

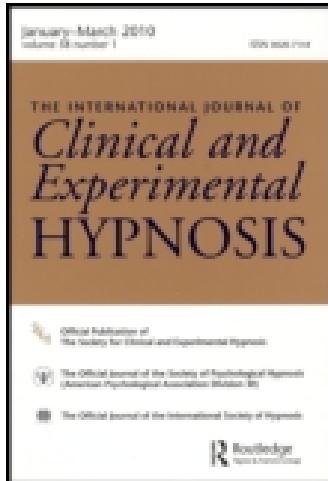
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CARDIOVASCULAR REACTIVITY DURING HYPNOSIS AND HYPNOTIC SUSCEPTIBILITY: *Three Studies of Heart Rate Variability*

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Abstract: This paper was designed to examine the relationship between hypnotic susceptibility and cardiovascular measures, especially parasympathetic activity, in 3 separate studies. In these studies, neither heart rate nor heart rate variability differed between the high and low hypnotically susceptible individuals at the initial baseline. Furthermore, in the first study, experimental tasks designed to elicit differential sympathetic and parasympathetic cardiac responses demonstrated no interaction with hypnotic susceptibility. Overall, these 3 studies suggest that hypnotic susceptibility in itself is not associated with parasympathetic aspects of either basal cardiac states or cardiac responses. In addition, a hypnotic induction itself did not differentially influence parasympathetic activity for the high versus low susceptible individuals.

Nonphysiological studies of hypnotic susceptibility have shown remarkable reliability (see Fromm & Nash, 1992, for an overview of hypnotic susceptibility research). Various measures of hypnotic susceptibility have been shown to be correlated above .60 (Bowers, 1983). Even more impressive is the finding that hypnotic susceptibility has been shown to be as stable a measure of individual differences as measures of IQ or various personality inventories, with a test-retest correlation of at least .71 over 10-, 15-, and 25-year periods (Piccione, Hilgard, & Zimbardo, 1989). Given this stability, it is surprising that few individual

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difference variables exist that can reliably predict hypnotizability (Kirsch & Council, 1992; Silva & Kirsch, 1992). Even conceptually similar measures such as dissociative experiences show an orthogonal relationship with hypnotic susceptibility (Faith & Ray, 1994; Ray, 1995; Ray & Faith, 1995). One possibility may lie in psychophysiological measures, which have been shown to relate to psychological constructs in other contexts (e.g., Tomarken, 1995). Indeed, previous work from our lab and others show central nervous system psychophysiological differences, especially in terms of the relationship of electroencephalograph (EEG) theta activity and hypnotic susceptibility (Graffin, Ray, & Lundy, 1995; cf. Sabourin, Cutcomb, Crawford, & Pribram, 1990). With regard to activity of the autonomic nervous system, some speculation connects hypnotic processes with heart rate variability, although there is limited empirical research.

Heart rate variability increasingly is employed to aid in understanding the relationship between psychological and physiological processes (see Berntson et al., 1997, for an extensive review of this measure). There are two basic approaches to quantifying heart rate variability: (a) global descriptive statistics of heart periods and (b) modeling of periodic cardiac patterns to extract specific frequency components. The global statistics approach examines the differences in time between successive heart beats (R-R intervals) and uses a measure such as mean successive differences (MSD), or standard deviation of these differences, to describe heart rate variability. The second approach uses Fourier (FFT) analysis or autoregressive (AR) techniques to create a frequency spectrum. Those using FFT or AR procedures view the high frequency component (.12-.4 Hz) as reflecting parasympathetic activation and thus refer to it as vagal tone, whereas the low frequency component (.06-.1 Hz) is proposed to involve both sympathetic and parasympathetic activation. The heart is controlled by both vagal and sympathetic efferents, which may act independently, reciprocally, or non-reciprocally (Berntson, Cacioppo, & Quigley, 1993). Because of this, it is important to have some understanding of the independent contributions of sympathetic and parasympathetic control of the heart. Our understanding of high frequency heart rate variability and the autonomic contributions to it has resulted largely from studies utilizing pharmacological blockades. For example, following administration of atropine, a parasympathetic blockade, high frequency heart period variability was virtually eliminated in human participants. Whereas, when a sympathetic blocker, metoprolol, was administered, high frequency heart rate variability remained unaltered (Berntson et al., 1994). Furthermore, it has also been shown that atropine reduces overall heart rate variability, suggesting that much of the power in overall variability is due to parasympathetic influences (Akselrod et al., 1985). Thus, it is possible to use heart rate variability to estimate parasympathetic activity in humans.

