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This paper should be cited as follows:

Casiglia, E., Rempelou, P., Tikhonoff, V., Giacomello, M., Lapenta, A.M., Finatti, F., Favaro, J., Facco, E. (2016). "Hypnotic Focused Analgesia
Obtained Through Body Dysmorphism Prevents Pain and its Cardiovascular Effects", Athens: ATINER'S Conference Paper Series, No: HSC2016-2080.

Athens Institute for Education and Research 8 Valaoritou Street, Kolonaki, 10671 Athens, Greece Tel: + 30 210 3634210 Fax: + 30 210 3634209 Email: info@atiner.gr URL: www.atiner.gr URL Conference Papers Series: www.atiner.gr/papers.htm Printed in Athens, Greece by the Athens Institute for Education and Research. All rights reserved. Reproduction is allowed for non-commercial purposes if the source is fully acknowledged. ISSN: 2241-2891 12/12/2016

## Hypnotic Focused Analgesia Obtained Through Body Dysmorphism Prevents Pain and its Cardiovascular Effects

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#### Abstract

Hypnotic focused analgesia, comparable to chemical local anesthesia, has been widely documented in our Laboratory after hypnotic suggestions. This study is aimed at producing hypnotic local anesthesia suggesting that a hand does not belong to the body (body dismorphism) without any direct suggestions of analgesia.

Eight healthy, highly hypnotizable volunteers underwent a cold pressor test keeping left hand at 0 °C, a painful maneuver, being free to stop the test at any time. Such procedure was repeated after hypnotic induction with suggestion of dismorphism. The highest pain reached at the first minute and at the end of the experiment, both in prehypnotic conditions and during dismorphism, was subjectively quantified through a decimal visual scale. The objective measure of local anesthesia was based on time of tolerance and on reflex response to pain.

During dismorphism, pain perception was 92.5% lower at 1st minute and 87.5% lower at the end of the experiment (highest tolerable pain) than in prehypnotic conditions, and nullified in 5 subjects (62%). Tolerance to pain (minutes of voluntary immersion in icy water) increased by 315%. While in prehypnotic conditions pain produced a reflex increase in blood pressure, heart rate and resistance, no increase was found during dismorphism.

Hypnotic dismorphism without any specific suggestion of analgesia reduced and often nullified subjective pain perception. Objective pain tolerance contextually raised, and the reflex stimulation of the sympathetic drive was prevented. Analgesia produced through hypnotic dismorphism is therefore not a mere consequence of dissociation but a real physiological phenomenon.

**Keywords:** Dismorphism, Cardiovascular pain reflexes, Hypnotic local anesthesia, Subjective pain perception

#### Introduction

In the last years the Laboratory of Experimental Hypnosis of the Department of Medicine of the University of Padua in collaboration with the Institute Franco Granone of Turin (Italy) has demonstrated that, through suggestions given to a subject in deep hypnosis, it is possible to induce cognitive modifications which can be studied in controlled experimental setting (Casiglia et al., 1997; Casiglia et al., 2006; Casiglia et al., 2007; Casiglia et al., 2010; Casiglia et al., 2012a; Casiglia et al., 2012b; Casiglia et al., 2015; Facco et al., 2009; Tikhonoff et al., 2012). Thus, it has been shown that the effects of the hypnotic suggestions are not merely subjective but induce objective cerebral and physiological modifications that are real in everyday physical world. One of the most interesting modifications induced by hypnosis is pain control (Casiglia et al., 2012; Casiglia et al., 2015; Facco et al., 2009; Tikhonoff et al., 2012b; Casiglia et al., 2015; Facco et al., 2009; Casiglia et al., 2012b; Casiglia et al., 2015; Facco et al., 2009; Casiglia et al., 2012b; Casiglia et al., 2015; Facco et al., 2009; Casiglia et al., 2012b; Casiglia et al., 2015; Facco et al., 2009; Casiglia et al., 2012b; Casiglia et al., 2015; Facco et al., 2009; Casiglia et al., 2012b; Casiglia et al., 2015; Facco et al., 2009; Tikhonoff et al., 2012b; Casiglia et al., 2015; Facco et al., 2009; Tikhonoff et al., 2012b; Casiglia et al., 2015; Facco et al., 2009; Tikhonoff et al., 2012b; Casiglia et al., 2015; Facco et al., 2009; Tikhonoff et al., 2012b; Casiglia et al., 2015; Facco et al., 2009; Tikhonoff et al., 2012; Chaves et al., 1997; Holroyd, 1996).

