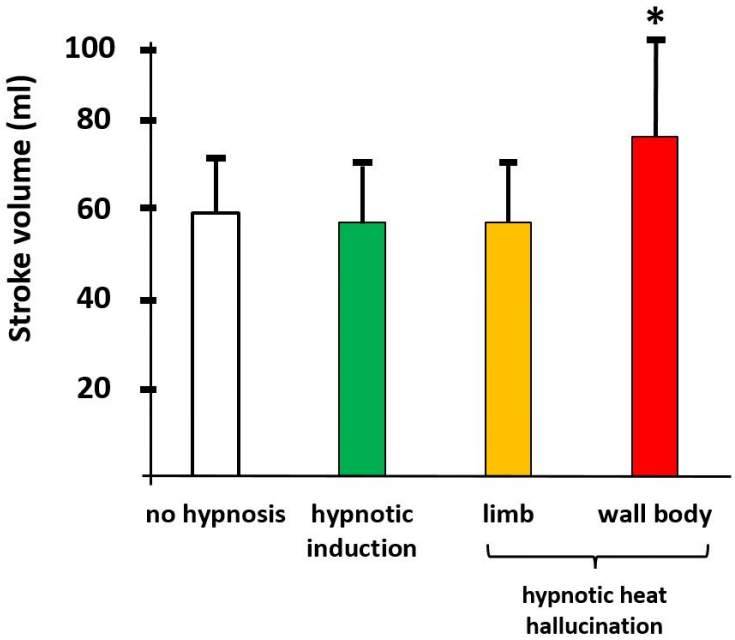


Figure 24. SV under baseline conditions without hypnosis, during hypnotic induction (■), and during hypnotic suggestion of a limb in warm water (■) and the whole body in warm water (■). *p<0.01 vs. no hypnosis. Other differences are not significant [60]. The increase in SV due to body warming is an unconscious event [66].

[76] As has been specified on several occasions, the reference here is not to any particular Unconscious among those theorized by the many psychoanalysts who have addressed the subject [20].

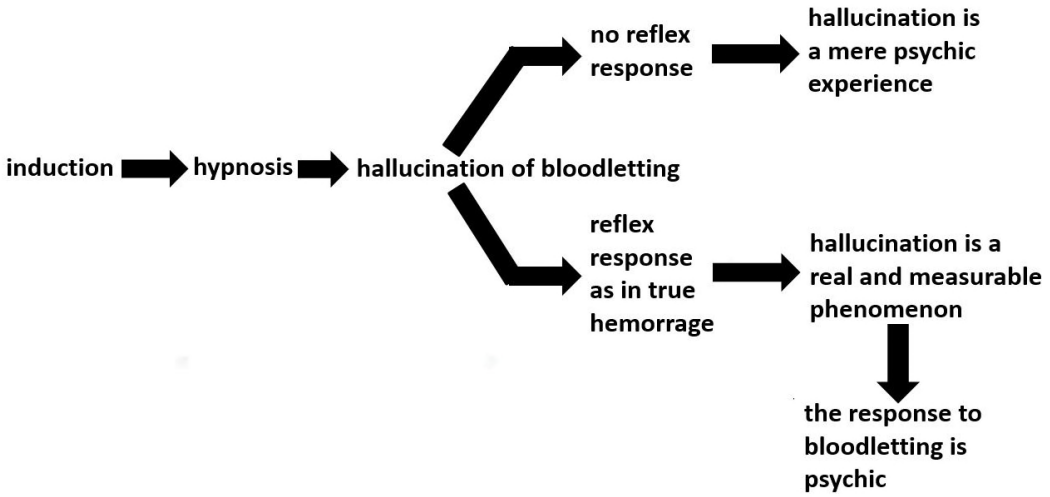


Figure 25. Outline for demonstrating the reality of the situational bloodletting hallucination.

In another experiment cited above, the one involving hypnotic alexia (§ 2.17), the existence of the Unconscious was demonstrated using RT to visual stimuli as an indicator [61]. As will be recalled, the sequence of experimental events was: hypnotic induction → suggestion of alexia → mental visualization of the alexia becoming *plastic* → Stroop task → measurement and comparison of any variations in RT. The theoretical premise of this model was that 1) cognitive processes can be controlled either unconsciously or voluntarily, that 2) involuntarily activated processes (triggered unconsciously) cannot easily be interrupted, and that 3) words are processed at an unconscious level [184]. The principle postulated by Stroop is that reading color names unconsciously interferes with the color in which the word is written, thereby reducing responsiveness and slowing down word identification in incongruent conditions. Indeed, in cases of incongruence, RT were 120 ms longer than in the congruent condition (i.e., the Stroop effect was 120 ms); however, following the suggestion of hypnotic alexia, the Stroop effect was halved [61]. The detection of a significant interaction between suggestion, congruence, and hypnotizability ($p<0.04$) confirmed that hypnosis was indeed the cause of the hypnotic alexia. Since no conscious or voluntary control is possible within this RT interval, once hypnotic alexia characterized by the loss of word meaning was achieved, an unconscious component must have come into play: the mental image of hypnotic alexia evidently generated an unconscious neglect of written language, leading to greater unconscious congruency in cases where colors conflicted with the words naming those colors. This once again demonstrated the existence of the Unconscious [66].

A similar situation arose in the experiment [125] concerning hypnotic amusia (§ 2.17). Briefly, after inducing neglect for rhythm following the sequence of hypnotic induction → suggestion of amusia → mental visualization of the amusia taking effect → presentation of deviant stimuli → measurement of any variations in the mismatch response, the amplitude of the mismatch negativity (the response typically following an anomalous signal within a series of identical ones) decreased. Clearly, this response to the mental image of hypnotic amusia must be unconscious in nature, as there is no way to consciously or voluntarily control EEG traces (particularly within the ~100 ms stimulus time window, which is well below the threshold of conscious perception). This once again demonstrates the existence of the Unconscious [66].

In the experiment [226] regarding hemineglect (§ 2.17), the sequence of experimental events was: hypnotic induction → suggestion of hemineglect → formation of a mental image of hemineglect → presentation of lateral visual stimuli → measurement of any variations in RT. Once again, this response could only be unconscious in nature, given that it is impossible to control RT consciously or voluntarily within this timeframe. Naturally, this was a subliminal response (falling within the realm of psychodynamics) rather than an automatic response resulting from a brain lesion (a matter for clinical neurology), as there was no organic lesion in the experimental context based on hypnotic suggestion of neglect. Consistent with observations reported by other authors in pathological conditions, this phenomenon stems from an unconscious spatial

attraction that draws attention away from the ipsilateral side and strongly attracts it toward the contralateral side [164,185]. Neglect is, therefore, simply an unconscious shift of attention *from left to right* or *from right to left* [226]. All the aforementioned results confirm the existence of the Unconscious [66].
As highlighted above (§ 2.9), achieving analgesia and even surgical anesthesia (§ 2.10) is surprisingly simple with hypnosis. The best method involves suggesting direct mental imagery (§ 2.8) or dysmorphism (§ 2.9) that leads to insensitivity to pain-inducing stimuli. The sequence is as follows: hypnotic induction → suggestion of HFA or dysmorphism → mental imagery of HFA (which can be extended to the entire body, causing it to take on a plastic-like consistency) → administration of a painful stimulus → imaging of sensory brain areas + measurement of pain perception, pain tolerance, and any hemodynamic reflex response. Non-trigeminal (peripheral) pain is associated with increased sympathetic discharge and a rise in PR, whereas trigeminal pain is associated with a reduction in PR [63,118,119]. Knowledge of this hemodynamic response is not part of the general public's understanding, being instead the province of a select group of physiologists, anesthesiologists, and cardiologists. Nevertheless, when HFA was suggested, the cardiovascular response to pain was consistently inhibited, resulting in a smaller PR increase following non-trigeminal pain and a smaller PR reduction following trigeminal pain (§ 2.8). Since the participants were unaware of the difference between the cardiovascular consequences of the two types of pain, this differential response to the mental imagery of HFA and HGA can only be unconscious in nature, thereby demonstrating the existence of the Unconscious [66].
Hypnotic age revivification (§ 2.19) [142] following the model hypnotic induction → revivification suggestion → transformation of the mental image into a *plastic* form → quantification of any variations in projective tests (for age-specific personality) and Raven tests (for age-specific intelligence) proved illuminating even at the protocol stage. Indeed, the projective test administered during the revivified age revealed a childlike personality; the Raven score for adults decreased, while the time required to complete the test increased. Ordinary individuals are unable to control their responses to projective or intelligence tests while simulating a younger age (a skill that might perhaps belong to an expert psychologist). However, what truly demonstrates the existence of the Unconscious is the cardiovascular response to the Raven test, viewed as a stressor. Under baseline conditions, the administration of Raven's matrices was accompanied as expected by a significant increase in PR and a reduction in SV due to a reflex drop in afterload; yet, when the test was administered during hypnotic childhood revivification, no variation in these cardiovascular parameters was observed. This was because the playful approach typical of childhood stripped the Raven test of its stress-inducing effect, thereby reducing its impact on sympathetic outflow. These objective variations in cardiovascular response, which are obviously involuntary and cannot be simulated, confirm the Unconscious exists [66] as has long been stated; furthermore, its effects are physical and measurable in the units of human physiology. The Unconscious revealed by experimental data eludes all conscious or voluntary control, it is blind and totipotent, can manifest with all the characteristics of a physiological agent (as seen in psychosomatic disorders), and can be demonstrated experimentally using instruments and devices typical of Galilean sciences, such as physiology and cardiology. Most importantly, it must not be confused with an automatic neurovegetative response, as it is evoked by a mental image (rather than a simple reflex) and operates through the higher function of plastic monoideism.

