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THE INVOLVEMENT OF FRONTALLY MODULATED ATTENTION IN HYPNOSIS AND HYPNOTIC SUSCEPTIBILITY: CORTICAL EVOKED POTENTIAL EVIDENCE

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Abstract

The frontal N100 difference wave (N100d) between cortical evoked potentials to frequent and infrequent tones was compared before and during hypnosis in subjects with high and low hypnotic susceptibility (n = 10,11). This was to test putative alterations in attention, novelty detection in particular, modulated by anterior functions including the anterior cingulate to auditory stimuli extraneous to hypnosis. Susceptibility was measured with the HGSHS:A and validated with a laboratory scale during the experiment. Results showed that in the highly susceptible participants the N100d was clearly manifested in the pre-hypnosis control condition, was later attenuated following instructions of hypnosis and was virtually absent after further hypnosis. The effect was due to attenuated responding to the infrequent stimulus, without change to the frequent stimulus. This compromise extended to the later frontal P300 wave whose amplitude was reduced by 50% with hypnosis and was negligible by the end of hypnosis. The parietal P300 was reduced in both hypnosis conditions to a similar degree. These results were consistent with a progressive reduction throughout the hypnotic induction in focused attention to stimuli incidental to hypnosis capable of frontal modulation and frontal activation to the novel stimulus. In contrast the participants with low susceptibility showed an opposite pattern of change in the N100d. Though absent before hypnosis there was a progressive increase in the N100d from the first to the second hypnosis recording. This was underpinned by changes in response amplitudes to both frequent and infrequent stimuli, without systematic changes to the P300. Their results supported a dispersion of attentional resources and an absence of frontal activation in the pre-hypnosis baseline, compatible with a lack of focused attention and engagement of frontal functions theorized to be necessary for hypnosis. Furthermore subjects with low hypnotizability showed a longer P300 latency at the central parietal peak compatible with more prolonged processing of the rare stimulus, in keeping with a more posterior bias in processing. This may militate against the induction of hypnosis, theorized to require initial engagement of anterior processes, and here supported by the results in the highly hypnotizable participants.

Key words: hypnosis, hypnotic susceptibility, frontal lobe, attention, novelty detection, event-related potentials, N100, P300

Introduction

There is a range of evidence implicating the primary involvement of the frontal lobes in the hypnotic process as well as in the orchestration of top down influences on brain processing. The involvement of frontal functions in hypnosis has been the implication of both theoretical considerations and empirical evidence. Theory has included a neuropsychological translation of the hypnotic induction process, such as the suspension of reality testing, automaticity and handing over the planning of behaviour to the hypnotist (Gruzelier, 1990; 1998), and failing to foresee the emotional consequences of future actions all allowing for the permissive and otherwise incongruous behaviour of participants in stage hypnosis shows (Gruzelier, 2000a). Theory has also involved application of cognitive models of high order executive and attentional systems to the hypnotic process (Crawford and Gruzelier, 1992; Woody and Bowers, 1994; Woody and Sadler, 1998; Kaiser, Barker, Haenschel, Baldeweg and Gruzelier 1997; Gruzelier, 1998; Oakley, 1999a,b).

Empirical evidence has included measures of neuropsychological function with tests of ideational fluency, attention and executive functions which have disclosed compromise following hypnosis (Gruzelier and Warren, 1993; Woody and Farvolden, 1998; Kallio, Revonsuo, Hamalainen, Markela and Gruzelier 2001). Blood flow and metabolism studies have implicated *inter alia* the anterior cingulate in hypnotic analgesia (Crawford, Gur, Skolnick, Gur and Benson 1993; Rainville, Hofbauer, Paus, Duncan, Bushnell and Price 1999; Faymonville, Laureys, Degueldre, DelFiore, Luxen, Franck, Lamy and Maquet 2000), hallucinations (Szechtman, Woody, Bowers and Nahmias, 1998) and revivification of pleasant memories (Maquet, Faymonville, Degueldre, Delfiore, Franck, Luxen and Lamy 1999). Electrophysiological measures have included: electrodermal orienting responses modulated by fronto-limbic systems (Gruzelier and Brow, 1985); EEG coherence, which disclosed a reduction within the left anterior region following hypnosis (Kaiser in Gruzelier, 1998); and EEG oscillations to painful stimuli where relations between the amplitude of gamma oscillations recorded over anterior cortex and the intensity of discomfort were dissociated after hypnosis (Croft, Williams, Haenschel and Gruzelier, 2002). Cortical evoked potentials have also shown in hypnosis, in combination with errors in performance, the absence of the error-related positivity wave. This is a positive potential thought to signify corrective processing, which follows error-related negativity and the detection of errors in performance (Kaiser et al., 1997), and, what will be detailed here, a progressive reduction of the N100d wave over frontal cortex signifying a loss of sustained attention to stimuli incidental to hypnosis (Gruzelier, 1996). These last mentioned results have not been reported in full and form the subject of this report.