Hypnotic focused analgesia (Casiglia et al., 2007; Casiglia et al., 2015; Facco et al., 2009), comparable to chemical local anesthesia, is an attentive deficit induced *via* hypnotic or posthypnotic commands (Chaves et al., 1997; Holroyd, 1996). We recently showed that, through specific hypnotic commands, analgesia can also be extended to the whole body and associated to narcosis, muscular paralysis (except for respiratory muscles) and amnesia, reproducing *via* hypnosis the picture of spontaneous-breathing general anesthesia or deep sedation (Casiglia et al., 2015).

Although the anesthetic effect of such suggestions is well documented, there is still open discussion on the underlying mechanisms and the chain of events bringing to analgesia, in particular if it represents a mere negative hallucination on dissociative basis, or a real block in pain transmission. Many procedures belonging to the field of human physiology can answer this question. Point-rating visual scales have been questioned because they are subjective and can be manipulated by the participant (Mader et al., 2003), but pain has reflex cardiovascular effects (vasoconstriction with increase of peripheral resistances) that cannot be simulated (Casiglia et al., 2010). Showing that hypnotic local anesthesia reduces the reflex cardiovascular effects of pain would demonstrate that pain has been really blocked in its transmission from the painful area to the brain, as these reflexes are carried by autonomic branches which do not lay under cortical control.

The experimental research described herein examined the possibility of producing local anesthesia by creating a hypnotic dismorphism with modification of body representation, without any direct suggestion of analgesia. During dismorphism, a hand was excluded from brain representation. Purpose of this research was to evaluate whether or not this exclusion would contextually exclude the painful sensations coming from the hand.

#### **Materials and Methods**

#### Subjects

The study included 8 healthy volunteers, who had been defined suitable for hypnosis on the basis of personal anamnesis, an interview and a personality test (Minnesota Multiphasic Personality Inventory) (Hathaway et al., 1985; Butcher et al., 1989). This preliminary procedure was used to screen and exclude subjects more prone to develop unwanted secondary effects to hypnotic dissociation. The general characteristics of the volunteers were those of a population of healthy young adults (Casiglia et al., 1997; Casiglia et al., 2006; Casiglia et al., 2007; Casiglia et al., 2010; Casiglia et al., 2012; Casiglia et al., 2012b; Casiglia et al., 2015; Facco et al., 2009; Tikhonoff et al., 2012).

#### Ethics

The study was approved by the Ethics Committee of the University Hospital of Padua and was conducted according to the principles of the Declaration of Helsinki for Human Research (41<sup>st</sup> World Medical Assembly, 1990). Every subject had been previously and individually informed in an appropriate place and with necessary time about the purpose, the execution and the possible risks of the procedure. The subjects were free to ask all questions for a complete comprehension of the procedure. All participants gave valid informed consent and signed a form also according to the Italian Law 675/1996 and to the Law of the Veneto Region 34/2007.

#### Preliminary Session

Hypnotizability was previously ascertained through the Italian version of the Harvard Group Scale of Hypnotic Susceptibility (De Pascalis et al., 2000; Younger, 2005). Highly hypnotizable subjects were selected for the research, since previous studies have shown that they tend to show stronger experiences of hypnotic pain inhibition (Horton et al., 2004).

Before the experimental procedure, each volunteer individually underwent a hypnotic induction, in order to establish an interpersonal rapport between the hypnologist and the subject himself. The purpose of the session was that of rapidly reaching an effective ideoplastic monoideism before the ensuing session of experimental measuring. Induction was brief (under 3 minutes) as usual at the Padua Laboratory. Before dehypnotization, a posthypnotic conditioning was left in order to obtain very rapid hypnotic trance in occasion of the experimental setting. This posthypnotic conditioning was then removed at the end of the whole experimental procedure.

