Measured Outcomes With Hypnosis as an Experimental Tool in a Cardiovascular Physiology Laboratory

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MEASURED OUTCOMES WITH HYPNOSIS AS AN EXPERIMENTAL TOOL IN A CARDIOVASCULAR PHYSIOLOGY LABORATORY1

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Abstract: The authors detail their multidisciplinary collaboration of cardiologists, physiologists, neurologists, psychologists, engineers, and statisticians in researching the effects of hypnosis on the cardiovascular system and their additions to that incomplete literature. The article details their results and provides guidelines for researchers interested in replicating their research on hypnosis’ effect on the cardiovascular system.

Hypnosis is mainly employed in a psychotherapeutic setting, while a limited number of researchers deal with hypnosis in an experimental context. The investigations with hypnosis require the integrated cooperation of different scientists, such as cardiologists, physiologists,

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neurologists, statisticians, engineers, etc., collaborating with hypnotists in a research-oriented setting.

For physiologists and cardiologists, hypnosis is a very useful experimental tool as its mono-ideisms are experienced as real and have physical consequences that can be repeated and measured (Casiglia et al., 1994). Our group is particularly interested in the clinically measurable, repeatable, physical consequences of hypnosis and performed a coordinated set of experiments on this topic (Casiglia et al., 1997; Casiglia et al., 2006; Casiglia et al., 2007; Casiglia et al., 2010; Facco et al., 2009; Facco et al., 2011). The main results of these studies were used to prepare the present article of integrative theory showing our experience in the field of experimental hypnosis. The aim of this article is to enable other researchers to replicate our experiments and to supply a survey of the answers that the researcher can expect when experimenting with hypnosis in the cardiovascular field.

**How We Get Hypnosis**

After an explanation about the procedure the participants are asked to relax in response to verbal commands. They are all highly hypnotizable (De Pascalis, Russo, & Marrucci, 2000). Through the voice of an expert hypnotist, they are guided towards focusing their attention on a single idea, excluding any other external or internal stimuli.

A traditional hypnotic form of induction and deepening, consisting in a brief enumeration coupled with suggestions of general well-being, eyelid heaviness, regular deep breathing, and staring at a point, is most commonly used in our laboratory. After spontaneous eyelid closure is obtained, subjects are invited to concentrate on their own body from head to foot, whilst a feeling of heaviness and muscular relaxation is being suggested. Alternatively, nonrelaxing techniques consisting of rapid sequences of induction→de-hypnotization→re-induction (Hammond, Haskins-Bartsch, McGhee, & Grant, 1987; Klein, 2010) are used when thought proper (see below).

The check of hypnosis is commonly based on some signals, such as arm levitation, the easing of facial tension, a dropped lower jaw with a slight opening of the mouth, the slowing down of breathing rate, and a slight eyelid flutter.

To reduce the time needed for induction, a posthypnotic conditioning is predisposed in all our volunteers during the preliminary setting. A subject who is in hypnosis tends to accept and carry out the commands given by the hypnotist. When these commands are programmed to be executed after de-hypnotization, they are called “posthypnotic conditioning.” A posthypnotic command is therefore a suggestion made during hypnosis indicating that a particular experience will occur
on cue following the termination of the hypnotic session (Raz, 2005). After the de-hypnotization, the subject automatically adheres to the posthypnotic commands. An important posthypnotic conditioning is that of being prone to immediately fall into a hypnotic state under request: This conditioning reduces the time needed for induction, making the procedure easier and shorter. This command is very useful in an experimental context as well as in medical and dental clinical practice.

**Arterial Blood Flow**

Arterial blood flow indexed for the volume of tissue (in \( \text{mL} \times \text{min}^{-1} \times \text{dL}^{-1} \)) is measured at a forearm and/or at a leg with a strain-gauge plethysmograph (Angioflow, Microlab Electronics, Padova, Italy). This method entails periodic occlusion of venous outflow by a cuff automatically inflated at over-venous and under-diastolic pressure, while the arm’s volume is measured by indium-gallium-indium gels. In such conditions, the segmental blood inflow is proportional to arterial flow, allowing real-time detection of peripheral flow (Casiglia, 1996). This method has been widely employed by our group in different conditions and in different subjects, such as normotensive persons (Casiglia, Palatini, et al., 1998) and patients with arterial hypertension (Casiglia, Palatini, et al., 1998; Casiglia, Staessen, et al., 1998), spinal cord injury (Casiglia et al., 1999), and heart transplant (Casiglia et al., 2000).

