GIANCARLO CARLI'S CONTRIBUTIONS TO THE SCIENTIFIC UNDERSTANDING OF HYPNOTIC ANALGESIA

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ABSTRACT

This article summarizes one of a number of talks presented at a conference entitled 'Pain, Hypnosis and Sport Physiology: A Tribute to Giancarlo Carli', which was held in honour of Dr Carli upon his retirement as a Professor and Chair of the Department of Physiology, University of Siena. Professor Giancarlo Carli is a true Renaissance Man who has developed a translational research programme that spans multiple fields (e.g. hypnosis, pain, exercise physiology), different species (e.g. humans, cats, rats, rabbits), and different foci of study (e.g. behaviour, beliefs, neuroendocrine systems, neuroelectrical responses, brain chemistry). This article reviews his important contributions to just one of these many areas: hypnotic analgesia. His research in this area has shown that: (1) hypnotic analgesia has multiple underlying mechanisms that can impact all components of the pain matrix; (2) individuals with low hypnotic ability can, and do, benefit from hypnotic analgesia treatment; although, (3) individuals with low versus high hypnotic ability display important differences in the neurophysiological and psychological mechanisms underlying hypnotic responding. These critical discoveries have contributed to the scientific understanding of hypnosis and hypnotic analgesia, and also have important practical clinical implications for using hypnosis to treat patients with pain conditions.

Key words: hypnosis, hypnotic analgesia, mechanisms, hypnotic ability, Giancarlo Carli

INTRODUCTION

Professor Giancarlo Carli is a true Renaissance Man. During his productive career, he has developed a translational research programme that spans multiple topic areas (including hypnosis, pain, and exercise physiology), different species (including humans, cats, rats, and rabbits), and different foci of study (including behaviour, beliefs, neuroendocrine system, neuroelectrical responses, and brain chemistry). He is also a gracious host, wonderful mentor, and facilitator of national and international educational events that have contributed significantly to the professional development of other scientists and scientific knowledge.

Just one of the numerous areas he has devoted time to understanding is hypnotic analgesia. His contributions to this field have been many, but three key discoveries and conclusions that he and his research colleagues have made have had perhaps the greatest impact on the field: (1) hypnotic analgesia has multiple underlying mechanisms that can impact all components of the pain matrix; (2) individuals with low hypnotic ability can, and do, benefit from hypnotic analgesia treatment; although, (3) individuals with low versus high hypnotic ability display important differences in the mechanisms that underlie their response to hypnotic treatments.

HYPNOTIC ANALGESIA HAS MULTIPLE UNDERLYING MECHANISMS

In 1998 Professor Carli and colleagues published an important paper examining the effects of hypnotic analgesia on the nociceptive flexion (RIII) reflex in humans, a polysynaptic spinal reflex modulated by supraspinal descending pathways (Danziger et al., 1998). Previous researchers had demonstrated that response to hypnotic analgesia was associated with a reduction in both the somatosensory evoked cortical potentials and in the RIII component of the withdrawal reflex, suggesting that inhibition of nociceptive signals at the spinal level contributed to the efficacy of hypnotic analgesia (Kiernan et al., 1995). Danziger et al. (1998) administered a painful electrical stimulation on the left leg (left sural nerve) to individuals who scored high on the Stanford Hypnotic Susceptibility Scale (SHSS) under two conditions: (1) a control condition and (2) while being given hypnotic suggestions for analgesia. Throughout the procedures, the participants' pain threshold and physiological responses were assessed. All of the highly hypnotizable subjects demonstrated significant increases in pain threshold and a decrease in the late somatosensory evoked cerebral potential in the hypnosis condition, relative to the control condition. No differences in the autonomic parameters were observed.

What was striking about this study, however, was that all of the participants showed large changes in the amplitude of their RIII reflex, and two clear patterns of changes in this reflex emerged. In 11 of the 18 participants, and similar to Kiernan and colleagues (1995), a strong inhibition of the RIII reflex occurred. However, in the other 7 participants, there was a strong facilitation of the reflex. The findings provided important early evidence that not all highly hypnotizable individuals respond to hypnotic suggestions for analgesia in the same way and that there is not a single physiological mechanism of hypnotic analgesia.