Part 3
Conclusions

Approaching problems through a narrative framework is often effective; indeed, from an anthropological perspective, this is precisely what *Homo sapiens* does in every daily activity: an approach no one questions. It is therefore not surprising that the various narratives surrounding hypnosis have consistently been accepted as valid, even when they contradicted one another, or were later proved demonstrably incorrect. At a certain point in history, however, a segment of society (namely, the scientific community) recognized the need to bring clarity by distinguishing through science subjective narratives from objective reality. recognized the need to bring clarity by distinguishing

Science arrived late to the field of hypnosis, so it is only natural that it has struggled to establish itself as the *definitive truth*. Over the decades, a multitude of overlapping interpretations has accumulated; stripping away these layers can be difficult, and at times unsettling. The only way forward is through experimentation, relying on results that have been rigorously confirmed and reconfirmed, as Galileo taught, and ensuring these findings permeate both the professional sphere and the general public. This process strengthens certain concepts while refuting others, ultimately leading to greater clarity. To achieve this, everything must be open to questioning.

The contributors to this Position Paper have dedicated significant portions of their lives to this scientific research, and some of their key findings are summarized here. Today, it can be affirmed that many of the insights held by the masters of the past, passed down through tradition, have proven to be accurate. For instance, hypnosis is a real phenomenon, is a distinct state, can be intentionally induced, relies on the emergence of plastic monoideism, requires the subject's deliberate acceptance, and manifests in varying degrees of depth. Furthermore, it is confirmed that hypnosis, when properly conducted, can produce both psychological effects (such as anxiolysis, sedation, age revivification, hallucinations, neglect, etc.) and physical effects (analgesia, anesthesia, paralysis, the control of blood vessels and visceral muscles, etc.). All of this is now scientifically established, whereas other aspects, such as whether hypnosis constitutes a modified state of consciousness or the existence and nature of the *rapport*, still await confirmation.

Moreover, the use of hypnosis as a research tool has enabled the experimental demonstration of the Unconscious, thereby shifting it from the realm of narrative-based disciplines to that of empirical science.

Much remains to be confirmed or refuted, and it is highly desirable that more scholars engage in experimental research in this field: a challenging prospect, given that, while hypnosis itself is a niche discipline, experimental hypnosis is even more so.

References

[1] Abrams, S. (1964). The use of hypnotic techniques with psychotics; a critical review. *American Journal of Psychotherapy* 18: 79-94.

[2] Agrillo, C. (2011). Near-Death Experience: Out-of-Body and Out-of-Brain? *Review of General Psychology* 15: 1-10.

[3] Aleksandrowicz, J.W. et al. (2006). Obrazowanie hipnozy w funkcjonalnym rezonansie magnetycznym. *Psychiatria Polska* 40: 969-983.

[4] Alho, K. (1995). Cerebral Generators Of Mismatch Negativity (MMN) and Its Magnetic Counterpart (Mmnm) Elicited by Sound Changes. *Ear & Hearing* 16: 38-51.

[5] Amigó, S., & Ferrández, C. (2015). Experiencing Effects of Cocaine and Speed with Self-Regulation Therapy. *Spanish Journal of Psychology* 18 (e49): 1-10.

[6] Anderson, J.W., et al. (2016). Private Prayer Associations with Depression, Anxiety and Other Health Conditions: An Analytical Review of Clinical Studies. *Postgraduate Medicine* 128: 635-641.

[7] Angioi, A., et al. (2025). Ultrasound-Guided Kidney Biopsy: A Ten-Year Retrospective Single-Center Experience and the Promising Role of Clinical Hypnosis. *International Urololy and Nephrology* 57: 553-559.

[8] APA Official Actions. (2009). Position Statement on Hypnosis. Approved by the Board of Trustees, September 2009. Approved by the Assembly, May 2009. Vedere: *https://www.psychiatry.org/file%20 library/about-apa/organization-documents-policies/policies/position-2009-hypnosis.pdf*.

[9] Bagrov, A.Y., et al. (2002). Endogenous Digitalis-Like Ligands of the Sodium Pump: Possible Involvement in Mood Control and Ethanol Addiction. *European Neuropsychopharmacology* 12: 1-12.

[10] Baird, J.R.C., & Lewis, J.J. (1964). The Effects of Cocaine, Amphetamine and Some Amphetamine-Like Compounds on the in Vivo Levels of Noradrenaline and Dopamine in the Rat Brain. *Biochemical Pharmacology* 13: 1475-1482.

[11] Baird, T.J. & Gauvin, D. (2000). Characterization of Cocaine Self-Administration and Pharmacokinetics as a Function of Time of Day in the rat. *Pharmacology Biochemistry and Behavior* 65: 289-299.

[12] Barabasz, A., et al. (2013). Efficacy of Single-Session Abreactive Ego State Therapy for Combat Stress Injury, PTSD, and ASD. *International Journal of Clinical & Experimentral Hypnosis* 61: 1-19.

[13] Barber, T.X. (1961). Experimental Evidence for a Theory of Hypnotic Behavior: II Experimental Controls in Hypnotic Age Regression. *International Journal of Clinical & Experimentral Hypnosis* 9: 181.

[14] Barber, T.X. (1962). Hypnotic Age Regression: A Critical Review. *Psychosomatic Medicine* 24: 286-299.

[15] Barber, J., & Mayer, D. (1977). Evaluation of the Efficacy and Neural Mechanism of a Hypnotic Analgesia Procedure in Experimental and Clinical Dental Pain. *Pain* 4: 41-48.

[16] Barbosa, R.A., et al. (2010). A Comparative Study Among Three Techniques of General Anesthesia for Ultrasound-Guided Transrectal Prostate Biopsy. *Revista Brasileira de Anestesiologia (ora Brazilian Journal of Anesthesiology)* 60: 457-465.

[17] Bartholini, G. (1985). GABA Receptor Agonists: Pharmacological Spectrum and Therapeutic Actions. *Medicinal Research Review* 5: 55-75.

[18] Beck, J., et al. (2021). Exposure to Relaxing Words During Sleep Promotes Slow-Wave Sleep and Subjective Sleep Quality. *Sleep* 44: 148.

[19] Bhattacharya, S., et al. (2017). Genetically Induced Retrograde Amnesia of Associative Memories After Neuroplastin Ablation. *Biological Psychiatry* 15; :124-135.

[20] Billig, M. (1997). The Dialogic Unconscious: Psychoanalysis, Discursive Psychology and the Nature of Repression. *British Journal of Social Psychology* 36: 139-159.

[21] Bisiach, E., & Vallar, G. (2000). Unilateral Neglect in Humans. In: Boller, F., et al. *Handbook of Neuropsychology*, Elservier, Amsterdam (2nd ed., vol. 1, pp. 459-502).

[22] Blanke, O., & Mohr, C. (2005). Out-of-Body Experience, Heautoscopy, and Autoscopic Hallucination of Neurological Origin. Implications for Neurocognitive Mechanisms of Corporeal Awareness and Self-Consciousness. *Brain Research. Brain Research Reviews* 50: 184-199.

[23] Bodemann, H.H. (1981). The Current Concept for the Cardiac Glycoside Receptor. *Clinical Cardiology* 4: 223-228.

[24] Braid J. (1843). *Neurypnology or the Rationale of Nervous Sleep Considered in Relation with Animal Magnetism. Illustrated by Numerous Cases of its Successful Application in the Relief and Cure of Disease*. Churchill, London.

[25] Braid J. (2009). Hypnotic Therapeutics. In: Braid J. *The Discovery of Hypnosis: The Complete Writings of James Braid.* National Council for Hypnotherapy, London.

[26] Brookshire, C.E., et al. (2014). Frequency, Nature, and Predictors of Alexia in a Convenience Sample of Individuals with Chronic Aphasia. *Aphasiology* 28: 1405-1421.

[27] Bruhn, J., et al. (2000). Bispectral Index (BIS) and Burst Suppression: Revealing a Part of the BIS Algorithm. *Journal of Clinical Monitoring and Computing* 16: 593-596.

[28] Buchser, E., et al. (1994). Hypnosis and Self-Hypnosis, Administered and Taught by Nurses, for the Reduction of Chronic Pain: A Controlled Clinical Trial. *Schweizerische Medizinische Wochenschrift* Suppl 62: 77-81.