Blood pressure was formerly measured (in mmHg) by an automatic Omron 705 IT device (Omron Europe, Hoofddorp, Netherlands), while in recent years, we changed to the Finometer recorder (Finapress Medical Systems, Amsterdam, Netherlands), a standalone solution for accurate automatic blood pressure monitoring also providing heart rate recording without any action by operators. This device records the blood pressure curve by a finger cuff; the filtered pressor curve is used to build the beat-to-beat values of systolic, diastolic, and mean blood pressure in the brachial artery.

The amount of blood ejected from the left ventricle at each systole (stroke volume, in mL) and in one minute (cardiac output, in \( \text{L} \times \text{min}^{-1} \)) is measured by means of a cardiograph featuring enhanced bio-impedance signal morphology analysis by means of six leads positioned on the subject’s thorax (PhysioFlow TM-Lab-1, Manatec Biomedical, Ebersviller, France) (Richard et al., 2001). Cardiac index (in \( \text{L} \times \text{min}^{-1} \times \text{m}^{-2} \)) is calculated by indexing cardiac output by body surface area (Du Bois & Du Bois, 1916).

Forearm peripheral resistance is calculated (in \( \text{mmHg} \times \text{min} \times \text{mL}^{-1} \)) from the mean blood pressure/forearm flow ratio and total
peripheral resistance (in mmHg × min × L$^{-1}$ × m$^2$) from mean blood pressure/cardiac output ratio (Casiglia, 1996).

These methods have been validated and largely tested by our research group in many experimental conditions (Casiglia et al., 1996; Casiglia, Palatini, et al., 1998; Casiglia, Staessen, et al., 1998; Casiglia et al., 1999; Casiglia et al., 2000) including hypnosis (Casiglia et al., 1997; Casiglia et al., 2006; Casiglia et al., 2010; Facco et al., 2009, 2011).

**HOW WE GET STATISTICS**

A preliminary power analysis is always performed to be sure that the sample taken into consideration is large enough to avoid $\beta$ error. Analysis of variance is used to compare continuous variables, while we are prevented against the $t$ test because the samples are often numbers less than 30 subjects or the distribution is not normal; for these reasons, or because they are not independent of each other when included as covariates in a regression, variables are in many cases log-transformed before they are used in statistical analysis. The Bonferroni’s post hoc correction is always applied. Frequencies are usually compared with the chi$^2$ test. The null hypothesis is rejected for values of $p < .05$. Logistic regression is used when searching for items predicting a dichotomic categorical variable.

**OUR EXPERIMENTAL MODELS WITH HYPNOSIS**

*Cardiovascular Response to Hypnotic Induction*

As hypnosis is mainly reached through suggestions of relaxation and well-being, the general belief is that the hypnotic induction is a relaxed and relaxing phase. Nevertheless, when we started our experiments with hypnosis, we realized that this had never been demonstrated in an experimental setting, and that the possibility that induction might be a neutral or even a stressing experience did exist.

We therefore decided to explore this field of research. After monitoring the cardiovascular system with the above-mentioned devices, we noted that the forearm and total peripheral resistance, blood pressure, heart rate, and cardiac index were comparable before and immediately after a hypnotic induction (Table 1).