#### **Experimental Session**

The experimental session was performed two days after the preliminary one. During the first 20 minutes of the session, the subjects were held in a supine position, while the devices necessary for hemodynamic monitoring were applied. Once hemodynamic stability was reached, basal hemodynamic parameters were measured.

Each subject then underwent a painful stimulus through immersion of left hand in icy water at a temperature of  $0^{\circ}$  C. As known, this procedure (cold pressor test) (Casiglia et al., 2007; Casiglia et al., 2012a; Laura et al., 2004; Peckerman et al., 1998; Sevre et al., 1999), commonly employed as a stressor by cardiologists, is accompanied by an ischemic pain which is usually tolerated for few minutes only. Such test, when applied to subjects free to interrupt the procedure in every moment, presents three advantages: 1) it can be standardized, 2) its duration reflects pain tolerance, and 3) its hemodynamic consequences can easily be measured, thus providing another demonstration of the reduction or resolution of the painful stimulus.

At the end of basal measurements, the posthypnotic command was recalled placing subject in hypnotic trance. Body dismorphism with absence of left hand was then obtained. In practical terms, it was suggested that the left hand, the wrist and the forearm up to the elbow no longer belonged to the body, as if they had been temporarily detached and placed upon an operating table where they would be subjected to the experiment. No direct suggestion of analgesia was given.

At the end of the experiment, the body representation was restored, while hemodynamic parameters continued to be registered until the eyes opening and complete dehypnotization.

#### Hemodynamic Monitoring

In all subjects blood pressure (mmHg) was measured at right upper-arm through an automatic oscillometric device (705 IT, Omron, Hoofddorp, The Netherlands). At the same time, cardiac output  $(1 \times \text{min}^{-1})$  was automatically calculated with a TM-Lab-1 (PhysioFlow, Medatec, Ebersvillier, France) from the stroke volume (ml) x heart rate (beats×min<sup>-1</sup>) product. Arteriolar resistance was calculated in units of resistance (UR = mmHg×min×l<sup>-1</sup>) from the ratio between systolic blood pressure and cardiac output. The purpose of these measurements was to verify whether during body dismorphism the painful stimulus was accompanied by activation of the sympathetic nervous system analogue to those observed during the usual state of consciousness or, on the contrary, the hemodynamic response to pain was reduced or absent.

#### **Statistics**

Preliminary power analysis based upon previous experience of our Laboratory showed that 8 subjects were sufficient to determine the hemodynamic modification avoiding  $\beta$  error. The continuous variables were expressed as mean  $\pm$  standard deviation and compared with the analysis of variance and the Bonferroni's post-hoc test. The null hypothesis was rejected for a probability <0.05.

#### Results

#### Subjective Quantification of Pain

In the prehypnotic basal conditions, subjective pain perception reported by subjects was  $7.9\pm2.1$  (CI 6.5 to 9.3) at the first minute and  $9.5\pm0.7$  (CI 9.1 to 10.0) at the end of the test. During hypnotic dismorphism, subjective pain perception was reduced to  $0.5\pm1.5$  (CI -0.5 to 1.5, p<0.001 vs. prehypnosis) at the first minute, and to  $2.0\pm3.2$  (CI -0.2 to 4.2, p<0.01 vs. prehypnosis) at the end of the test. In 5 of the 8 subjects (62%) pain perception was reported to be nullified (complete local anesthesia).

#### **Objective Quantification of Pain Tolerance**

The maximal duration of the immersion of the hand in the icy water, which expressed the objective pain tolerance and which in prehypnotic conditions was  $4.8\pm2.1$  minutes (CI 3.2 to 6.5), significantly increased during hypnotic dismorphism to  $15.1\pm8.3$  min (CI 9.8 to 20.4, p<0.001 vs. pre-hypnosis).

#### Hemodynamic Monitoring

The values of arterial pressure, heart rate, stroke volume, cardiac output and arteriolar resistance during the different stages of the study are summarized in Table 1.