[29] Burgmer, M., et al. (2013). The Mirror Neuron System Under Hypnosis - Brain Substrates of Voluntary and Involuntary Motor Activation in Hypnotic Paralysis. *Cortex* 49: 437-445.

[30] Caillois, R. (1956). *L'Incertitude Qui Vient des Rêves*. Gallimard, Paris. Or: The Modern Edition Gallimard, Coll. Idées, 1983.

[31] Callara, A.L., et al. (2023). Is Hypnotic Induction Necessary to Experience Hypnosis and Modify Autonomic Activity? *Brain Sciences* 13(6): 875.

[32] Cao, C.J., et al. (1989). Putative Cocaine Receptor in Striatum Is a Glycoprotein with Active Thiol Function. *Membrane Biochemistry* 8: 207-220.

[33] Cardinal, E., et al. (2026). Medical Hypnosis for Migraine Management: A Systematic Review. *Complementary Therapies in Medicine* 9: 103374.

[34] Casiglia, E. (2008). Hypnosis in the Theory of the Bicameral Mind. *The Jaynesian* 2008; 2: 12-14.

[35] Casiglia, E. (2020*). L'Io e le Sue Voi. Antropologia e Archeo-Psicologia della Coscienza Egoica*. CLEUP, Padova.

[36] Casiglia, E. (2024). *Dove e Come Agisce l'Ipnosi. Aree, Recettori e Sistemi Endogeni*. CLEUP, Padova.

[37] Casiglia, E. (2015). *Trattato d'Ipnosi e Altre Modificazioni di Coscienza*. CLEUP, Padova.

[38] Casiglia, E. (2015a). Notazioni Generali Sui Modelli e Sulla Tecnologia**.** Gigsaw Puzzle**.** In: Casiglia, E. et al: *Trattato d'Ipnosi e Altre Modificazioni di Coscienza*. CLEUP, Padova (pp. 567-584).

[39] Casiglia, E. (2022a). *Le Cose Accadono. Libero Arbitrio, Coscienza e Volontà, Capacità di Intendere e Volere del Reo e Libero Convincimento del Giudice in un'Ottica Deterministica*. CLEUP, Padova.

[40] Casiglia, E. (2022a). Consciousness, Hypnosis, and Free Will. In: Kuijsten M. *Conversations on Consciouscness and The Bicameral Mind. Interviewes With Leading Thinkers on Julian Jaynes's Theory*. Julian Jaynes Society. Henderson (pp. 200-216).

[41] Casiglia, E. (2023). Ipnosi in Criminologia. In: Conte M. *Manuale di Criminologia*. Youcanprint, Lecce (pp. 625-6813 + 695-701).

[42] Casiglia, E. (2025). *Ipnosi Veramente Moderna. Significato, Metodi, Tecniche, Coscienza, Volontà, Libertà, Antropologia e Intelligenza Artificiale*. CLEUP, Padova.

[43] Casiglia, E. (2025a). Where is Your Ego? A Population Study Over Males and Females Aged 5 To 68 Years Based on Anthropological Considerations. *Psychology* 16: 1034-1050.

[44] Casiglia, E., & Gadotti, E. (2026). Auto-Hypnosis: Defining the Phenomenon. *Advanced Social Science in Research Journal* 13: 32-48.

[45] Casiglia, E., & Gadotti, E. (2026a). Freedom, Volition, Ego Development, Personal Responsibility, and Internal Chattering Within Us. *Advanced Social Science in Research Journal* 13: 168-177.

[46] Casiglia, E., & Gadotti, E. (2026b). The Nature of Consciousness Characterized by Means of Hypnosis. *Advanced Social Science in Research Journal* 13: 188-207.

[47] Casiglia, E., & Gadotti, E. (2026c). Why It's So Easy to Hypnotize, and Why Hypnotizability Has Persisted to This Day – A Scientific and Anthropological Answer. *Advances in Social Sciences and Management* 2026; 4: 1-15.

[48] Casiglia, E., & Mazza, A. (2013). *Urgenze In Medicina. Teoria e Pratica***.** CLEUP, Padova.

[49] Casiglia, E., & Mentesana, L. (2018). Instrumental Demonstration of Hypnotic Flaccid Paralysis of Corrugator Muscles and its Use in Esthetic Medicine. *Sleep & Hypnosis* 20: 20-66.

[50] Casiglia, E., et al. (1986). *Farmacocinetica Clinica*. Cortina, Padova.

[51] Casiglia., E., et al. (1989). Antihypertensive Therapy Worsens Blood Flow in Arteriopathic Limbs. In: Strano, A., & Novo, S. *Advances in Vascular Pathology*, Vol. 2, Excerpta Medica, Amsterdam (pp. 961-962).

[53] Casiglia, E., et al. (1991). Hemodynamic Effects of Coffee and Caffeine in Normal Volunteers: A Placebo-Controlled Clinical Study. *Journal of Internernal Medicine* 229: 501-504.

[54] Casiglia, E., et al. (1992). Circadian Rhythm of Peripheral Resistance: A Non-Invasive 24-Hour Study in Young Normal Volunteers Confined In Bed. *High Blood Pressure* 1: 249-255.

[55] Casiglia, E., et al. (1994). Hypnosis as a Tool for Evaluating the Cortical Component of Haemodynamic Variations. Pilot Study During Blood Letting. *High Blood Pressure* 3 (Suppl.3): 8.

[56] Casiglia, E., et al. (1996). 24-Hour Rhythm of Blood Pressure and Forearm Peripheral Resistance in Normotensive and Hypertensive Subjects Confined to Bed. *Journal of Hypertension* 14: 47-52.

[57] Casiglia, E**.**, et al. (1997). Haemodynamics Following Real and Hypnosis–Simulated Phlebotomy. *American Journal of Clinical Hypnosis* 4: 368-375.

[58] Casiglia, E., et al. (1998). Leg Versus Forearm Flow: 24H Monitoring in 14 Normotensive Subjects and In 14 Age-Matched Hypertensive Patients Confined to Bed. *American Journal of Hypertension* 11: 190-195.

[59] Casiglia, E., et al. (2000). 24-Hour Rhythm of Blood Pressure Differs from that of Leg Hemodynamics in Orthotopic Heart Transplant Recipients. *American Journal of Hypertension* 140: 941-944.

[60] Casiglia, E., et al. (2006). Local and Systemic Vasodilation Following Hypnotic Suggestion of Warm Tub Bathing. *International Journal of Psychophysiology* 62: 60-65.

[61] Casiglia, E., et al. (2010). Neurophysiological Correlates of Post-Hypnotic Alexia. A Controlled Study with Stroop Test. *American Journal of Clinical Hypnosis* 52: 219-234.

[62] Casiglia, E., et al. (2012). Relaxation Versus Fractionation as Hypnotic Deepening: Do They Differ in Physiological Changes? *International Journal of Clinical & Experimentral Hypnosis* 60: 338-55.

[63] Casiglia, E., et al (2012a). Measured Outcomes with Hypnosis as an Experimental Tool in a Cardiovascular Physiology Laboratory. *International Journal of Clinical & Experimentral Hypnosis* 2012; 60: 241-261.

[64] Casiglia, E., et al. (2015). Hypnotic General Anesthesia vs. Focused Analgesia in Preventing Pain and its Cardiovascular Effects. *Athens Journal of Health* 3: 145-158.

[65] Casiglia, E., et al. (2015a). Allucinazioni Negative (Deficits Attentivi). In: Casiglia, E., et al: *Trattato d'Ipnosi e Altre Modificazioni di Coscienza*. CLEUP, Padova (pp. 603-610).

[66] Casiglia, E., et al. (2016). The Unconscious Experimentally Demonstrated by Means of Hypnosis. *Psychology* 7: 469-479.

[67] Casiglia, E., et al. (2017). Effects of Caffeine and Coffee on Incident Heart Failure in General Population. Role of the CYP1A2 -163C>A Polymorphism. *World Journal of Research and Review* 5: 42-48.

[68] Casiglia, E., et al. (2018). Functional Magnetic Resonance Imaging Demonstrates that Hypnosis is Conscious and Voluntary. *Psychology* 9: 1571-1581.

[69] Casiglia, E., et al. (2018a). Hypnotic Focused Analgesia Obtained Through Body Dysmorphism Prevents Both Pain and Its Cardiovascular Effects. *Sleep & Hypnosis* 19: 89-95.

[70] Casiglia, E., et al. (2018b). Experimental Approach to the Transmission of Information in Hypnosis. *Psychology* 9: 1-13.

[71] Casiglia, E., et al. (2018c). The Mysterious Hypnotic Analgesia: Experimental Evidences. *Psychology* 9: 1935-1956.

[72] Casiglia, E., et al. (2019). Plastic Monoideism Demonstrated by Functional Magnetic Resonance Imaging (fMRI). *Psychology* 10: 434-448.