Recent findings have provided a possible interpretation for the unexpected increases in RIII concomitant with decreases in the cortically evoked response to nociceptive stimulation. Specifically, it has been shown that distraction by neutral pictures increases RIII, while increased arousal potentiates the effects of pictures inducing negative emotions on pain and the RIII reflex (Roy et al., 2011). In conclusion, attention and emotion appear to modulate pain through partially dissociable neurophysiological mechanisms (Roy et al., 2011). This may account for the two different RIII modulation patterns seen in the highly hypnotizable subjects described by Danziger et al. (1998); that is, a cognitive style based on distraction (instead of attention to analgesia suggestions) may have induced the increase in RIII observed in a subgroup of highs.

Recently, Giancarlo Carli published an article summarizing two hypotheses that help explain the neurophysiology of pain, and discussed the implications of these hypotheses for understanding hypnotic analgesia (Carli, 2009). The first of these, put forth by Craig (2002, 2003), hypothesizes pain as a *homeostatic emotion* rather than a simple sensation. As a homeostatic emotion, pain sensations are not only processed in the primary and secondary sensory cortices, but are also processed in multiple areas, including the brainstem (e.g. parabrachial nucleus, which is a primary integration site for all homeostatic afferent activity), diencephalon (e.g. hypothalamus, which organizes goal-directed autonomic, neuroendocrine, and behavioural activity), and telencephalon (e.g. anterior cingular, insular, and prefrontal cortices, which generate representations of self and the meaning of one's experience). The second hypothesis, put forth by Zimmermann (1979) and Baron and Jänig (in press), argues that some chronic pain conditions involve positive (but maladaptive) feedback loops between efferent and afferent neurons, enhancing their activity, leading to sensory-affective, autonomic, motor, and endocrine abnormalities, and associated chronic pain.

Professor Carli's important contribution was helping us to comprehend the implications of these neurophysiological models for understanding the effects of hypnotic analgesia on the experience of pain. Specifically, he noted that hypnotic suggestions for analgesia can influence pain through *multiple* mechanisms; via their effects on the initial generation of pain signals (nociception), secondary neuron sensitization, and endocrine/immune responses through the modulation of sympathetic activity. This understanding provides researchers in the field with a wealth of potential targets to examine in the search for neurophysiological correlates of hypnotic analgesia.

The direct clinical implication of Professor Carli's work in this area is that clinicians should not only target the sensory cortices with their hypnotic suggestions (i.e. they should avoid *only* making suggestions for pain reduction) but rather provide multiple suggestions that impact multiple systems in the pain matrix. Suggestions can, and often should, include those that target the gating mechanisms in the spinal cord ('… you can filter out any uncomfortable sensations, allowing any and all comfortable and relaxation sensations to grow and expand'), the insula (perception of homeostatic state: '… and you feel so comfortable and well, the body is whole'), the anterior cingulate cortex (motivational state: '… so there is not really anything you need to *do* about the pain, you know you are healthy'), and the prefrontal cortex (meaning of the pain: '… as you think about your life and what is most important, pain seems to float into the background, because you know that it is possible to live a life consistent with your most deeply held values no matter what else is happening').

INDIVIDUALS WITH LOW HYPNOTIC ABILITY CAN BENEFIT FROM HYPNOTIC TREATMENT

Laboratory research on hypnotic analgesia conducted in the 1960s and 1970s demonstrated a moderate to strong association between the trait of global hypnotizability and response to hypnotic analgesia suggestions (Hilgard & Hilgard, 1975). Perhaps due to this early—and generally consistent—finding, many clinicians believe that only those individuals who have high hypnotic abilities can benefit from hypnosis treatments for pain. However, experimental pain induced in a laboratory setting is not the same as clinical pain experienced by patients in their daily life; the findings from laboratory studies do not necessarily generalize to real clinical settings. Importantly, if these findings do not generalize to clinical populations—that is, if individuals with clinical pain problems who have low hypnotic ability might benefit from hypnotic treatments—and if clinicians screen patients out of hypnotic treatment based on their responses to hypnotizability tests, patients who could benefit from treatment would not be treated. There is therefore a need to test the hypothesized associations between global hypnotizability and response to hypnotic analgesia in clinical populations. Professor Carli and colleagues have examined this important question.