While on the one hand the focusing of attention is central to the induction of hypnosis (Crawford and Gruzelier, 1992), on the other attention is central to the role of the frontal lobes (Luria, 1973; Shallice, 1988). On the basis of electrophysiological evidence Luria (1973: 189) concluded that 'the frontal lobes participate in the regulation of the activation processes lying at the basis of voluntary attention'. This has been subsequently endorsed by investigation with blood flow and metabolic procedures (Tzourio, El Massioui, Crivello, Joliot, Renault and Mazoyer, 1997; Ebmeier, Steele, MacKenzie, O'Carroll, Kydd, Glabus, Blackwood, Rugg and Goodwin, 1995; Kiehl and Liddle, 2001). Theoretically Norman and Shallice (1986) have conceptualized attention as involving two mechanisms distinguished by controlled and automatic processing. The first is an executive supervisory attention system (SAS), which reflects conscious

awareness and sets priorities for action on the basis of internal knowledge states. This involves the frontal lobes. This may override the second, the contention scheduler, which responds automatically to environmental stimuli or to the priming of stored knowledge through conceptual thought. Contention scheduling involves subcortical processes. Damage to the frontal lobes leads to impulsive and distractible behaviour and over-responsiveness to automatic demands of the environment (Shallice, Burgess, Schon and Baxter, 1989).

Electrophysiological recording has been the approach most often utilized in elucidating the complexities of attention (Nataanen, 1992). The phenomenon perhaps most associated with voluntary attention is the large negative potential occurring at around 100 msecs, graphically demonstrated by the larger amplitude at vertex of the N100. Both exogenous stimulus characteristics and endogenous factors, such as state of arousal and attention, contribute to the amplitude of the N100, such that the N100 is the summation of multiple processes and generators (Nataanen and Picton, 1987). These include what is termed an 'attentional supervisor'. This is thought to be generated by the anterior frontal cortex. This is because of the anterior location of the Nd wave, which is obtained by subtracting the response to unattended stimuli from the response to attended stimuli (Hansen and Hillyard, 1980), and because of accompanying metabolic changes, which involve frontal circuitry and the absence of the Nd wave in patients with damage to the frontal lobes (Knight, Hillyard, Woods and Neville, 1981; Knight 1984).

With Positron Emission Tomography (PET) Tzourio et al. (1997) endorsed the importance of a frontal network capable of modulating a local temporal network. They proposed that the bilateral precentral elevations in blood flow underpinned the arousal properties of the N100 while activation of the anterior cingulate underpinned both target detection, after Posner and Petersen (1990), and the execution of action, as in a button press to identify the infrequent target (Posner, 1995). Gabriel (1990) had on the basis of animal experiments also concluded that the anterior cingulate was involved in the detection of novelty. Orienting responses to tones modulated by fronto-limbic mechanisms including the cingulate have been shown to be attenuated in the electrodermal system in hypnosis (Gruzelier and Brow, 1985).

Following the N100 there is a later positive wave to the infrequent stimulus at around 300 msecs termed the P300. This has been subclassified as the P3a, which has a frontal peak and the P3b with a parietal peak. Novelty has also been associated with the frontal P3a, while the P3b has been associated with stimulus significance and has a longer latency. Ebmeier, Steele, MacKenzie, O'Carroll, Kydd, Glabus, Blackwood, Rugg and Goodwin (1995) in a SPECT study of the two-stimulus oddball paradigm used here concluded that the anterior cingulate is implicated in the frontal generation of the P3a occurring to the target stimulus, for a positive correlation was found between the tracer uptake and the P3a amplitude; the P3b correlated with uptake in both frontal and temporal regions. Here we examined the effect of hypnosis on the anterior N100 across the frontal chain and on the centrally located P300 at frontal and parietal locations to infrequent and frequent tones. Binaural stimuli were used because they evoke larger responses than monaural stimuli (Celesia, 1976).

Method

Subjects

From an initial screening of medical student volunteers with the Harvard Group Scale of Hypnotic Susceptibility, Form A (Shor and Orne, 1962), 10 individuals with high

hypnotic susceptibility, having scores of nine or above, and 11 with low hypnotic susceptibility having scores of three or lower were selected. They included both male and female subjects, equally distributed between the groups, and with a mean age of 23.5 years. As done with all hypnosis experiments in the laboratory hypnotic susceptibility was examined with a laboratory scale during the experimental procedure to ensure that the initial allocation was valid (Gruzelier, 2000b). Subject allocation was not altered. The local Ethics Committee approved the research and subjects gave written informed consent.

Measures

The electroencephalogram was recorded from 28 scalp derivations, to include the 10–20 system, with surface tin electrodes and referenced to linked earlobe electrodes. Cortical evoked responses were recorded while the subject with eyes closed was required to press a button with the right index finger to infrequent higher pitched tones (1500 Hz). These were randomly embedded in a series of frequent lower pitched tones (1000 Hz) with an overall probability of 80%. The tones were presented through headphones binaurally for 50 msec, with a rise and fall time of 5 msec, and with an intensity of 80 dB SPL. The interstimulus interval was 1 sec. Signal bandpass was set to 0.3–40.0 Hz, the digital sampling epoch was 650 msec and the length of the sampling epoch was 650 msec including a 50 msec pre-stimulus baseline. Baseline to peak amplitudes and latencies were scored using the tangential method (Goodin, Squires, Henderson and Starr 1978) (1) for the Nd at the anterior electrode placements, Fz, F3, F4, Cz following the procedures of Knight et al. (1981); and (2) for the P300 at Fz and Pz. Recording took place in a sound attenuated, electrically shielded chamber.