[73] Casiglia, E., et al. (2020). Mechanisms of Hypnotic Analgesia Explained by Functional Magnetic Resonance (fMRI). *International Journal of Clinical & Experimentral Hypnosis* 2020; 68: 1-15

[74] Casiglia, E., et al. (2022). *Consensus Delle Scuole Italiane di Ipnosi Circa i Principï Generali dell'Ipnotismo e dell'Ipnosi*. Edizioni 31, Trento, 2022.

[75] Casiglia, E., & Tikhonoff, V. (2024). Sleep–Induced Limb Vasodilation in Individuals Confined to Bed for 24 H. *Brain & Heart* 2024; 2: 1886.

[76] Chen, K., et al. (2026). Simultaneous EEG and fMRI Investigation of Frontal Alpha Power And Frontal Alpha Asymmetry in Young Adults With a History of Major Depressive Disorder. *Journal of Affective Disorders* 398: 120905.

[77] Chen, A.C.N. (2001). New Perspectives in EEG/MEG Brain Mapping and PET/fMRI Neuroimaging of Human Pain. *International Journal of Psychophysiology* 42: 147-159.

[78] Chen, A.C.N., & Hermann, C. (2001). Perception of Pain Coincides With Spatial Expansion of EEG Dynamics in Human Subjects. *Neuroscience Letters* 297: 183-186.

[79] Chen, X. (2025). Correlation Between Job Burnout, Psychological Status, and Job Satisfaction Among Anesthesiologists in The Post-COVID-19 Era: A Cross-Sectional Study In China. *Frontiers in Public Health* 17; 13: 1555141.

[80] Clark, A. (2015). *Surfing Uncertainty: Prediction, Action, and the Embodied Mind*. Oxford University Press, Oxford.

[81] Clark, A., & Chalmers, D. (1998). The extended mind. *Analysis* 58: 7-19.

[82] Clark T. (1973). Cocaine. *Texas Medicine* 69: 74-78.

[83] Coe, W.C. (1973). Experimental Designs and the State-Nonstate Issue in Hypnosis. *American Journal of Clinical Hypnosis* 16: 118-28.

[84] Cojan, Y., et al. (2009). Motor Inhibition in Hysterical Conversion Paralysis. *Neuroimage* 47: 1016-1037.

[85] Conlong, P., & Rees, W. (1999). The Use of Hypnosis in Gastroscopy: a Comparison with Intravenous Sedation. *Postgraduate Medical Journal* 75: 223-225.

[86] Conn, J.H. (1972). Is Hypnosis Really Dangerous? *International Journal of Clinical & Experimentral Hypnosis* 20: 61-79.

[87] Cooper, L.M., & London, P. (1973). Reactivation of Memory by Hypnosis and Suggestion. *International Journal of Clinical & Experimentral Hypnosis* 21: 312-323.

[88] Cossu, G., et al. (2024). Hypnosis-Assisted Awake Craniotomy for Eloquent Brain Tumors: Advantages and Pitfalls. *Cancers* 16: 1784.

[89] Coulthard, P., & Craig, D. (1997). Conscious Sedation. *Dental Update* 24: 376-381.

[90] Cox, R.E., & Barnier, A.J. (2010). Hypnotic Illusions and Clinical Delusions: Hypnosis as a Research Method. *Cognitive Neuropsychiatry* 15: 202-232.

[91] Crandall, C.G, et al. (2008). Effects of Passive Heating on Central Blood Volume and Ventricular Dimensions in Humans. *Journal of Physiology* 586*:* 293-301.

[92] Craparo, G. (2013). Addiction, Dissociazione e Inconscio Non Rimosso. Un Contributo Teorico Secondo la Prospettiva Evolutivo-Relazionale. *Ricerca Psicoanalitica* 24: 73-84.

[93] Craparo, G., & Mucci, C. (2016). *Unexpressed Unconscious. Implicit Memory and Clinical Work*. Routledge, London.

[94] Cunningham, C.W., et al. (2011). Barker EL. Neuropharmacology of the Naturally Occurring κ-Opioid Hallucinogen Salvinorin A. *Pharmacological Reviews* 63: 316-347.

[95] Dahinterová J. (1967). Some Experiences with whe Use of Hypnosis in The Treatment of Burns. *International Journal of Clinical and Experimental Hypnosis* 15: 49-53.

[96] Dalayeun, J.F., et al (1993). Physiology of Beta-Endorphins. A Close-Up View and a Review of the literature. *Biomedicine & Pharmacotherapy* 47: 311-320.

[97] De Pascalis, V. (2024). Brain Functional Correlates of Resting Hypnosis and Hypnotizability: A Review. *Brain Sciences* 14, 115.

[98] De Pascalis, V., & De Benedittis, G. (2025). Experimental Pain Modulation *Via* Hypnosis: Neurophysiological Perspectives. *International Review of Neurobiology* 184: 83-127.

[99] Demertzi, A., et al. (2011). Hypnotic Modulation of Resting State fMRI Default Mode and Extrinsic Network connectivity. In: Van Someren, E.J.W., et al. *Progress in Brain Research*; Elsevier. Amsterdam (Vol. 193, pp. 309-322).

[100] Dong, M., et al. (2019). Botulinum and Tetanus Neurotoxins. *Annual Review of Biochemistry* 88: 811-837.

[101] Drake, L.R., & Scott, P.J.H. (2018). DARK Classics in Chemical Neuroscience: Cocaine. *ACS Chemical Neuroscience* 9: 2358-2372.

[102] Drake T.R., et al. (1990). Severe Acid-Base Abnormalities Associated with Cocaine Abuse. *The Journal of Emergency Medicine*. 8: 331-334.

[103] Drouet, N., & Chedeau G. (2017). Hypnopraxia, A New Hypnotic Technique for Hypnoanesthesia. *Journal of Clinical Anesthesia* 37: 14-16.

[104] Einstein, A. (1920). *Grundgedanken und Methoden der Relativitätstheorie in ihrer Entwicklung Dargestellt*, § 13. The Morgan Library & Museum di New York (Pierpont Morgan Library), *Autograph Manuscript.*

[105] Einstein, A. (2002). Grundgedanken und Methoden der Relativitätstheorie, in Ihrer Entwicklung Dargestellt. In: *The Collected Papers of Albert Einstein. Vol.7: The Berlin Years: Writings, 1918-1921*. Janssen M., et al. *Princeton University Press*, Princeton (Doc. 31, § 13, pp. 245-281).

[106] Eipel, C., et al. (2010). Regulation of Hepatic Blood Flow: The Hepatic Arterial Buffer Response Revisited. *World Journal of Gastroenterology* 16: 6046-6057.

[107] Elkins, G., et al. (2006). Hypnosis to Manage Anxiety and Pain Associated with Colonoscopy for Colorectal Cancer Screening: Case Studies and Possible Benefits. *International Journal of Clinical and Experimental Hypnosis* 54: 416-431.

[108] Elkins, G.R., et al. (2015). Advancing Research and Practice: The Revised APA Division 30 Definition of Hypnosis. *American Journal of Clinical Hypnosis* 57, 378-385.

[109] Ellenberger H. (1970. *The Discovery of the Unconscious: The History and Evolution of Dynamic Psychiatry*. Penguin Press, London.

[110] Ellis, A. (1958). Hypnotherapy with Borderline Schizophrenics. *The Journal of General Psychology* 59: 245-253.

[111] Ellis, H. (2015). Franz Mesmer: Pioneer in the Treatment of Functional Disease or Charlatan? *British Journal of Hospital Medicine* 76: 170.

[112] Emmett, M.R., et al. (2014). Integrative Biological Analysis for Neuropsychopharmacology. *Neuropsychopharmacolgy* 39: 5-23.

[113] Erickson, M.H. (1959). Further Clinical Techniques of Hypnosis: Utilization Techniques. *American Journal of Clinical Hypnosis* 2: 3.

[114] Erickson, M.H. (2008). Deep Hypnosis and its Induction. In: *The Collected Papers of Milton H. Erickson on Hypnosis*. Vol. 1: The Nature of Therapeutic Hypnosis. Rossi, E., et al. The Milton H. Erickson Press, Phoenix.

[115] Erickson, M.H., et al. (1959). A Transcript of a Trance Induction With Commentary. *American Journal of Clinical Hypnosis* 2: 49-84.

[116] Ernst, B.J., et al. (2015). The Physicochemical and Pharmacokinetic Relationships of Barbiturate. From the Past to the Future. *Current Pharmaceutical Design* 21: 3681-3691.

[119] Facco, E., et al. (2009). Effects of Hypnosis on Dental Pain Threshold. Preliminary Report. *Pain Practice* 9 (Suppl.1): 47-48.

[120] Facco, E, et al. (2011). Effects of Hypnotic Focused Analgesia on Dental Pain Threshold. *International Journal of Clinical and Experimental Hypnosis* 59: 454-468.