In one seminal study, Carli and colleagues examined the roles that hypnotizability, hypnotic relaxation, and suggestions for analgesia played in the modulation of pain perception in samples of individuals with fibromyalgia (Carli, Biasi et al., 2008). A sample of women with fibromyalgia reporting a pain intensity of at least 50 on a 0–100 visual analogue scale (VAS) were screened for hypnotizability using the Stanford Hypnotic Susceptibility Scale (Form C), and classified as having high (Highs, average SHSS score = 9.57/12) or low (Lows, average SHSS score = 1.22/12) hypnotizability. A sample of healthy controls who were also scored as Lows (average SHSS score = 1.32/12) also participated in the study. Pain intensity was

assessed in the fibromyalgic Highs and Lows at baseline and again following hypnotic suggestions for relaxation (neutral hypnosis) and twice again following hypnotic suggestions for analgesia. The healthy controls (who were Lows) were given the same hypnotic procedures as the patients, although instead of asking them to rate their clinical pain, they were asked to rate the intensity of pain associated with a deep pressure algometer (applied to the foot) that had previously elicited a rating of moderate pain (50/100 on a VAS).

The primary finding from this study was that *both* of the clinical groups reported reductions in clinical pain following the hypnotic induction and hypnotic analgesia suggestions, whereas the healthy controls who scored low on the SHSS reported no reductions in experimental pain with hypnosis. Interestingly, however, the pattern of pain reductions did differ between the clinical Highs and Lows (see next section on mechanisms of hypnotic responding), with the Lows showing a progressive decrease in pain over the course of the entire session (and a similar response to relaxation suggestions as Highs), while the Highs showed a greater response (than Lows) following suggestions for analgesia.

Although Carli and colleagues' findings regarding the ability of individuals with low hypnotizability to respond to hypnosis is inconsistent with laboratory hypnosis research, it has been subsequently replicated in other clinical populations (e.g. Butler et al., 2009; Jensen et al., 2009). Thus, even though this might be considered a 'negative' finding, it actually raises some very interesting questions that could help us to better understand and explain response to hypnotic treatment. For example, as discussed by Carli and colleagues in a review article on this topic (Carli, Huber et al., 2008), it is possible that having a history of or experiencing chronic pain may facilitate a 'state' or states that might increase response to hypnosis. It is also possible that, even if Lows with clinical pain problems can respond to hypnosis—for chronic pain does not differ significantly or substantially between Highs and Lows—the specific strategies used by Highs and Lows to modulate pain in response to hypnotic suggestions differs. For example, Carli and colleagues have suggested that Lows may make more use of relaxation coping strategies than Highs in response to hypnotic suggestions, and the importance of outcome expectancies (i.e. placebo responding) may play a larger role in Lows' responses to hypnotic analgesia (Carli, Huber, et al., 2008; see also the next section, below). The clinical implications of Professor Carli's work in this area are clear, however: patients with chronic pain who have low scores on hypnotizability can still benefit from hypnotic treatment, and should not be screened out of such treatment on the basis of such scores alone.

INDIVIDUALS WITH HIGH VERSUS LOW HYPNOTIZABILITY DIFFER IN MECHANISMS OF RESPONSE TO HYPNOSIS

Although the differences between Highs and Lows in global response to hypnotic suggestions appear to be less pronounced (and in some cases, are virtually non-existent) in persons with clinical pain, relative to persons experiencing experimental pain, it remains possible that Highs and Lows differ in their physiological and psychological responses to hypnosis; that is, they may differ in the underlying *mechanisms* of hypnotic responding. Professor Carli and his colleagues have also made important contributions to understanding differences between Highs and Lows in the way that they respond to hypnotic suggestions.