Although the general belief that the hypnotic induction is a relaxing phase, our conclusion is that induction is hemodynamically neutral. The feeling of relaxation usually experienced during induction is therefore only subjective and due to the fact that hypnosis is normally reached by suggestions of well-being and active relaxation.
Table 1
Cardiovascular Effects of Hypnotic Induction

<table>
<thead>
<tr>
<th>value</th>
<th>Before Induction</th>
<th>After Induction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP (mmHg)</td>
<td>121.9 ± 12.2</td>
<td>121.7 ± 11.4</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>73.2 ± 7.7</td>
<td>73.4 ± 8.1</td>
</tr>
<tr>
<td>Total PR (mmHg×min×L−1)</td>
<td>34.3 ± 14.8</td>
<td>34.5 ± 14.7</td>
</tr>
<tr>
<td>Cardiac index (L × min−1 × m−2)</td>
<td>3.0 ± 1.1</td>
<td>3.1 ± 1.3</td>
</tr>
<tr>
<td>Leg PR (mmHg×min×dL×mL−1)</td>
<td>32.1 ± 13.8</td>
<td>29.7 ± 15.0</td>
</tr>
<tr>
<td>Peripheral flow (mL×min−1×dL−1)</td>
<td>3.4 ± 1.5</td>
<td>3.9 ± 1.8</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>78.8 ± 14.4</td>
<td>77.4 ± 9.0</td>
</tr>
</tbody>
</table>

Notes. Hemodynamics before and immediately after induction in 60 healthy volunteers. No significant difference detected. BP = arterial blood pressure; PR = peripheral resistance.

**CARDIOVASCULAR RESPONSE TO HYPNOTIC DEEPENING**

Deepening is a crucial phase in every hypnotic session. Although classically obtained through suggestions of relaxation, leading to a profound and pleasant feeling of well-being, deepening can also be achieved through nonrelaxing techniques. After spending many years inducing and deepening volunteers with slow techniques based on relaxation, we decided to test a rapid technique for deepening hypnosis without relaxation in an experimental context.

A rapid sequence of induction→de-hypnotization→re-induction (Hammond et al., 1987; Klein, 2010) repeated for 3 minutes was used to reach a hypnotic state without relaxation in 20 healthy volunteers. The highly impressive power of this nonrelaxing technique is explained by the fact that the hypnotic trance is always accompanied in turn by hyper-hypnotizability: When a subject comes out of a hypnotic trance, he or she remains for a few moments in an unusual state of modified consciousness. In such conditions, it is extremely easy to re-induce hypnosis without going through the classic relaxation technique (Klein, 2010). This method was compared in the same subjects, in a Latin-square protocol, to a relaxing deepening state reached through ideas of well-being, regular deep breathing, feeling of heaviness, and muscular relaxation.

Forearm peripheral resistance increased by 51% after deepening without relaxation, while it remained unchanged after deepening with relaxation (Figure 1).

Although a well-conducted hypnosis is always accompanied by a subjective feeling of relaxation independently of the techniques that are used from time to time, deepening without suggestions of relaxation is associated to a significant and important increase of peripheral
resistance, while the relaxing techniques are free from this adverse effect. While induction per se is probably hemodynamically neutral, hypnotic deepening is not, and must be conducted carefully, gently, and smoothly, suggesting relaxation, to avoid an increase in the sympathetic drive, peripheral resistance, and postload that can be dangerous in coronary patients and in the elderly.

Cardiovascular Response to a Situational Hallucination. Can hallucinations be experimentally demonstrated? Hypnosis-induced hallucinations are a very interesting experimental model. In hypnosis, it is possible to administer special suggestions (thermal, tactile, visual, auditory, coenaesthesis) that rise to special relevance and can be experienced as belonging to real world (Bryant & Mallard, 2005) owing to the high level of attention and to the characteristics of modified consciousness. Our first model was the hallucination of a phlebotomy. In some studies available in literature, blood pressure had been found

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Figure 1. Forearm vasoconstriction during hypnotic deepening. Variation of forearm peripheral resistance (PR) in 12 healthy subjects during hypnotic deepening with and without suggestions of relaxation. Relaxation limits the vasoconstrictor effect of hypnotic deepening, preventing the PR increase due to rise in sympathetic drive. The symbol * indicates $p < .01$ vs. deepening without relaxation.
to increase (Kuchel, Avorn, Reed, & Fields, 1992), decrease (Casthely, Yoganathan, Salem, & Karyanis, 1990; Kirwan, Chir, Scrr, & Brindle Smith, 1981), or not change (Abraham et al., 1992; Marsh & Cooper, 1992) after a blood loss of 250–500 mL. Information on peripheral flow and peripheral resistance during and after phlebotomy was sparse. A transient reduction of the internal carotid flow had been observed after removing 400 mL of blood (Silvani et al., 1990), the cardiac output had been found to increase and total peripheral resistance to decrease insignificantly after a 500 mL removal (W. F. Kennedy et al., 1968), and the limb flow had been studied only in subgroups of patients (Ford, Berent, Speed, Rose, & Dormandy, 1978; Milligan, Tooke, & Davies, 1982; Yates, Berent, Andrews, & Dormandy, 1979). Furthermore, the question of whether the hemodynamic response to phlebotomy depended directly on the loss of blood or rather on the mental involvement in the ritual of bloodletting remained unanswered.