[121] Facco, E., et al. (2011a). The Effects of Hypnosis on Dental Pain Threshold. *International Journal of Oral and Maxiyllofacial Surgery* 2011; 40: 1058-1059.

[122] Facco, E., et al. (2013). Hypnosis as Sole Anaesthesia for Skin Tumour Removal in a Patient with Multiple Chemical Sensitivity. *Anaesthesia* 68: 961-965.

[123] Facco, E., et al. (2013a). Validation of Visual Analogue Scale for Anxiety (VAS-A) in Preanesthesia Evaluation. *Minerva Anestesiologica* 79: 1389-1395.

[124] Facco, E., et al. (2014). The Role of Hypnotherapy in Dentistry. *SAAD Dig*est *Journal of the Society for the Advancement of Anaesthesia in Dentistry* 2014; 30: 3-6.

[125] Facco, E., et al. (2014). Top-down Regulation of Left Temporal Cortex by Hypnotic Amusia for Rhythm: A Pilot Study on Mismatch Negativity. *International Journal of Clinical and Experimental Hypnosis* 62: 129-144.

[125] Facco, E., et al. (2022). Hypnosis and Hypnotic Ability Between Old Beliefs and New Evidences: An Epistemological Reflection. *American Journal of Clinical Hypnosis* 64: 20-35.

[126] Facco, E., et al. (2019). The Neurophenomenology of Out-Of-Body Experiences Induced by Hypnotic Suggestions. *International Journal of Clinical and Experimental Hypnosis* 67: 39-68.

[127] Facco, E., et al. (2021). Hypnosis as Sole Anesthesia for Oral Surgery: The Egg Of Columbus. *Journal of the American Dental Association* 152: 756-762.

[127] Farzampour, Z., et al. (2015). Endozepines. *Advances in Pharmacology* 2015; 72: 147-164.

[128] Fiorelli, M., et al. (1991). PET Studies of Cortical Diaschisis in Patients with Motor Hemi-Neglect. *Journal of the Neurolpgical Sciences* 104, 135-142.

[129] Forman, S.A. (2022). γ-Aminobutyric Acid Type A Receptor Subtypes and Circuit Connections in Midazolam-Induced Amnesia, Sedation, and Hypnosis. *Anesthesiology* 6: 880-882.

[130] Freud, S. (1941). *Abriss der Psychoanalyse*. Fischer Verlag. Frankfurt am Main.

[131] Freud S. (1964). *An Outline of Psycho-Analysis.* The Standard *Edition of the Complete Psychological Works of Sigmund Freud*. Hogarth Press, London.

[132] Frid, M., & Rana, C. (1979). Interactions Between Personal Expectations and Naloxone: Effects on Tolerance to Ischemic Pain. *Psychopharmacology* 65: 225-231.

[133] Frid, M., & Singer, G. (1979) Hypnotic Analgesia in Conditions of Stress is Partially Reversed by Naloxone. *Psychopharmacology* 63: 211-215.

[134] Frith, C. (2007). *Making up the Mind: How the Brain Creates Our Mental World*. Blackwell Publishing, Oxford.

[135] Fromm, E. (1965). Spontaneous Autohypnotic Age-Regression in a Nocturnal Dream. *International Journal of Clinical & Experimental Hypnosis* 13: 119-31.

[136] Fromm, E. (1970). Age regression with unexpected reappearance of a repressed childhood language. *International Journal of Clinical and Experimental Hypnosis* 18: 79-88.

[137] Gadotti, E., & Casiglia, E. (2026). Hypnosis and Predictive Models of Consciousness. *Advanced Social Science in Research Journal* 13: 146-159.

[138] Galilei, G. (1632). *Dialogo Sopra i Due Massimi Sistemi del Mondo*. Editore Giovanni Battista Landini, Firenze.

[139] Galilei, G. (1953). *Opere*. Ricciardi, Milano.

[140] Gava, R., et al. (1988). La Pletismografia ad Occlusione Venosa nelle Prime Fasi della Sperimentazione Farmacologica. *Le Basi Razionali della Terapia* 43: 33-35.

[141] Gebhard, J.W. (1961). Hypnotic Age Regression: A Review. *American Journal of Clinical Hypnosis* 3: 139-168.

[142] Giordano, N., et al. (2012). An Experimental Approach to Hypnotic Age Regression: Controlled Study Over 10 Healthy Participants. *Contemporary Hypnosis & Integrative Therapy* 29: 271-283.

[143] Gold, M., et al. (1994). Anosognosia for Hemiplegia: An Electrophysiologic Investigation of The Feed-Forward Hypothesis. *Neurology* 44, 1804-1808.

[144] Gonella, A. (2015). Aspetti Sperimentali della Regressione «Ad Un Tempo Irreale». In: Casiglia, E., et al. *Trattato d'Ipnosi e Altre Modificazioni di Coscienza*. CLEUP, Padova (pp. 639-647 + 663-665).

[145] Green, J.P., et al. Forging ahead: the 2003 APA Division 30 definition of hypnosis. *International Journal of Clinical & Experimental Hypnosis* 2005; 53: 259-264.

[146] Haggard, P., & Libet, B. (2001). Conscious Intention and Brain Activity. *Journal of Consciousness Studies* 11: 47-63.

[147] Hall, W.H., et al. (1967). Gastric Function During Hypnosis and Hypnotically-Induced Gastrointestinal Symptoms. *Journal of Psychosomatic Research* 11: 263-266.

[148] Halligan, P.W., et al. (2000). Imaging Hypnotic Paralysis: Implications for Conversion Hysteria. *Lancet* 355: 986-987.

[149] Hannah, B. (1980). *Jung: His Life and Work. A Biographical Memoir*. Shambhala Publ., New York / Boston.

[150] Hertenstein, E., et al. Cognitive Behavioral Therapy for Insomnia in Patients with Mental Disorders and Comorbid Insomnia: A Systematic Review and Meta-Analysis. *Sleep Medicine Review* 2022; 62: 101597.

[151] Hilgard, E.R. (1991). Suggestibility and Suggestions as Related to Hypnosis In: Schumaker JF. *Human Suggestibility: Advances in Theory, Research and Application*. Routledge, New York & London (pp. 37–58).

[152] Hoeft, F., et al. (2012). Functional Brain Basis of Hypnotizability. *Archives of General Psychiatry* 69, 1064-1072.

[153] Ibrahim, M.M. (1975). Localization of Lesions In Patients With Idiopathis Orthostatic Hypotension. *British Heart Journal* 37: 868-872.

[154] Iturriaga, H., et al. (2012). Frequent Evaluation to Improve Compliance in Patients Treated With Occlusion for Amblyopia: A Randomized Controlled Trial. *Binocular Vision & Strabology Quarterly Simms-Romano's* 27: 137-152.

[155] Jacobson, K.A., et al. (2022). Adenosine A2A Receptor Antagonists: From Caffeine to Selective Non-Xanthines. *British Journal of Pharmacology* 2022; 179: 3496-3511.

[156] Jacobson KA, Gao ZAG, Matricon P, Eddy MT, Carlsson J. Adenosine A2A Receptor Antagonists: From Caffeine to Selective Non-Xanthines. *British Journal of Pharmacology* 2022; 179: 3496-3511.

[157] Jacobson, E.D. & Pawlik, W.W. (1992). Adenosine Mediation of Mesenteric Blood Flow. *Journal of Physiology and Pharmacology* 43: 3-19.

[158] Johansen, J.W. (2006). Update on Bispectral Index Monitoring. *Best Practice & Research Clinical Anaesthesiology* 20: 81-99.

[159] Jamieson, G.A. (2015). Interoceptive predictive coding, active inference and the generation of hypnotic suggestion responses. *Inernational Review of Neurobiology* 185: 31-57.

[160] Jung, C.G. (2023). *Collected Works of C.G. Jung. The First Complete English Edition*. Adler, G., et al. Routledge, London.

[161] Kant, I. (1781). *Kritik der Reinen Vernunft*. Johann Friedrich Hartknoch, Riga.

[162] Karer, G., & Škrjanc, I. (2022). Improved Individualized Patient-Oriented Depth-of-Hypnosis Measurement Based on Bispectral Index. *Sensors* 23: 293.

[163] Kihlstrom, J.F. (1985). Hypnosis. *Annual Review of Psychology* 36: 385–418.

[164] Kinsbourne, M. (1993). Interactive Attentional Systems and Visual Neglect. In: I. H. Robertson, I.H., & J.C. Marshall J.C. *Unilateral Neglect: Clinical and Experimental Studies.* Lawrence Erlbaum, Hove, UK (pp. 63-86).

[165] Kirsch, I. (1997). Suggestibility or Hypnosis: What do Our Scales Really Measure? *International Journal of Clinical and Experimental Hypnosis* 45: 212-225.

[166] Kirsch, I., et al. (2011). Definitions of Hypnosis and Hypnotizability and their Relation to Suggestion and Suggestibility: A Consensus Statement. *Contemporary Hypnosis and Integrative Therapy* 28: 107-115.