In one study, for example, two groups of individuals with high and low hypnotizability were asked to stand upright with their eyes closed under three different conditions (presented in

a random order): (1) visual imagery (imagining a scene using visual suggestions); (2) tactile imagery (imagining a scene using tactile modalities); and (3) mental computation (serial subtractions and multiplications) (Carli et al., 2007). Measures included posture, movement/sway, imagery vividness, and effort required by the imagery or mental computation. The Lows rated visual imagery as 'easier' than tactile imagery, while the Highs reported that both were easy. Highs also judged the tactile imagery as less effortful and more vivid than Lows. Moreover, the Highs' body sway was not influenced by the cognitive tasks, whereas the Lows showed task-related changes in body sway. Professor Carli and colleagues concluded that greater attentional availability may be the basis of the absence of cognitive load on postural control in Highs.

In a follow-up study, Professor Carli and colleagues assessed sway, primary sensory modalities used to imagine the *absence of perception*, vividness of imagery, and effort required by imagery or mental contribution in a group of Highs and a group of Lows while standing with eyes closed under two conditions: (1) suggestions of 'no perception' (e.g. '… You don't see and hear anything … as if your body did not belong to you any more') and (2) mental computation (serial subtraction and multiplication) (Carli, Manzoni et al., 2008). The results indicated that Highs and Lows differed in their preferred imagery modalities; specifically, 100% of the Lows reported that they preferred a visual modality, whereas both visual and tactile modalities were reported by Highs. Interestingly, no other sensory channel (auditory, olfactory/gustatory) was used as the primary modality during the imagery by any participant. Also, Lows, but not Highs, demonstrated differences in sway during suggestions for decreased sensory availability.

The authors concluded that Highs are able to obtain satisfying mental imagery through both visual and tactile sensory modalities, whereas Lows clearly prefer the visual modality. The difference in the preferred modality of imagery may have been responsible for the difference observed between Highs and Lows in the postural response to vestibular stimulation (Santarcangelo et al., 2010). The earlier component of the vestibular reflex (VR) evoked by electrical stimulation of the labyrinth is not affected by voluntary control; its amplitude depends on the stimulus intensity, and the plane of body sway depends on the position of the head with respect to the trunk. It was found that the plane of body sway during imagery of having the head rotated was the same as it was during real head rotation in Highs, but not in Lows reporting the same vividness of imagery. Interestingly, in order to obtain an experience of 'head rotated', almost all of the Highs had chosen the somaesthetic modality of imagery, while all the Lows had chosen the visual one.

This study also confirmed the hypothesis supported in previous studies (Carli et al., 2006) that Highs can translate sensory imagery into real perception, as the earlier component of VR is not affected by voluntariness; this suggests that the response of Highs to suggestions is truly involuntary. However, the greater ability shown by Highs with respect to Lows during attention-demanding conditions like upright stance was not present in sitting subjects (Cavallaro et al., 2010). In these subjects, Professor Carli and colleagues have also reported on differences in Highs and Lows in brain activity measures in response to guided visual and somaesthetic imagery tasks. They asked groups of Highs and Lows to experience guided visual and somaesthetic imagery (counterbalanced) on two occasions while semi-reclined. Electroencephalography (EEG) activity during each imagery condition was assessed from 19 electrode sites for all study participants. In this study, there was no difference in preferred imagery modality (visual vs. somaesthetic), vividness, or effort between the Highs and Lows, although both groups reported that visual imagery was more vivid and less effortful than so-

maesthetic imagery. Importantly, EEG activity in response to the imagery condition did differ between groups. Overall, the investigators found a greater distribution of EEG modulation in the Highs (indicating what they called a more 'holistic' brain function), relative to the Lows.

