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Focused analgesia and generalized relaxation produce differential hypnotic analgesia in response to ascending stimulus intensity

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Abstract

This study was designed in order to examine the effects of different types of hypnotic suggestion on hypnotic analgesia. Generalized relaxation and focused analgesia were induced in seven high-hypnotizable (HH) and eight low-hypnotizable (LH) subjects. Subjects were not aware to which group they belonged. The two groups did not differ in their expectation rates to achieve analgesia under hypnosis. Pain intensity and unpleasantness were rated on visual analogue scales in response to painful electrical stimuli, delivered in random order in five ascending intensities. Both focused analgesia and generalized relaxation decreased pain intensity significantly ($P < 0.01$). However, stimulus-intensity response curves differed under the two hypnotic conditions. As stimulus intensity became higher pain reduction was enhanced under focused analgesia, while a constant reduction occurred under generalized relaxation. The interaction between hypnotic state and stimulus intensity was significant for focused analgesia ($P < 0.05$) but not for generalized relaxation ($P > 0.07$), difference became more pronounced when analyzed for HH subjects only ($P < 0.002$ for analgesia, $P > 0.10$ for relaxation). Pain reduction was significantly higher in HH than in LH subjects under focused analgesia ($P < 0.02$) but not under generalized relaxation ($P > 0.5$). We conclude that by utilizing two modes of hypnotic suggestions in response to ascending stimuli, we were able to discover two components of hypnotic analgesia. One shows a parallel shift in the stimulus–response function, has features similar to placebo and bears no clear relationship to hypnotic susceptibility. The other shows a slope change in the stimulus–response curve and has a positive relationship to hypnotic susceptibility.

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1. Introduction

There are different types of hypnotic suggestion for altering pain sensation under hypnosis with possible different outcomes on the efficacy of

hypnotic analgesia (Price, 1999). For example, sensory and affective dimensions of pain can be selectively modulated by hypnotic suggestions aimed specifically at each pain modality (Rainville et al., 1999). Specific hypnotic instructions for anesthesia produced profound analgesia in highly hypnotizable subjects, but the absence of such

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instructions did not produce any analgesia, the latter condition not different from normal wakefulness (Knox et al., 1974). Highly hypnotizable subjects reduced intensity of pain to electrical stimuli under suggestions for hypnotic analgesia focused at their foot, but not under hypnotic suggestions to experience a pleasant feeling of relaxation (Zachariae et al., 1998). Similarly, Danziger et al. (1998) showed that no change occurred in pain thresholds during hypnotic relaxation in high-hypnotizable (HH) subjects, but that these thresholds increased significantly during hypnotic suggestion of analgesia. Somatosensory evoked responses demonstrated a similar distinction between relaxation and analgesia in these subjects. In contrast, the nociceptive R-III reflex was slightly, but significantly affected during relaxation (Danziger et al., 1998). On the other hand, Kiernan et al. (1995) did not find any significant difference in pain sensation between their 'affective analgesia' that suggested being comfortably relaxed, and 'sensory analgesia' that utilized dissociation imagery and focused analgesia suggestions. Focused analgesia, dissociated imagery or deep relaxation associated with instructions not to feel any pain, were all found to be effective in producing analgesia (De Pascalis et al., 1999). However, in highly hypnotizable participants, focused analgesia was more effective than other methods of suggestion.

The effect of placebo was studied in relation to hypnotic susceptibility. McGlashan et al. (1969) demonstrated that subjects with high or low-hypnotic susceptibility did not differ in their placebo response to a 'powerful analgesic drug'. It is interesting that Knox et al. (1974) did not demonstrate any analgesia by hypnosis alone, meaning that hypnotic induction with no specific suggestions for analgesia did not produce placebo analgesia. Price (1999) suggested that an experiential distinction ('outside authoritative' as opposed to 'innate self-directed') between placebo and hypnotic analgesia might at least partially account for the complete lack of relationship between the two. Hypnotic analgesia is not reversed by naloxone and is, therefore, not dependant on endogenous opioid pain-inhibitory mechanisms (Goldstein and Hilgard, 1975; Barber and Mayer, 1977). Placebo

analgesia on the other hand can be reversed by naloxone (Benedetti et al., 1999), although there is evidence that certain types of placebo analgesia are not naloxone reversible (Gracely et al., 1983; Amanzio and Benedetti, 1999).

A subject's hypnotic susceptibility can predict hypnotic analgesia (Hilgard and Hilgard, 1983), while placebo analgesia, that depends on many factors such as conditioning, expectation and desire for pain relief, is less predictable and is more dependant on outside stimuli (Price, 1999, 2000). Recently, however, De Pascalis et al. (2002) demonstrated that placebo analgesia response could be predicted from a 'sensory suggestibility scale' (Gheorghiu et al., 1995). There are several studies that show that expectation is one of the main mediators of placebo analgesia (Montgomery and Kirsch, 1996; Amanzio and Benedetti, 1999; Price et al., 1999; Pollo et al., 2001), but a role for expectation in producing hypnotic analgesia is not clear. It is possible, for example, that hypnotic suggestions that produce higher hypnotic analgesia may also produce higher expectations.

Sharav and Tal (1989) found that placebo and hypnosis produced different stimulus intensity response curves in HH subjects. Thus, in response to ascending levels of electrical stimuli, pain reduction under placebo was constant (parallel shift), but enlarged under hypnotic analgesia (Sharav and Tal, 1989). This increased pain reduction to ascending stimuli under hypnotic analgesia was previously demonstrated by Price and Barber (1987). A parallel reduction in pain intensity, on a time vs. pain intensity curve, was also demonstrated in response to open and hidden infusions of an analgesic drug, a reduction attributed to a placebo effect (Amanzio et al., 2001). This parallel shift was suggested as typical for a placebo-produced analgesia (Price, 2001). Amanzio et al. (2001) also found that a placebo effect adds to the response variability of analgesia.

In summary, different types of instructions for hypnotic analgesia may produce different outcomes. Hypnotic susceptibility is not related to placebo analgesia, and high- and low-hypnotizable (LH) subjects respond equally to placebo analgesia. Hypnotic and placebo analgesia produce different stimulus-response curves to ascending

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