[167] Kissin, I. (2000). Depth of Anesthesia and Bispectral Index Monitoring. *Anesthesia & Analgesia* 90: 1114-1117.

[168] Kline, M.V. (1961). Hypotic age regression. Some dynamic considerations in relation to its validity as a research technique. *Diseases of the Nervous System* 22 (Suppl): 118-120.

[169] Kluft, R.P. (2018). Freud's Rejection of Hypnosis, Part I: The Genesis of a Rift. *American Journal of Clinical Hypnosis* 60: 307-323.

[170] Kluft, R.P. (2018a). Freud's Rejection of Hypnosis, Part II: The Perpetuation of a Rift. *American Journal of Clinical Hypnosis* 60: 324-347.

[171] Knight, Z. (1995). The Healing Power Of The Unconscious: How Can We Understand Past Life Experiences in Psychotherapy? *South African Journal of Psychology* 25: 90-98.

[172] Kuhlmann, S.M., et al (2005). A Mindfulness-Based Stress Prevention Training for Medical Students (MediMind): Study Protocol for a Randomized Controlled Trial. *Trials* 16: 40-51.

[173] Landry, M., et al. (2017). Brain Correlates of Hypnosis: A Systematic Review and Meta-Analytic Exploration. *Neuroscience & Biobehavioral Reviews* 81: 75-98.

[174] Laplane, D., & Degos, J.D. (1983). Motor Neglect. *Journal of Neurology Neurosurgery & Psychiatry* 46: 152-158.

[175] Leff, A.P., et al. (2006). Structural Anatomy of Pure and Hemianopic Alexia. *Journal of Neurology Neurosurgery & Psychiatry* 77: 1004-1007.

[176] Leffler, A., et al. (2012). Local Anesthetic-Like Inhibition of Voltage-Gated Na^+ Channels by the partial μ-Opioid Receptor Agonist Buprenorphine. *Anesthesiology* 116: 1335-1146.

[177] Leung, F.W. (2008). Methods of Reducing Discomfort During Colonoscopy. *Digestive Diseases and Sciences* 53: 1462-1467.

[178] Libet, B. (1985). Unconscious Cerebral Initiative and the Role of Conscious Will in Voluntary Action. *Behavioral and Brain Sciences* 8: 529-566.

[179] Libet, B. (1999). Do We Have Free Will? *Journal of Consciousness Studies* 6: 47-57.

[180] Libet, B., et al. (1983). Time of Conscious Intention to Act in Relation to Onset of Cerebral Activity (Readiness-Potential). The Unconscious Initiation of a Freely Voluntary Act. *Brain* 106: 623-642.

[181] Lieberman, L.R. (1973). The State-Nonstate Issue in Hypnosis: a Closer Look. *American Journal of Clinical Hypnosis* 16: 129-131.

[182] Lucas, M., et al. (2025). On the State-Nonstate Theory of Hypnosis: Network and Topological EEG Findings. *Cerebral Cortex* 35: bhaf262.

[183] Machado Brito-da-Costa, A., et al. (2021). Pharmacokinetics and Pharmacodynamics of Salvinorin A and *Salvia Divinorum*: Clinical and Forensic Aspects. *Pharmaceuticals* 14, 116-152.

[184] MacLeod, C.M. (1991). Half a Century of Research on the Stroop Effect: an Integrative Review. *Psychological Bulletin, 109*, 163-203.

[185] Mack, A., & Rock, I. (1998). *Inattentional Blindness*. MIT Press. Cambridge, USA.

[186] Madsen, M.K., et al. (2019). Psychedelic Effects of Psilocybin Correlate with Serotonin 2A Receptor Occupancy and Plasma Psilocin Levels. *Neuropsychopharmacology* 44: 1328-1334.

[187] Maquet, P., et al. (1999). Functional Neuroanatomy of Hypnotic State. *Biological Psychiatry* 45, 327–333.

[188] Marshall, J.C., et al. (1997). The Functional Anatomy of a Hysterical Paralysis. *Cognition* 1997; 64**:** B1–8.

[189] Matsunaga, S., & Mikami, T. (2025). Motor Vehicle Collision Due to Left Hemineglect Associated with a Stroke. *New England Journal of Medicine* 18; 393: e41.

[190] McCaffrey, A.M., et al. (2004). Prayer for Health Concerns. Results of a National Survey on Prevalence and Patterns of Use. *Archives of Internal Medicine* 164: 858-862.

[191] McKay, M.T., et al. (2022). The McLean Screening Instrument for Borderline Personality Disorder: What, and WHy in a Clinical Cohort? *Psychiatry Research* 317: 114868.

[192] Meador, K.J., et al. (1988). Right Cerebral Specialization for Tactile Attention as Evidenced by Intracarotid Sodium Amytal. *Neurology* 38: 1763-1766.

[193] Melzack, R., & Wall, P. D. (1965). Pain Mechanisms: A New Theory. *Science* 150: 971-979. *[and also:]*

[194] Melzack, R., & Wall, P.D. (1991). Pain Mechanisms: A New Theory. *Science* 1965: 150: 971-979.

[195] Mercer, M., & Gibson, R.W. (1950). Rorschach content in hypnosis: chronological age level regression. *Journal of Clinical Psychology* 6: 352-358.

[196] Merighi, S., et al. (2022). A_{2A} Adenosine Receptor Antagonists in Neurodegenerative Diseases. *Current Medicinal Chemistry* 29: 4138-4151.

[197] Meurisse, M., et al. (1999). Hypnosis With Conscious Sedation Instead Of General Anaesthesia? Applications in Cervical Endocrine Surgery. *Acta Chirurgica Belgica* 99: 151-158.

[198] Miller, S.J., et al. (2011). African-Americans' and Latinos' Perceptions of Using Hypnosis to Alleviate Distress Before a Colonoscopy. *Contemporary Hypnosis & Integrative Therapy* 28: 196-203.

[199] Miroglio, G. (1992). Possibilità dell'Ipnosi Clinica nella Preparazione e nella Condotta del Paziente da Sottoporre ad Indagine Endoscopica. *Live Endoscopy '92*. Milano, 25-27 marzo.

[200] Miroglio, G. (1994). Videoteca della Società Italiana di Endoscopia Digestiva (Vol.16). *Menarini*, Firenze.

[201] Miroglio. G. et al. (1998). Effetti dell'Ipnosi sulla Motilità Gastro-Duodenale. *Atti del Simposio Nazionale della Società Italiana di Endoscopia Digestiva.* Saint Vincent (pp. 66-67).

[202] Möhler, H., et al. (2002). A New Benzodiazepine Pharmacology. *The Journal of Pharmacology & Experimental Therapeutics* 300: 2-8.

[203] Montis, S., et al. (2019). L'ipnosi nell'Ecocardiografia Transesofagea. L'Esperienza di Un'Unità di Cardiologia Pediatrica e delle Cardiopatie Congenite. *Giornale Italiano di Cardiologia* 20: 651-657.

[204] Moretti A, et al. (2025). Implementation of the Enhanced Recovery after Bariatric Surgery (ERABS) Protocol With Clinical Hypnosis: Effects on Pain and Postoperative Nausea and Vomiting (PONV) Management in Bariatric Surgery. *Surgical Endoscopy and Other Interventional Techniques* 39: 7324-7335.

[205] Moretti, A., et al. (2025). Correction: Implementation of the Enhanced Recovery after Bariatric Surgery (ERABS) Protocol With Clinical Hypnosis: Effects on Pain and Postoperative Nausea and Vomiting (PONV) Management in Bariatric Surgery. *Surgical Endoscopy and Other Interventional Techniques* 39: 7852.

[206] Mos, L., et al (1989). *Intraarterial Blood Pressure Recording During Underwater Activity*. EUBS 90, Bitterman N & Lincoln R Eds, Israeli Navy, 1989 (pp. 153-157).

[207] Murray-Jobsis J. (1991). An Exploratory Study of Hypnotic Capacity of Schizophrenic and Borderline Patients in a Clinical Setting. *American Journal of Clinical Hypnosis* 33: 150-160.

[208] Muschalla, B., & Schönborn, F. (2021). Induction of False Beliefs and False Memories in Laboratory Studies – A Systematic Review. *Clinical Psychology & Psychotherapy* 28:1194-1209.

[209] Naatanen, R. (1995). The Mismatch Negativity: A Powerful Tool for Cognitive Neuroscience. *Ear & Hearing* 16*:* 6-18.

[210] Nash, M.R., & Wong, A. (2011). Hypnosis in the Laboratory Creates a Window on Psychopathology. *International Journal of Clinical & Experimental Hypnosis* 59: 469-476.

[211] O'Neil, J.A. (2018). Hypnosis and Psychoanalysis: Toward Undoing Freud's Primal Category Mistake. *American Journal of Clinical Hypnosis* 60: 262-278.

[212] Ossipov, M.H., et al. (2010). Central Modulation of Pain. *Journal of Clinical Investigation* 120: 3779-3787.