Overall, Carli's research confirms that the cognitive trait of hypnotizability is highly pervasive and that Highs and Lows differ on some (but not all) important physiological responses to cognitive and physical stimulation. A recent study by Professor Carli and colleagues demonstrated another way that individuals with high hypnotizability differ from those with low hypnotizability. This study compared Highs and Lows with respect to *endothelial dysfunction*, which is a reduction in the ability of the blood vessels to respond appropriately to changes in blood flow. Endothelial dysfunction is also associated with increased cardiac risk (Jambrik, Venneri et al., 2004). Previous research had shown that although mental stress induces endothelial dysfunction, stress-related endothelial dysfunction is lower in Highs than in Lows both at baseline (i.e. not following a hypnotic induction) and after a hypnotic induction (e.g. Jambrik, Santarcangelo et al., 2004; Jambrik, Sebastiani et al., 2005). However, it was not known if painful stimulation also induced endothelial dysfunction and, if so, if either high hypnotizability and/or suggestions for analgesia buffered any effects of painful stimulation on endothelial dysfunction. In this study, a group of not hypnotized Highs and a group of Lows underwent a cold pressor nociceptive stimulation procedure with and without suggestions for analgesia (Jambrik, Santarcangelo et al., 2005). As hypothesized, the investigators found that both Highs and Lows evidenced endothelial dysfunction in response to the painful (pressor) stimulation, but it was much less pronounced in the Highs. The authors concluded that hypnotizability may provide a natural beneficial protection against the vascular effects of acute pain.

In contrast with these clear results on endothelial function, findings regarding possible differences between Highs and Lows in heart rate and heart rate variability (HRV) during the same nociceptive stimulation were not so clear-cut (Santarcangelo et al., 2008). In this second study, a group of Highs and a group of Lows were assessed during five experimental conditions: (1) simple relaxation, (2) painful stimulation (pressure applied at the second costochondral junction via a deep pressure algometer), (3) resting baseline, (4) painful stimulation with analgesia suggestions, and (5) resting baseline. Assessments included pain intensity ratings, respiration rates, heart rate, and HRV. Although Highs reported significantly greater pain reduction than Lows in the hypnotic analgesia condition, relative to the painful condition without hypnosis, the investigators found no group differences in respiration rate, heart rate, or HRV across the different conditions. The authors concluded that HRV may be less responsive to hypnotizability than endothelial function.

Overall, the results of this programme of research suggest that individuals with high versus low hypnotizability differ in some important ways (e.g. with respect to how they process sensory imagery as well as extent of their vascular reaction to pain and stress), but may be similar in others (e.g. cardiac response to pain). Thus, even when hypnotizability does not influence (or has a minimal influence) treatment outcome for clinical pain, it may influence *how* individuals respond to and use hypnosis for symptom management. The findings support the need to study these potential differences in greater detail. For example, hypnotic protocols for pain management often include suggestions incorporating visual 'safe place' imagery for dissociation and deepening the hypnotic experience, coupled with suggestions for changes in sensory experiences, such as morphing uncomfortable (painful) sensations into more neutral and tolerable sensations (e.g. Jensen, 2011). The findings from Professor Carli's research programme

suggest that an interesting avenue of further research would be to examine the extent to which suggestions for experiencing each sensory modality enhances analgesic effects, and if their relative efficacy differs as a function of hypnotizability.

Also, given the finding that different individuals (even those with high hypnotizability) likely use different cognitive (and associated neurophysiological) responses to hypnosis and hypnotic suggestions, a clear clinical implication of Professor Carli's research in this area is that hypnotic treatment must be tailored to each patient's capabilities and responses; one size does not fit all. Although it might be reasonable to begin treatment using hypnotic procedures and suggestions with proven efficacy on average, treatment outcome will likely be maximized if the clinician pays close attention to the patient's response to each hypnotic suggestion. Future inductions and suggestions should be modified based on the patient's response during previous sessions (Jensen, 2011).

THANK YOU, PROFESSOR GIANCARLO CARLI

Many people have much to thank Professor Giancarlo Carli for. Scientists should thank him for showing us how to be a modern Renaissance Man—for modelling how to study and understand complex issues from multiple perspectives. Scientists and researchers should thank him for his numerous contributions to our scientific understanding of many fields he has studied, including pain and hypnotic analgesia. Patients with clinical pain problems should thank him for his contributions which have specific important clinical implications that will increase response to treatment. Last, but not least, we all should thank him for being an inspiration and model of generosity, graciousness, and international collaboration. Thank you for your past, current, and future contributions!

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