We decided to organize a study (Casiglia et al., 1997) aimed at evaluating whether or not phlebotomy was capable of inducing peripheral hemodynamic variations, and whether such variations, if any, were secondary to the hydraulic blood of loss or, rather, to the mental component of the procedure. We felt that the tool “hypnosis” was the right way to answer this question. Twenty-two informed-consent neophyte blood donors were studied. The procedure included three distinct phases: stabilization, bloodletting, and recovery. They were placed in bed, in a comfortable semi-darkened room, to minimize the effect of external stimuli. Hemodynamics were continuously monitored. Subjects underwent phlebotomy with the removal of 300 mL blood in 6 minutes. Hemodynamics were measured at 3-minute intervals. Immediately after the end of bloodletting (6th minute from baseline) the needle was removed. Patients remained in bed for a further 18 minutes and the measurements of BP and flow were repeated every 3 minutes. In 22 other informed-consent subjects, age and sex-matched with the blood donors, who never underwent phlebotomy, the ritual of bloodletting was simulated by hypnotic hallucination (Crawford, 1994; A. Kennedy, 1957; Kunnunen, Zamansky, & Block, 1994) without any real subtraction of blood. This procedure was aimed at evaluating the role of mind in determining the hemodynamic variations, if any, that accompanied phlebotomy, that is, at separating the mental from the hydraulic effects of bloodletting. The subjective feeling of the reality of this experience was confirmed a posteriori by all subjects. The same 22 subjects who were suggested during the hypnotic trance also underwent the BP flow measurement with the same timing described above, without any intervention (either phlebotomy or hypnosis). This sham procedure was aimed at showing the effects of ongoing time and those due to cuff inflation and was performed in half of the subjects
1 week before and in the other half 1 week after hypnosis, according to a randomized list.

Interestingly, the increase in peripheral resistance observed during the study was comparable in subjects who had undergone a real phlebotomy with the loss of 300 mL blood and in those who had undergone a hallucinated phlebotomy without any blood loss, while in control subjects no variation of leg resistance was observed (Figure 2).

The removal of 300 mL venous blood during a bloodletting is an excellent experimental model of acute, controlled hemorrhaging in man. Blood loss is followed by an increase in plasma renin activity, aldosterone, adrenocorticotropic hormone, and norepinephrine as a consequence of an increased sympathetic discharge (Casiglia, Angeli, De Toni, Gatta, & Pessina, 1990; Casiglia et al., 1989; Kuchel et al., 1992; O'Shaugnessy & Slome, 1934). Although classic physiology attributes this phenomenon to hypovolemia, the results of our study with hypnosis demonstrate that peripheral resistance increases not only in subjects who really experiment bloodletting (hypovolemia) but also in those who hallucinate a bloodletting (no hypovolemia). Subjects were able to embody the content of suggestions (Santarcangelo et al., 2010) and to behave a stressing situation. This demonstrates that the cardiovascular response to bloodletting may simply be due to the mental participation to the event and not to the hydraulic subtraction of blood. This