In basal prehypnotic conditions, systolic (+12%, p<0.05), diastolic (+12%) and mean blood pressure (+11%, p<0.005), as well as heart rate (+17%, p<0.05), increased in response to pain at the 1<sup>st</sup> minute of pain. Systolic resistance also increased by 8%, although insignificantly because of a contextual increase of cardiac output due to both heart rate and stroke volume rise secondary to sympathetic activation. At the end of the experiment, the increases were respectively +11% (p<0.005), +11% (p<0.005), +12% (p<0.05) and +7%.

When hypnotic dismorphism was operating, no increase of the above mentioned parameters was observed. Resistance even tended to be reduced (-3% at the  $1^{st}$  minute) rather than increased.

#### ATINER CONFERENCE PAPER SERIES No: HSC2016-2080

stimulation of left hand, both in usual consciousness (prehypnosis) and during hypnotic dysmorphism of the left hand. BP: arterial blood pressure. CO: cardiac output. SR: systolic resistance. $p<0.05$ , $p<0.005$ vs. Basal			
Parameter	Basal	1 minute CPT	End CPT
	Prehypnosis (usual consciousness)		
Systolic BP (mmHg)	123.7±8.7	138.3±14.5*	137.0±12.0**
Diastolic BP (mmHg)	74.5±5.7	$84.8 \pm 8.1$	82.8±7.4
Mean BP (mmHg)	$107.3 \pm 7.0$	120.4±11.9**	119.0±9.5**
Heart rate (bpm)	76.4±12.7	89.4±20.6*	85.6±15.4*
Stroke volume (ml)	69.2±10.8	64.3±14.1	62.4±13.8

5.9±2.6

 $23.5 \pm 8.8$ 

Hypnotic dismorphism

 $81.8 \pm 8.3$ 

 $82.9 \pm 18.0$ 

66.3±13.2

5.5±1.8

23.3±7.0

134.6±9.5

 $117.0\pm8.3$ 

5.5±1.7

23.3±6.8

 $131.5 \pm 8.8$ 

79.4±7.5 114.2±13.6

75.8±13.6

64.6±10.9

5.0±1.5

 $24.3 \pm 5.4$ 

5.1±1.2

21.8±3.9

126.3±9.3

75.8±7.1

 $109.4 \pm 7.6$ 

75.6±13.5

63.4±9.3

4.8±1.1

24.0±5.0

**Table 1.** Peripheral hemodynamics in basal conditions and during cold

#### Discussion

SR (UR)

 $CO(1 \times min^{-1})$ 

MBP (mmHg)

 $CO(1 \times min^{-1})$ 

Heart rate (bpm)

Stroke volume (ml)

Systolic BP (mmHg)

Diastolic BP (mmHg)

SR (UR)

The results support the notion that dismorphism-induced analgesia, being able not only to reduce and in most cases to nullify the subjective pain perception, but also to prevent its neurovegetative effects, is accompanied by (and probably due to) a real block of painful stimuli, and is therefore comparable to that previously obtained in our Laboratory through direct suggestion of analgesia (Facco et al., 2009; Chaves et al., 1997). Evidently, the exclusion of the hand from the body scheme was sufficient to produce a classic HFA, even though no specific suggestions of analgesia were given.

The mechanism underlying HFA is still uncertain. It has been suggested it could be the result of a mere dissociation, while other Authors suggested a real block of the painful stimuli along the nervous system. The present study with dismorphism, like previously ones conducted in our Laboratory with specific suggestions of analgesia (Casiglia et al., 2007; Casiglia et al. 2012a; Casiglia et al., 2015; Facco et al., 2009), tips the scale in favor of real antalgic block rather than of dissociation. In fact, if, during dismorphism, pain were simply dissociated from consciousness, its cardiovascular consequences would be fully kept and free of act, since the sympathetic mechanisms responsible for the reflex increase in peripheral resistance work on a lower level than that subjective consciousness and are not under the control of the I, regardless of the fact that the I itself is dissociated or not.