Recently, three studies have examined the role of heart rate variability changes in relation to hypnotic susceptibility and hypnosis. In the first study, hypnotic susceptibility scores across the entire range were correlated with baseline heart rate and heart rate variability (Harris, Porges, & Vincenz, 1993). These authors reported that hypnotic susceptibility scores on the Harvard Group Scale of Hypnotic Susceptibility, Form A (HGSHS:A; Shor & Orne, 1962) were correlated with both heart rate ($r = -.47$) and the high-frequency component of heart rate variability ($r = .45$) at baseline, suggesting that low heart rate and high vagal tone were associated with greater hypnotic susceptibility. A second study examined high and low hypnotically susceptible individuals during a baseline and a standard hypnotic induction procedure (DeBenedittis, Cigada, Bianchi, & Signorini, 1994). In general, there was a decrease of heart rate following the hypnotic induction, especially for the high susceptible group. However, there were neither high frequency nor low frequency heart rate variability changes between the baseline condition and the hypnotic induction for either hypnotically susceptible group. Furthermore, this study did not replicate the baseline differences reported in the Harris et al. (1993) study. Finally, Hautkappe and Bongartz (1992) reported no difference in low frequency heart rate variability in high and low hypnotically susceptible individuals at an initial baseline condition before a hypnotic induction. In summary, although low frequency heart rate variability is not associated with hypnotic susceptibility, the question of high frequency heart rate variability and its relation to hypnotic susceptibility remains an open question.

To help clarify the relationship between hypnotic susceptibility and parasympathetic activity as reflected by heart rate variability, we examined cardiovascular changes in three larger studies related to the psychophysiology of hypnotic susceptibility and hypnosis. In terms of the cardiovascular aspects of the three studies, the goals follow.

Study 1. The three goals of this study were: (a) to examine initial baseline cardiac differences between high and low hypnotically susceptible individuals; (b) to demonstrate differential cardiac effects using tasks designed to evoke sympathetic or parasympathetic reactivity; and (c) to determine whether high and low hypnotically susceptible individuals show differential cardiac responses to these tasks.

Study 2. The goals of this study were: (a) to examine initial baseline cardiac differences between high and low hypnotically susceptible individuals and (b) to examine cardiovascular changes following a standard and nonstandard hypnotic induction.

Study 3. The goals of this study were: (a) to examine initial baseline cardiac differences between high and low hypnotically susceptible individuals; (b) to examine cardiovascular changes at two time points

following a standard hypnotic induction; and (c) to replicate the first two studies with a European rather than American population.

METHODS

Study 1

Participants were chosen from an introductory psychology course offered at Pennsylvania State University. Following an initial group screening procedure, which included administration of the HGSHS:A (Shor & Orne, 1962) to approximately 400 undergraduates, 8 high susceptible and 6 low susceptible individuals were chosen to participate in the psychophysiological component of the study, based on their hypnotic susceptibility score (3 or less for low hypnotic susceptibility and 10 or greater for high susceptibility).

Heart rate was recorded using silver/silver chloride electrodes attached over the right and left rib cage. A strain gauge respirometer was placed around the participant over the thorax below the recording electrodes to measure respiration. Heart rate and respiration were analyzed by computer with software developed for this lab. Heart rate variability was estimated both by mean successive differences (the average of the absolute values of successive differences in R-R intervals in milliseconds) and by an FFT analysis (the power spectrum) of the high frequency component (.12-.4 Hz) of the heart period time series after a polynomial filter had been used to remove slow trends in the data (see Porges & Bohrer, 1990). These two measures have been found to correlate highly in previous studies (Fox, 1983).