[213] Parr, T., et al. (2022). Active Inference: The Free Energy Principle in Mind, Brain, and Behavior. *MIT Press*, Cambridge.

[214] Peretz, I., & Vuvan, D.T. (2017). Prevalence of Congenital Amusia. *European Journal of Human Genetics* 25: 625-630.

[215] Peretz, I., et al. (2007). The Genetics of Congenital Amusia (Tone Deafness): A Family Aggregation Study. *The American Journal of Human Genetics* 81: 582-588.

[216] Pernice, M., & Casiglia, E. (2015). Valutazione Strumentale della Profondità dell'Ipnosi. In: Casiglia, E., et al: *Trattato d'Ipnosi e Altre Modificazioni di Coscienza*. CLEUP, Padova (pp. 591-592).

[217] Perry C. (2004). Can Anecdotes Add to an Understanding of Hypnosis? *International Journal of Clinical & Experimental Hypnosis* 52: 218-231.

[218] Piché, M., et al. (2010). Dissection of Perceptual, Motor and Autonomic Components of Brain Activity Evoked by Noxious Stimulation. *Pain* 149: 453-462.

[219] Pilia, E., et al. (2022). A Clinical Operative Sequence for Hypnosis Implementation to General Anesthesia During Major Surgery for Orthotopic Liver Transplantation. *Annals of Medicine and Surgery* 4; 80: 104345.

[220] Podda, M., et al. (2017). A Prospective Non-Randomized Controlled, Multicenter Trial Comparing Appendectomy and Conservative Treatment for Patients with Uncomplicated Acute Appendicitis (the ACTUAA Study). *International Journal of Colorectal Disease* 32: 1649-1660.

[221] Podda, M., et al. (2021). Appendectomy Versus Conservative Treatment with Antibiotics for patients with Uncomplicated Acute Appendicitis: A Propensity Score-Matched Analysis of Patient-Centered Outcomes (the ACTUAA Prospective Multicenter Trial). *International Journal of Colorectal Disease* 36: 589-598.

[222] Podda, M., et al. (2021). Correction to: Appendectomy Versus Conservative Treatment with Antibiotics for patients with Uncomplicated Acute Appendicitis: A Propensity Score-Matched Analysis of Patient-Centered Outcomes (the ACTUAA Prospective Multicenter Trial). *International Journal of Colorectal Disease* 36: 599.

[223] Posner, M.I., et al. (1982). Neural Systems Control of Spatial Orienting. *Philosophical Transactions of the Royal Society of London, B, Biological Sciences* 298, 187-198.

[224] Posner, M.I., et al. (1987). How Do the Parietal Lobes Direct Covert Attention? *Neuropsychologia* 25, 135-145.

[225] Presciuttini, S., et al. (2018). Polymorphism of Opioid Receptors μ_1 in Highly Hypnotizable Subjects. *International Journal of Clinical & Experimental Hypnosis* 66: 106-118.

[226] Priftis, K., et al. (2011). Hypnosis Meets Neurosciences: Simulating Visuospatial Neglect in Healthy Participants. *Neuropsychologia* 49: 3346-3350.

[227] Qin, S., et al. (2020). Effectiveness and Safety of Massage in the Treatment of Anxiety and Depression in Patients with Cancer: A Protocol for Systematic Review and Meta-analysis. *Medicine* 99: 39 (e22262).

[228] Rainville, P. (2002). Brain Mechanisms of Pain Affect and Pain Modulation. *Current Opinion in Neurobiology* 12, 195-204.

[229] Rainville, P., et al. (1997). Pain Affect Encoded in Human Anterior Cingulated but not Somatodensory Cortex. *Science* 277: 968-971.

[230] Rainville, P., et al. (2012). Cerebral Mechanisms of Hypnotic Induction and Suggestion. *Journal of Cognitive Neuroscience* 11, 110-125.

[231] Rainville, P., et al. (2019). Hypnotic Automaticity in the Brain at Rest: An Arterial Spin Labelling Study. *International Journal of Clinical & Experimentral Hypnosis* 67: 512-542.

[232] Ramanchadran, V.S. (2003). *The Emerging Mind*. *BBC*, London.

[233] Rampil, I.J. (1998). A Primer for EEG Signal Processing in Anesthesia. *Anesthesiology* 1998; 89: 980-1002.

[234] Read, N.W. (2000). Bridging the Gap Between Mind and Body: Do Cultural and Psychoanalytical Concept of Visceral Disease Have an Explanation in Contemporary Neuroscience? *Progress in Brain Research* 122: 425-443.

[235] Regaldo, G. (2015). *Manuale di Ipnosi Medica Rapida. La Tecnica dDi Induzione Analizzata nei suoi Meccanismi per Renderla Efficace e Veloce con il Metodo Ipnosi R.A.P. Ratifica Appena P*ossibile. Regaldo G. ed., Torino. ISBN 9786050331561.

[236] Relinger, H. (1984). Hypnotic Hypermnesia: A Critical Review. *American Journal of Clinical Hypnosis* 26: 212-225.

[237] Rempelou, P., et al. (2015). Preventing Pain and its Hemodynamics Consequences Through Hypnosis: Hypnotic Focused Analgesia Vs. Hypnotic General Anesthesia. *ATINER's Conference Paper Series* 87-88 (ISSN 2241-2891).

[238] Rempelou, P., et al. (2016). Hypnotic Focused Analgesia and Hypnotic Focused Anaesthesia Prevent The Cardiovascular Effects of Pain. Pilot Study. *Journal of Hypertension* 32 (e-Suppl.1): e234.

[239] Ribeiro, J.A., & Sebastião, A.M. (2010). Caffeine and Adenosine. *Journal of Alzheimers Disease* 20 (Suppl 1): 3-15.

[240] Rivello, P. (2022). Prefazione a: Casiglia, E. *Le Cose Accadono. Libero Arbitrio, Coscienza e Volontà, Capacità di Intendere e Volere del Reo e Libero Convincimento del Giudice in un'ottica Deterministica*. CLEUP, Padova (pp. 7-20).

[241] Röder, C.H. (2007). Pain Response in Depersonalization: A Functional Imaging Study Using Hypnosis in Healthy Subjects. *Psychotherapy and Psychosomatics* 76: 115-121.

[242] Sadato, N., et al. (1996). Activation of the Primary Visual Cortex by Braille Reading in Blind Subjects. *Nature* 380: 526-528.

[243] Sánchez-Jáuregui, T., et al. (2019). Clinical hypnosis and music in breast biopsy: a randomized clinical trial. *American Journal of Clinical Hypnosis* 61: 244-257.

[244] Santoso, S., et al. (2025). Psychotherapeutic Modalities for Phantom Limb Pain: A Scoping Review. *East Asian Archives of Psychiatry* 35: 37-49.

[245] Sarbin, T.R. (1950). Mental Changes in Experimental Regression. *Journal of Personality* 19: 221-228.

[246] Sawynok, J. (2016). Adenosine Receptor Targets for Pain. *Neuroscience* 338: 1-18.

[247] Scagnelli, J. (1976). Hypnotherapy with Schizophrenic and Borderline Patients: Summary of Therapy with Eight Patients. *American Journal of Clinical Hypnosis* 19: 33-38.

[248] Scagnelli, J. (1980). Hypnotherapy with Psychotic and Borderline Patients: The Use of Trance by Patient and Therapist. *American Journal of Clinical Hypnosis* 22: 164-169.

[249] Sheehan, P.W. (1980). Factors Influencing Rapport in hypnosis. *Journal of Abnormal Psychology* 89: 563-581.

[250] Schnur, J.B., et al. (1992). Liver Biopsy Under Hypnosis. *Journal of Clinical Gastroenterology* 15: 122-124.

[251] Seth, A.K. (2013). Interoceptive Inference, Emotion, and the Embodied Self. *Trends in Cognitive Sciences* 17: 565-573.

[252] Seth, A.K. (2021). *Being You: A New Science of Consciousness*. Faber & Faber, London.

[253] Sigl, J.C., & Chamoun, N.G. (1994). An Introduction to Bispectral Analysis for the Electroencephalogram. *Journal of Clinical Monitoring* 10: 392-404.

[254] Soon, C.S., Brass, M., Hans-Jochen Heinze, H.J., Haynes, J.D. (2008). Unconscious Determinants of Free Decisions in the Human Brain. *Nature Neuroscience* 11: 543-545.

[255] Sosio, L. (2002). *Galileo Galilei. Dialogo Sopra i Due Massimi Sistemi Del Mondo*. Biblioteca Einaudi (pp. CIV-597).

[256] Spaños, N.P., et al. (1986). Hypnotic Interview and Age Regression Procedure in Elicitation of Multiple Personality Simptoms: A Simulation Study. *Psychiatry* 49: 298-311.

[257] Spaños, N.P., et al. (1991). Secondary Identity Enactments During Hypnotic Past-Life Regression: A Sociocognitive Perspective. *Journal of Personality and Social Psychology* 61: 308-320.