On the contrary, the reduction of reflex increase in peripheral resistance observed during pain in conditions of hypnotic dismorphism points toward a real block of pain at certain level of the nervous system. The results of the present study therefore fully embraces the models that consider correct the hypotheses of a reduction of the nociceptive stimuli during hypnotic analgesia, such as those involving the so-called gate control theory (Kiernan et al., 1995; Sandrini et al., 2000). To be honest, the concrete physiological mechanism which blocks pain during hypnotic dismorphism can only be object of speculation. The gate control theory hypothesizes the existence of a gating mechanism in the dorsal horn of the spinal cord, able to modulate the transmission of nociceptive signals. Selective cognitive processes transmitted by descending fibers might modulate the properties of the spinal cord giving concreteness to the gating. And in fact, modifications of the spinal nociceptive reflexes R<sub>III</sub> have actually been registered during hypnosis (Hilgard, 1977). It is also possible that hypnosis works on multiple levels, as pain is a multidimensional experience that involves not only sensorial but also dissociative, motivational, cognitive and emotional factors. Hypnotic suggestions could therefore work by modulating not only  $R_{III}$  in the spinal cord but also the sensation of pain above and beyond  $R_{III}$ (Kiernan et al., 1995). The down-regulation of pain and of its effects requires a reduction of the polysynaptic spinal reflexes and a cortical reshaping of the affective and perceptive dimension of pain. Finally, conscious and unconscious control processes are certainly associated to a supervision system of attentive nature involving the frontotemporal cortex and the limbic system.

In the light of the present results, the so-called dissociative models, which acknowledge a mere hypnotic dissociation (Gruzelier et al., 1998; Croft et al., 2002) in the genesis of the hypnotic analgesia, must be rejected. According to such theories, pain is still registered in conditions of hypnotic anesthesia, but remains dissociated from the critical and emotional consciousness and masked by an amnesic barrier (Hilgard et al., 1977; Hilgard et al., 1979; Holroyd et al., 1996; Pribram, 1991; Crawford et al., 1990; Becker & Yiling, 1998). It is true that a certain level of dissociation is inherent to hypnosis, even when neutral, but, if this were the only mechanism implied in the dismorphic analgesia, a normal cardiovascular reflex response should be registered.

The sociocognitive theory, in which hypnotic analgesia is attributed to a sort of distraction of attention (Spanos et al., 1984; Spanos et al. 1979; Chapman & Nakamura, 1998), must be rejected as well. In fact in such model too no reduction of the cardiovascular pain reflexes could be observed.

For the same reason we must reject the constructivistic theory as an explanation of dismorphic analgesia. Followers of this theory believe that, since consciousness has limited capacities, only a single scheme of reality (i. e. one that excludes consciousness of pain) can occupy it in every moment (Du Bois & Du Bois, 1916). In this case as well, however, differently from what we observed in our experience, the cardiovascular reflex response to pain should be fully kept during pain.

Unfortunately, the absolute lack of data in literature about local analgesia induced by body dismorphism does not allow further discussion of this topic.

In conclusion, the results of the present study demonstrate once more that an important local anesthesia can be reached though hypnotic suggestions. They also confirm the data previously obtained with cold pressor test and with other painful maneuvers by the Laboratory where this thesis was conducted. In fact, subjective perception of pain measured with an analogue scale during body dismorphism with absence of the left hand was 92.5% lower at the first minute and 87.5% lower at maximal stimulation than it was during prehypnotic basal conditions. Moreover, it was found to be null in 5 out of 8 cases. Pain tolerance expressed as voluntary duration of the immersion in icy water was 31.5% higher in dismorphic conditions than in prehypnotic conditions.

In addition, when the hypnotic dismorphism was active, the systolic vasoconstriction observed in prehypnotic basal condition (+4% at the first minute and +14.8% at the end of the test) was nullified at the first minute (when 5% vasodilatation rather than vasoconstriction was even observed) and strongly reduced at the end of the test, thus showing a reduced pain-related stress. These results indicate that body dismorphism is able to reduce *per se* not only subjective pain perception but also its adrenergic consequences. This is exactly what happens during real analgesia produced through chemical local anesthesia.

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