Participants were tested individually. They were brought into the lab and given a written explanation of the study and an informed consent form. Participants were then brought into the laboratory room and seated adjacent to a computer/television screen. Electrocardiograph (ECG) electrodes and the respiration device were attached. Participants were instructed to remain seated and quiet. An initial 3-minute eyes-closed baseline ECG was taken. Following the baseline, the participants were administered four autonomically evocative tasks. Two tasks (mental arithmetic and the Stroop) were designed to elicit increases in sympathetic activity, whereas the other two tasks (a calming video of birds flying over the ocean and imagining a meal) were designed to elicit increases in parasympathetic activity. A 2-minute baseline was recorded between each task.

Following the completion of the experimental protocol, all participants were asked what they thought the primary hypothesis was. None of the participants correctly identified the link between the hypnotic and psychophysiological components of the experiment. A *p* level of .05 was used for all statistical computations. An ANOVA was used to determine significant group differences. Tasks were broken down into those seen as

increasing parasympathetic activation (Imagery and Video) and those seen as increasing sympathetic activation (Math and Stroop). We used these task descriptions as a short-hand simplification because any of these tasks may produce changes in both sympathetic and parasympathetic activation. Based on previous research (Ray & Cole, 1985), tasks were further differentiated on the basis of stimuli-presentation type: internal (Imagery and Math) and external (Stroop and Video). Cardiovascular reactivity scores were calculated and used for statistical analyses (i.e., change from baseline).

Baseline Differences

No heart rate group differences were found between the high and low hypnotically susceptible groups at baseline, $F(1, 12) = .04, p = .85$. Mean heart rate and standard deviations are as follows: high (78.7 ± 12.7) and low (77.2 ± 17.7). No MSD differences were found between groups at baseline, $F(1, 12) = .62, p = .45$. Mean MSD and standard deviations are as follows: high (36.82 ± 16.12) and low (49.7 ± 42.71). These differences were paralleled by FFT measures of heart period, $F(1, 11) = .01, p = .94$. Mean vagal tone and standard deviations are as follows: high ($6.81 \pm .91$) and low (6.74 ± 2.4). Thus, no baseline group differences were found for any of the cardiovascular measures.

A main effect for parasympathetic versus sympathetic tasks was found, $F(1, 12) = 8.67, p = .01$. This main effect results from the significant decrease in MSD from baseline during the administration of the sympathetically arousing tasks (Math and Stroop). The mean MSD change from baseline for the parasympathetic tasks was $-.334$ and for the sympathetic tasks was -17.02 . A significant main effect for internal versus external stimulus presentation was also found, $F(1, 12) = 10.42, p = .0073$. The mean MSD change from baseline for the internal tasks was significantly larger than for the external tasks, -13.28 and -4.07 , respectively. In terms of hypnotic susceptibility, no group differences were found between high and low individuals for MSD measures during the four tasks, $F(1, 12) = .01, p = .906$.

Study 2

Participants were undergraduate students recruited from a Pennsylvania State University introductory psychology course. Following an initial group screening procedure, which included administration of the HGSHS:A (Shor & Orne, 1962), 8 individuals who scored 10 or above (high susceptibility) and 10 individuals who scored 3 or below (low susceptibility) were selected to participate in this study. All individuals included in this study described themselves as right-handed.

All 18 individuals participated in each of the two experimental sessions. The two laboratory sessions followed the same procedural guidelines except for the induction procedure. Each individual participated in

one session utilizing the Stanford Hypnotic Susceptibility Scale, Form C (SHSS:C; Weitzenhoffer & Hilgard, 1962) induction and one session where an informal induction procedure was used. This induction was adapted from a brief self-induction instruction used in Bertrand, Stam, and Radtke (1993). These induction procedures were administered in a counterbalanced manner so that half of the subjects in each hypnotizability condition experienced the Stanford induction during the first session and the informal induction during the second, and the other half were exposed to the two induction procedures in the reverse order.