**Figure 2.** Leg vasoconstriction during real and hallucinated phlebotomy. Variation of leg peripheral resistance (PR) in 22 volunteer blood donors undergoing 300 mL bloodletting (-●-), and in age-matched volunteers hallucinating bloodletting without any real blood loss (-□-). A comparable degree of vasoconstriction was observed in the two groups, indicating that the cardiovascular consequences of bloodletting were simply due to the procedure. No variation during sham procedure (controls, -▲-). The symbol * indicates $p < .01$ vs. -●- and vs. -□-.
is not surprising, and is in keeping with the observation that, when a normal subject stands up from the supine position, 300 mL blood are pooled into legs by force of gravity and subtracted from circulation, like in bloodletting, without any appreciable cardiovascular response (Casiglia et al., 1997). In fact, there is no emotional participation in standing up, and 300 mL blood is not sufficient to produce a reflex response. Confirmation could come from bloodletting during anesthesia, avoiding subjective participation to the procedure, but to our knowledge no experimental study has been published in this field. So, hypnotic hallucination remains up to date the only experimental tool really used to discriminate between emotional and hydraulic effects of phlebotomy.

Cardiovascular Response to a Thermal Hallucination

Thermal hallucination is easy to be obtained in a hypnotic context (Hayek, Jakoubek, Kyhos, & Radil, 1992). Nevertheless, when we tackled this problem (Casiglia et al., 2006), it was unclear whether the feeling of heat referred by subjects after receiving this specific command was real or the expression of a merely subjective experience. It was decided to use segmental vasodilation, if any, as a marker for warming. In fact, segmental temperature can only increase through a rise in arterial blood flow, and measuring segmental flow is a more sensitive maneuver than measuring skin temperature.

Once a valid neutral hypnotic trance was obtained in 12 volunteers, the special suggestions aimed at obtaining thermal hallucinations were administered. Warming of the right forearm was suggested by hallucinating bathing in a warm tub. Forearm peripheral resistance and arterial flow were contextually monitored throughout the study with the devices described above in order to ascertain whether the increase of temperature of the segment, if any, was attributable to vasodilation. Blood pressure, heart rate, cardiac index, and stroke volume were also simultaneously monitored to clarify whether the local vasodilation, if any, derived from systemic modifications or rather from redistribution of the blood flow; a possibility that had previously been demonstrated by our group in different conditions (Casiglia et al., 1996; Casiglia, Palatini, et al., 1998; Casiglia, Staessen, et al., 1999; Casiglia et al., 2000).

After the end of the above described experiment (30th minute) and after reaching hemodynamic stability, a whole-body immersion in a bathtub of warm water for 30 minutes was hallucinated, while the parameters specified above were once again continuously monitored. The aim of this procedure was to give evidence to the cardiovascular modifications due to thermal systemic vasodilation, if any, that is, the reduction of total peripheral resistance. At the end of this period, the subject’s usual consciousness was gradually restored and the subjects
were de-hypnotized. As a control, on a different day, the same subjects underwent a sham procedure represented by a simple induction and resting in bed, without any suggestion but relaxation, to observe if neutral hypnosis produced any cardiovascular effects.

During the hallucination of a forearm in warm water, significant and biologically important local vasodilation was detected, with a 21% decrease of forearm peripheral resistance and an 18% increase of forearm blood flow, as in a real local passive warming (Figure 3a).

During the hallucination of the whole-body in a warm water tub, there was a significant and biologically important systemic vasodilation with a 20% decrease of total peripheral resistance and a 28% increase of cardiac index, as in a real total-body passive warming (Figure 3b). During the neutral hypnosis, no hemodynamic variations were observed.

**Figure 3.** Local and systemic vasodilation during hallucination of heat. (a) Reduction of forearm peripheral resistance (PR) in 12 healthy volunteers hallucinating forearm heating. (b) Reduction of total PR in the same volunteers hallucinating total-body heating (warm tub bathing). No variation during sham procedure (controls without hypnosis). The symbol * indicates $p < .01$ vs. hallucinated warming.
These results clarify that the feeling of heat that can be hallucinated with hypnotic suggestions is real and objective and is due to blood redistribution and vasodilation implying thermal dispersion (it cannot be used, for instance, to warm a frozen person).