[258] Spiegel, H. (1972). An Eye-Roll Test for Hypnotizability. *American Journal of Clinical Hypnosis* 15: 25-28.

[259] Spiegel, H. (1974). *Manual for Hypnotic Induction Profile: Eye Roll Levitation Method*. Soni Medica, New York, 1974.

[260] Spiegel, H. (1974a). The Grade 5 Syndrome: The Highly Hypnotizable Person. *International Journal of Clinical & Experimental Hypnosis* 1974; 22: 303-319.

[261] Spiegel, H. (1977). The Hypnotic Induction Profile (HIP): A Review of its Development. *Annals of the New York Academy of Sciences* 296:129-142.

[262] Spiegel, H. (1980). Hypnosis and Evidence: Help or Hindrance? *Annals of the New York Academy of Sciences* 347: 73-85.

[263] Staples, E.A., & Wilensky, H. (1968). A Controlled Investigation of Hypnotic Age Regression. *Journal of Projective Techniques and Personality Assessment* 32: 246-252.

[264] Stöckigt, B., et al. (2021). Experiences and Perceived Effects of Rosary Praying: A Qualitative Study. *Journal of Religion and Health* 60: 3886-3906.

[265] Streeter, C.C., et al. (2012). Effects of Yoga on the Autonomic Nervous System, Gamma-Aminobutyric-Acid, and Allostasis in Epilepsy, Depression, and Post-Traumatic Stress Disorder. *Medical Hypotheses* 78: 571-579.

[266] Tallis, F. (2002). *Hidden Minds: A History of The Unconscious*. Arcade Publ., New York.

[267] Terao, T., & Collinson, S. (2000). Imaging Hypnotic Paralysis. *Lancet* 356: 162.

[268] Tikhonoff, V. (2007). L'ipnosi nella Ricerca Sperimentale e Clinica. In: Casiglia, E., et al. *Compendio di Ipnosi Sperimentale e Clinica*. Libreria Padovana Editrice, 2007.

[269] Tikhonoff, V., et al. (2012). Increase of Isometric Performance *Via* Hypnotic Suggestion: Experimental Study Over 10 Young Healthy Volunteers. *Contemporary Hypnosis & Integrative Therapy* 29: 352-262.

[270] Tikhonoff, V., et al. (2018). Hypnotic Hallucination of Body Heating Modifies Splanchnic Circulation: Haemodynamic and Ultrasonographic Pilot Study in Normal Volunteers. *Sleep & Hypnosis* 20: 31-39.

[271] Tirone, G. (1983). Lo «Stato Ipnotico»: Dimensione Psicofisica o Concetto Inutile? *Minerva Medica* 74: 2147-2151.

[272] Torre, E. (2008). *I Sentimenti e le Conflittualità del Perito*. Giuffrè, Milano.

[273] Tosello, M.T. (2008). Induzione dell'Ipnosi. In: Casiglia, E., & Rossi, A.M. *Teoria e Prarica dell'Ipnosi.* Libraria Padovana Editrice, Padova (pp. 51-53).

[274] Tosello, M.T. (2015). Breve Storia Evolutiva dell'Ipnosi. In: Casiglia, E. et al. (2015). *Trattato d'Ipnosi e Altre Modificazioni di Coscienza*. CLEUP, Padova (pp. 117-124).

[275] Tosello, M.T. (2015a). I Condizionamenti. In: Casiglia, E., et al. *Trattato d'Ipnosi e Altre Modificazioni di Coscienza*. CLEUP, Padova (pp. 717-729).

[276] Umanskiĭ. S.V., & Utkin, V.A. (2004). Особенности гипнотических состояний у больных с пограничными психическими расстройствами [The Peculiarities of Hypnotic States in Patients with Borderline Mental Disorders]. *Журнал неврологии и психиатрии имени С.С. Корсакова [Zhurnal Nevrologii i Psikhiatrii imeni S.S. Korsakova]* 104: 28-33.

[277] Van der Kruijs, S.J., et al. (2014). Neurophysiological Correlates of Dissociative Symptoms. *Journal of Neurology Neurosurgery and Psychiatry* 85: 174-179.

[278] Vázquez, P.G., et al. (2024). Brain Functional Connectivity of Hypnosis Without Target Suggestion – An Intrinsic Hypnosis rs-fMRI Study. *The World Journal of Biological Psychiatry* 25: 95-105.

[279] Vicente-Sanchez, A., et al. (2016). The Delta Opioid Receptor Tool Box. *Neuroscience* 338: 145-159.

[280] Vickers, A., & Zollman, C. (1999). Hypnosis and Relaxation Therapies. *British Medical Journal* 319: 1346-1349.

[281] Vignato, G. (2015). Simbologia del Cavo Orale. In: Casiglia, E., et al. (2015). *Trattato d'Ipnosi e Altre Modificazioni di Coscienza*. CLEUP, Padova (pp. 417-424).

[282] Vuilleumier, P. (2014). Brain Circuits Implicated in Psychogenic Paralysis in Conversion Disorders and Hypnosis. *Neurophysiologie Clinique* 44: 323-337.

[283] 和田淳 [Wada J]. (1949). Sodium Amytal 頸動脈注射の臨床実験的観察. [A New Method for the Determination of the Side of Cerebral Speech Dominance. A Preliminary Report on the intracarotid injection of Sodium Amytal in Man]. *医学と生物学 [Medicine and Biology]* 14: 221-222.

[284] Waddington, J.L., & Crow, T.J. (1976). A Behavioural Model of the GABA-Facilitating Action of Benzodiazepines: Rotational Behaviour after Unilateral Intranigral Injection of Chloridiazepoxide. *British Journal of Pharmacology* 58: 453P.

[285] Weitzenhoffer, A.M. (1980). Hypnotic Susceptibility Revisited. *American Journal of Clinical Hypnosis* 22: 130-146.

[286] Wells, W.R. (1924). Experiments in Waking Hypnosis for Instructional Purposes. *Journal of Abnormal and Social Psychology* 18: 389-404.

[287] Wilbur, C.B. (1960). Age Regression by Hypnosis. *Diseases of the Nervous System* 21: 520-526.

[288] Wilson I. (1981). *Mind Out of Time? Reincarnation Claims Investigated*. Gollancz, Londra.

[289] Winnicott, D.W. (1975). *Gli Aspetti Metapsicologici e Clinici della Regressione nell'Ambito della Situazione Psicoanalitica*. In: Dalla Pediatria alla psicoanalisi. Giunti-Barbera, Firenze.

[290] Wittkower, E.D. (1964). Treatment of Psychosomatic Disorders. *Canadian Medical Association Journal* 90: 1055-1060.

[291] Wobst, A.H. (2007). Hypnosis and Surgery: Past, Present, and Future. *Anesthesia & Analgesia* 104: 1199-1208.

[292] Wolf, T.G., et al. (2022). Efficacy of Hypnosis on Dental Anxiety and Phobia: A Systematic Review and Meta-Analysis. *Brain Sciences* 20; 521.

[293] Wunderlich, C.A. (1870). *Das Verhalten der Eigenwärme in Krankheiten.* Wigand, Leipzig.

[294] Zachariae, R., et al. (1998). Effects of an Opioid Antagonist on Pain Intensity and Withdrawal Reflexes During Induction of Hypnotic Analgesia in High- and Low-Hypnotizable Volunteers. *European Journal of Pain* 2: 25-34.

[295] Zech, N., et al. (2023). Hypnosis Measured With Monitors of Anesthetic Depth – EEG Changes During the Test for Harvard Group Scale of Hypnotic Susceptibility. *Frontiers in Psychoogy* 14: 1267658.

[296] Zhornitsky, S., et al. (2019). Midcingulate Cortical Activations Interrelate Chronic Craving and Physiological Responses to Negative Emotions in Cocaine Addiction. *Neuropsychopharmacology* 44: 1328-1334.

[297] Zimmerman, M, & Balling C. (2021). Screening for Borderline Personality Disorder With the McLean Screening Instrument: A Review and Critique of the Literature. *Journal of Personality Disorders* 35: 288-298.

[298] Zimmerman, M., et al. (2010). Screening for Bipolar Disorders and Finding Borderlin Personality Disorder. *Journal of Clinical Psychiatry* 71: 1212-1217.

[299] Zimmermann, M., et al. (2019). Screening for Bipolar Disorder and Finding Borderline Personality Disorder: A Replication and Extension. *Journal of Personality Disorders* 33: 633-543.

[300] Zimmerman M, et al. (2019a). Screening for Borderline Personality Disorder in Psychiatric Outpatients With Major Depressive Disorder and Bipolar Disorder. *Journal of Clinical Psychiatry* 15; 80: 18m12257.

[301] Zorzan, M., & Rossetto, O. (2022). Molecular Mechanisms of Muscular and Non-Muscular Actions of neuromuscular blocking agents. *British Journal of Anaesthesia* 129: 841-845.

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