Prior to the start of the first session, the recording procedures were described clearly to the participants and any questions answered. Participants were then seated in a comfortable chair in a private room, and the electrodes were applied. Care was taken to establish rapport and help the individual to feel at ease during this part of the procedure. After the experimenter left the room, the individual was asked, via intercom, to close his or her eyes and just relax for a few moments while keeping movement to a minimum. Cardiovascular data used in this report include the initial baseline, a baseline immediately following the standard hypnotic induction, and two baselines at the conclusion of the session.

A 2 (high-low susceptibility group) \times 4 (baselines) ANOVA using MSD showed no significant differences between groups, $F(1, 16) = .75$; $p < .40$, or within groups by baseline interaction, $F(3, 48) = .81$; $p < .4963$. Heart rate did decrease across the session as would be expected. However, there were no differences between susceptibility groups, $F(1, 16) = 1.49$; $p < .24$, and no group by baseline interaction, $F(3, 48) = .05$; $p < .99$.

Study 3

Participants at the University of Rome were given the SHSS:C. Twelve high susceptible individuals (score 10 or above) and 10 low susceptible individuals (score 4 or below) were used in the study. Cardiovascular and electrocortical measures were recorded prior to and at two points during a hypnotic induction. The electrocortical measures have been reported elsewhere. An ANOVA examining group (high vs. low hypnotic susceptibility) by 3 periods (pre-induction, and two induction measures) demonstrated no MSD differences between groups and no significant interaction of group and hypnotic susceptibility, $F(2, 40) = .76$, $p = .64$, with Greenhouse-Geisser correction.

DISCUSSION

This paper was designed to examine the relationship between hypnotic susceptibility and cardiovascular measures, especially parasympathetic activity as estimated by MSD, in three separate studies. In Studies 2 and 3, we also examined the influence of a hypnotic induction. In three separate studies, we found that neither heart rate nor heart rate

variability differed between the high and low hypnotically susceptible individuals at the initial baseline. Likewise, the first study also showed that the FFT high frequency component of heart rate variability did not differentiate between groups. Also, in our first study, experimental tasks designed to elicit differential sympathetic and parasympathetic cardiac responses demonstrated no interaction with hypnotic susceptibility. That is, both groups responded similarly. As would be expected, the tasks that reciprocally activate the sympathetic branch and decrease activity in the parasympathetic branch produce a decrease in MSD and an increase in heart rate compared to baseline. This finding does not support empirical and theoretical speculation that relates parasympathetic activity with hypnotic susceptibility (cf. Harris, Porges, & Vincenz, 1993). Given the present results and the finding that electrocortical measures consistently have been associated with hypnotic susceptibility, it appears that hypnotic susceptibility is more likely related to central rather than autonomic nervous system factors. In our Studies 2 and 3, we examined heart rate and heart rate variability following hypnotic inductions. In both of these studies, high and low susceptible individuals did not differ in either heart rate or heart rate variability, either at baseline or after a hypnotic induction.

Overall, our three studies suggest that hypnotic susceptibility in itself is not associated with parasympathetic aspects of either basal cardiac states or cardiac responses. Furthermore, a hypnotic induction itself did not differentially influence parasympathetic activity for the high versus low susceptible individuals. Given that the SHSS:C was administered as part of Study 2 and that high susceptible individuals passed significantly more hypnotic challenges, we have a manipulation check that shows the hypnotic procedure worked. Thus, our lack of cardiac differentiation between high and low hypnotically susceptible individuals cannot be due to ineffectual hypnotic procedures. This, of course, is not to suggest that various aspects of a hypnotic induction (e.g., relaxation) would not result in increased parasympathetic responding but only that such aspects may similarly influence both high and low hypnotically susceptible individuals.