**Hypnotic Analgesia.** A very special field of research with hypnosis is represented by pain modulation (Chaves & Dworkin, 1997; Holroyd, 1996). The efficacy of hypnosis in this respect is well documented (Chaves, 1994; Howland, Nakamura, & Cleeland, 1995; Kiernan, Dane, Phillips, & Price, 1995; Miller & Bawers, 1993; Rainville, 2008; Zachariae & Bjerring, 1994), and neuroimaging demonstrated that hypnotic analgesia is mediated by the anterior cingulate cortex (Faymonville et al., 2000). Nevertheless, many papers on this topic, also performed using functional magnetic resonance, are focused on sensation of pain (Derbyshire, Whalley, & Oakley, 2009), while we were more interested in its cardiovascular consequences. We believed that studying the unconscious cardiovascular effects of pain would help us to clarify whether, during hypnotic analgesia, pain was really blocked at a certain level of the nervous system, rather than simply dissociated from consciousness (Hilgard, 1977).

**Nontrigeminal pain** is the most frequent form of nociception, representing the painful stimuli coming from all parts of the body except the mouth and the nose. To clarify whether hypnotic analgesia was a mere inhibitory hallucination/dissociation (not accompanied by cardiovascular modifications) or rather represented a real block of painful stimuli (accompanied by reduction of sympathetic cardiovascular response to pain), we conceived a study aimed at quantifying hypnotic focused analgesia (HFA) and its ability to block not only pain but also its cardiovascular consequences.

The nontrigeminal pain accompanying the cold pressor test—a painful maneuver commonly used in hemodynamics lab—was employed as a pain-generator. Twenty subjects first underwent a cold pressor test in basal conditions by putting their right hand in a tub containing icy water (0°C). As is well known, this procedure induces local pain that is accompanied by an increase in systolic blood pressure, total peripheral resistance, and heart rate and a decrease in cardiac output (Peckerman, Saab, & McCabe, 1991; Peckerman et al., 1994; Sendowski et al., 2000; Sevre & Rostrup, 1999). The pain reached at the end of cold pressor test was quantified by both rating a 0–10 subjective scale (Dirks, Wunder, Kinsman, McElhinny, & Jones, 1993) and measuring the pain tolerance threshold in seconds. For this latter purpose, the participants were asked to keep their right hand in the icy water tub as long as possible; only when pain became intolerable were they allowed to interrupt the test (Langlade, Jussiau, Lamonerie, Marret, & Bonnet, 2002). The pain-induced variations of the cardiovascular parameters
were recorded as a measure of the reflex consequences of the pain. The same day, after the hemodynamic stabilization was reached, the same participants were asked to repeat the same procedure while undergoing HFA. Once a valid neutral hypnosis was induced, it was suggested that their right hand was insensitive to pain. The pain intensity and the pain tolerance threshold were recorded once more, while the hemodynamic parameters were continuously monitored before and during the entire painful procedure.

When the test was conducted in basal conditions without any suggestion of analgesia, the length of the test was $121.5 \pm 96.1$ seconds, the maximum rating score of the pain measured immediately before the subject asked to interrupt the test was $7.75 \pm 2.29$, and the total peripheral resistance progressively increased in comparison to the baseline reaching $+32\%$ (Figure 4, dashed line) due to the systemic arteriolar constriction in response to the pain. When the test was repeated in HFA, it lasted $411.0 \pm 186.7$ seconds ($p < .0001$ vs. the same test performed without analgesia) with a subjective score of $2.45 \pm 2.98$ ($p < .0001$ vs. without), and the total peripheral resistance remained unchanged (Figure 4, solid line).

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**Figure 4.** Hypnotic focused analgesia (HFA) for nontrigeminal pain. Twenty volunteers in normal state of consciousness keeping a hand in icy water in the frame of cold pressor test (CPT) experiment increase of total peripheral resistance (PR), a typical response to nontrigeminal pain. When the experiment is repeated in conditions of analgesia (CPT+HFA), no significant vasoconstriction is observed. The symbol * indicates $p < .01$ vs. . . . .
Our results (Casiglia et al., 2007) proved that HFA was really reached, as the subjective perception of pain was more than reduced by half and the objective pain more than doubled. Not only this, but a certain degree of blocking the painful stimuli along the nervous ways was reached, as the reflex vasoconstriction accompanying the pain was reduced. The cardiovascular reflexes that are responsible for the pain-induced increase of peripheral resistance act at a lower level than that of the consciousness, so a simple dissociation cannot be an explanation for this reduction. Our results are therefore in agreement with the models that involve a block of nociceptive stimuli at the dorsal horn of the spinal cord (Kiernan et al., 1995; Sandrini et al., 2000), such as the gate control theory (Melzach & Wall, 1965). The gating is modulated by selective cognitive processes transmitted through descending fibers that could be activated during HFA (Miller & Bawers, 1993). On the contrary, our results are in disagreement with the dissociative models assuming that hypnosis simply acts by dissociating pain experiences from conscious awareness (Croft, Williams, Haenschel, & Gruzelier, 2002; Hilgard, 1977). In fact, if hypnosis merely dissociated painful experience from consciousness, a normal cardiovascular reflex response to pain would occur and an increase in peripheral resistance would be recorded during HFA as in basal conditions.

Nevertheless, we have no explanation about the mechanisms underlying the pain block. Using cold pressor test + HFA as in our studies, other authors observed electroencephalographic shifts in hemispheric dominance of high-frequency band power, confirming an active attentional/inhibitory processing in the anterior temporal cortex, with a shift towards greater power in the right hemisphere and an aspecific inhibition feedback circuit regulating the thalamo-cortical activity (Becker & Yingling, 1998). Our results demonstrate that hypnotic analgesia really blocks the nontrigeminal painful stimuli at a certain level of the nervous pathways, fully legitimating its use in surgery and in painful maneuvers. HFA, in fact, prevents not only the pain but also its cardiovascular consequences, pledging neuroprotection during painful procedures.

*Trigeminal pain* is different from the nontrigeminal pain in that, it is accompanied by vasodilation + bradycardia rather than by vasoconstriction + tachycardia (Prabhakar, Ali, & Rath, 2008). This is important in dental practice (Patel, Potter, & Mellor, 2000), where syncope is sometimes experienced following painful trigeminal stimulation. When we began experimenting with hypnosis, very little was known about the cardiovascular response to trigeminal pain, and practically no information was available about the possibility to prevent it by hypnotic suggestions.

We conceived a couple of studies (Facco et al., 2009; Facco et al., 2011) aimed at quantifying the effect of HFA during the electrical stimulation
of the teeth, where trains of electrical impulses of increasing intensity were applied to the first premolar of each side of the jaw in 31 healthy volunteers (a Digitest Serial D-22663 03/2005, Parkell Inc., Edgewood, USA was used). The basal pain threshold was measured asking the subjects to identify the moment when the pain appeared during stimulation. HFA in the right low arch was then suggested during hypnosis and the test was repeated in each first premolar.

The pain threshold was in average three times higher during HFA than at baseline and was significantly higher in the site of analgesia when compared to the contralateral one. Furthermore, preliminary data of a study in progress suggest that in the baseline condition the tooth electrical stimulation yields a mild decrease of peripheral resistance (about 9%), which is prevented by HFA (Figure 5); however, these data are to be checked in further study.

These findings confirm the analgesic power of hypnosis and show that HFA adds a significant local increase of pain threshold; during HFA the decrease of trigeminal pain perception does not seem to be only a matter of dissociation from consciousness but a real protection from pain, since its cardiovascular consequences seem to be prevented. This is of importance in dental practice, where hypnosis

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**Figure 5.** Hypnotic focused analgesia (HFA) for trigeminal pain. In thirty-two volunteers in normal state of consciousness receiving a train of electrical impulses on tooth 44, a reduction of total peripheral resistance (PR)—a typical reflex response to trigeminal pain—is shown. When the experiment is repeated in conditions of analgesia (pain+HFA), no vasodilation is observed. The symbol * indicates \( p < .01 \) versus baseline.
may stand in comparison with pharmacological sedation, limiting the risk of syncope and offering an adequate neuro- and organ-protection.

Both in trigeminal and nontrigeminal pain, HFA was not limited to dissociation but really blocked the painful stimuli at a certain level of the nervous system, pledging full protection to the brain and the heart.

Neuropsychological Tasks as Stressors. Many tasks commonly used for evaluating neuropsychological performance or for other special aims have stressor effects with cardiovascular consequences. We analyzed this topic by means of a test of global performance and the Stroop test.