An intriguing area for future research involving hypnosis and cardiac activity is the ability of such psychophysiological measures to give insight into how high and low susceptible individuals perform hypnotic challenges. Because there is psychophysiological data to suggest that various cognitive and emotional tasks differently activate the cardiac system, these measures may be useful for describing the underlying mechanisms for specific hypnotic phenomena and their relation to individual differences. For example, various studies have shown that cardiovascular responses are not inhibited during hypnotic analgesia (e.g., Hilgard & Hilgard, 1975), in which individuals reduce the

perception of pain. However, during posthypnotic amnesia, Hautkappe and Bongartz (1992) were able to demonstrate more FFT low frequency heart rate variability in low hypnotically susceptible individuals, suggesting to these authors that the amnesia condition required more cognitive activity on the part of high susceptible individuals. Thus, although heart rate variability may not be directly related to hypnotic susceptibility or a hypnotic induction, it may offer a means of understanding how high and low susceptible individuals approach various hypnotic challenges, including pain perception or amnesia.

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Kardiovaskuläre Reaktivität bei Hypnose und hypnotische Suggestibilität: Drei Untersuchungen zur Variabilität der Herzfrequenz

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Karen Quigley, Deane Aikins, und Melissa Tubbs

Zusammenfassung: In dieser Studie wurde in drei getrennten Untersuchungen die Beziehung zwischen Hypnose-Suggestibilität und kardiovaskulären Maßen, insbesondere von parasympathischer Aktivität, erforscht. In diesen Untersuchungen gab es an der anfänglichen Baseline bei den hoch- bzw. niedrigsuggestiblen Vpn. in Bezug auf Herzfrequenz und Variabilität der Herzfrequenz keine Unterschiede. In der ersten Untersuchung waren außerdem experimentelle Aufgaben daraufhin angelegt, unterschiedliche sympathische und parasympathische Herzreaktionen hervorzurufen. Es zeigte sich dabei jedoch keine Interaktion mit Hypnose-Suggestibilität. Insgesamt deuten diese 3 Untersuchungen an, dass Hypnose-Suggestibilität als solche nicht mit den parasympathetischen Aspekten von Baseline-Herztätigkeit oder mit deren Variabilität in Zusammenhang steht. Und die Hypnose-Induktion als solche hat die parasympathetische Aktivität bei hoch- und niedrigsuggestiblen Vpn. nicht in unterschiedlicher Weise beeinflusst.

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Réactivité cardio-vasculaire pendant l'hypnose et sensibilité hypnotique : Trois études sur la variation du pouls cardiaque

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Résumé: Ce travail a pour objectif d'examiner, dans trois études séparées, la relation entre la sensibilité hypnotique et les constantes cardio-vasculaires, plus spécialement l'activité parasympathique. Dans ces études, ni le pouls cardiaque ni la variation du pouls n'ont différé entre les sujets hautement ou faiblement hypnotisables par rapport aux chiffres de base. En outre, dans la première étude, les exercices à effectuer qui étaient susceptibles d'augmenter la différence entre des réponses parasympathiques ou sympathiques ont montré qu'il n'y avait pas d'interaction avec la sensibilité hypnotique des sujets. Au total, ces trois études montrent que la sensibilité hypnotique en elle-même n'est pas associée aux états cardiaques de base ou aux réponses cardiaques. De plus, une induction hypnotique elle-même ne peut influencer et initier une différence dans l'activité parasympathique chez les patient hautement hypnotisables par rapport aux faiblement hypnotisables.

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La respuesta cardiovascular durante la hipnosis y la susceptibilidad hipnótica: Tres estudios de variabilidad de tasa cardiaca

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Karen Quigley, Deane Aikins, y Melissa Tubbs

Resumen: Diseñamos este trabajo para examinar tres estudios distintos sobre la susceptibilidad hipnótica y medidas cardiovasculares, especialmente la actividad parasympática. En estos estudios, ni la tasa cardiaca ni su variabilidad difieren en la línea base entre individuos con alta o baja susceptibilidad hipnótica. Asimismo, no se encontró una interacción con la susceptibilidad hipnótica en el primer estudio, que utilizó tareas experimentales diseñadas para obtener diferentes respuestas cardíacas simpáticas y parasympáticas. En conjunto, los tres estudios sugieren que la susceptibilidad hipnótica en sí no está asociada con aspectos parasympáticos de los estados cardíacos basales o las respuestas cardíacas. La inducción hipnótica no influyó en forma distinta la actividad parasympática de los individuos con alta o baja susceptibilidad hipnótica.

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