For global performance we used a jigsaw puzzle. After the hemodynamic stability was reached in the seated posture, the participants had to complete the jigsaw in the shortest possible time while the hemodynamics were monitored, both in the normal state of consciousness and in the posthypnotic conditioning “be performing.” When in the posthypnotic command, the subjects completed the jigsaw in 21.6 ± 3.1 minutes, a time that was 20% lower than in the usual consciousness state \((p < .01)\). The reflex sympathetic response to the task was reduced in the posthypnotic state with a suggestion of high performance.

Total peripheral resistance increased by 1.7% in basal conditions, while they significantly decreased by 13.6% when the test was performed in the posthypnotic state (Figure 6). Heart rate, a further index of activation of the sympathetic drive, increased in the basal conditions \((+1.7\%)\) and decreased in the posthypnotic conditioning of high performance \((-2.5\%)\). The results demonstrate that, through a posthypnotic command, it is possible and easy to increase global performance, probably through an increase in concentration and previsualization (Crawford & Allen, 1983; Hill & Redden, 1984). Not only this, but also that a stressor can be tackled with more relaxation under hypnosis. The cognitive task increases the sympathetic drive probably as a response to increased cerebral metabolic demand. The suggestions of relaxation and well-being administered together with the posthypnotic task, as well as the subjective feeling of ability, might reduce the stressing effect of the task.

Furthermore, when we used the Stroop test in 6 volunteers to demonstrate that hypnosis can deactivate the metaphor of written language inducing hypnotic alexia (Casiglia et al., 2010), we observed that this task was a strong stressor able to induce in basal nonhypnotic conditions a sharp increase of peripheral resistance \((8\%, p < .01)\). On the contrary, in the posthypnotic phase with alexia no hemodynamic variation was observed (Figure 7). As a matter of fact, the reflex reaction to the Stroop task as a stressor was abolished when the incongruence between color and word was reduced.
Figure 6. Total peripheral resistance (PR) detected during a jigsaw puzzle in state of usual consciousness and in posthypnotic conditioning of increased global performance. The symbol * indicates $p < .05$ vs. □.

Figure 7. Variation of total peripheral resistance (PR) during Stroop test in basal conditions (without alexia) and in conditions of hypnotic alexia in 6 highly hypnotizable healthy volunteers. The symbol * indicates $p < .01$ vs. basal conditions. The Stroop test is a stressor. Hypnotic alexia abolishes the word/color incongruence, preventing the stressing effect of the task.
Conclusions

Hypnosis is an excellent tool for performing experimental research in a multidisciplinary context in a laboratory of cardiovascular physiology. In some cases, it allows questions raised by classic physiology to be answered. Nevertheless, all the studies with hypnosis, including those described in this integrative theory paper, are limited, the dimensions of the samples, the use of highly hypnotizable subjects, and the Hawthorne-Rosenthal factor always should be confirmed by replication.

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Gemessene Ergebnisse mit Hypnose als experimentelles Werkzeug in einem kardiovaskulären Labor

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Évaluation de résultats obtenus à l’aide de l’hypnose en tant qu’outil expérimental dans un laboratoire de physiologie cardiovasculaire

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Résumé: Les auteurs exposent en détail leur collaboration multidisciplinaire de cardiologues, physiologues, neurologues, psychologues, ingénieurs et statisticiens dans leur recherche des effets de l’hypnose sur le système cardiovasculaire et présentent leur apport à cette documentation encore incomplète. Cet article détaille leurs résultats et fournit des lignes de conduite aux
chercheurs désireux de reproduire ces recherches sur l’effet de l’hypnose sur le système cardiovasculaire.

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Mediciones de resultados de la hipnosis como herramienta experimental en un laboratorio de fisiología cardiovascular

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Resumen: Los autores detallan la colaboración multidisciplinaria de cardiólogos, fisiólogos, neurólogos, psicólogos, ingenieros y estadistas en la investigación de efectos de la hipnosis en el sistema cardiovascular y sus aportaciones a una literatura incompleta. El artículo detalla sus resultados y provee lineamientos para investigadores interesados en replicar el estudio sobre los efectos de la hipnosis en el sistema cardiovascular.

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