Design

First a baseline event-related potential (ERP) recording was obtained followed by a live standard hypnotic relaxation induction for 20 minutes. The ERP recording was repeated and a final ERP recording was recorded after further hypnosis of approximately 30 minutes. The ERP sequences took two minutes each. An easy discrimination between frequent and infrequent tones was chosen so that the subjects could perform at ceiling, which they did. Items for the Experimental Susceptibility Scale were inserted throughout and immediately after the hypnotic induction.

Statistics

The groups were compared with repeated measures analysis of variance with Group (High/Low), Condition (Baseline, Hypnosis 1, Hypnosis 2) as factors, together with electrode (Cz, Fz, F3, F4) in the case of the N100, and electrode (Fz, Pz) in the case of the P300. Significant results were further elucidated with one way ANOVAs and *t* tests.

Results

N100 difference wave

The mean amplitudes of the N100 difference waves for the four anterior electrode placements are shown in Figure 1 (a), (b) for the high (a) and low (b) hypnotically susceptible subjects. Differential patterns of response across conditions were disclosed between the groups (Group x Condition: $F=5.76$, $p<0.03$). There was a progressive decline in difference wave amplitudes from baseline to the final stage of hypnosis in the highly susceptible subjects ($F=9.03$, $p<0.02$). The opposite effect was found in the subjects with

low levels of hypnotic susceptibility (F=4.93, $p<0.05$) where, unexpectedly, there was a virtual absence of the difference wave at baseline, a small but attenuated difference wave at the early stage of hypnosis, and only in the final condition was there a difference wave approaching the magnitude of the one seen in the high susceptibility subjects at baseline. A significant group x condition interaction was also found when comparing the two hypnosis conditions (F = 5.84, $p<0.03$) due to the reduction in the difference wave in highly susceptible subjects ($t = 3.42$, $p<0.005$) and the increase in the subjects low in susceptibility ($t = 4.78$, $p<0.002$). Furthermore, there was a highly significant difference between the groups at baseline with the advantage to the highly susceptible subjects ($t = 6.48$, $p<0.001$). The differential changes between the groups from baseline to hypnosis were significant for all anterior electrodes: Cz (F = 8.61, $p<0.01$); Fz (F = 4.84, $p<0.04$); F3 (F = 7.26, $p<0.02$); F4 (F = 7.70, $p<0.01$).

These differential group effects were elucidated by examining response amplitudes to the rare and frequent tones separately. These results are shown in Figure 2 averaged across electrodes. In the highly susceptible subjects there was little reduction of amplitude to the frequent stimuli (F=2.8, ns) in contrast to the sizeable reduction to the rare stimuli (F=10.80, $p<0.001$), in line with the results in frontal lobe patients. A different patterns of results was seen in the subjects with low hypnotic susceptibility in whom the staircase effect shown in Figure 1 was found to arise from alterations in the processing of both the rare and frequent stimuli. There was an increase in response amplitude to the rare stimuli with hypnosis per se (F=5.01, $p<0.05$), and no differentiation between the two hypnosis conditions (F=0.8). On the other hand there was a reduction in response amplitudes to frequent stimuli in the final hypnosis condition, giving rise to their greatest differentiation between the rare and frequent stimuli.

P300

The means and standard deviations for response amplitudes at Fz and Pz in the three conditions are shown in Table 1. There was a significant interaction between Group, Condition and Electrode (F=3.56, $p<0.04$). As can be seen in Table 1, the P300 response at Fz in the hypnotically highly susceptible subjects demonstrated the progressive reduction in amplitude found in the N100 to the infrequent stimulus; with hypnosis there was more than a 50% reduction in amplitude, while by the end of hypnosis there was only a negligible P300 (F=12.17, $p<0.008$). Importantly there was no parallel progressive reduction to the infrequent stimulus at Pz, where there was a 50% reduction from

Table 1. P300 amplitudes and latencies at Fz and Pz

	Susceptibility		Baseline		Hypnosis 1		Hypnosis 2	
			mean	sd	mean	sd	mean	sd
Amplitude μ	HIGH	Fz	12.30	4.37	5.25	5.45	0.12	4.91
		Pz	16.90	3.85	8.20	4.32	9.05	4.48
	LOW	Fz	12.78	4.12	10.08	5.34	9.33	5.12
		Pz	14.92	5.35	7.62	7.02	13.67	6.05
Latency msec	HIGH	Fz	318.38	17.39	320.25	14.12	320.63	15.14
		Pz	324.63	18.11	323.88	20.72	323.88	14.86
	LOW	Fz	327.50	26.03	325.33	30.52	324.40	23.83
		Pz	338.80	30.72	336.40	30.29	340.22	26.95

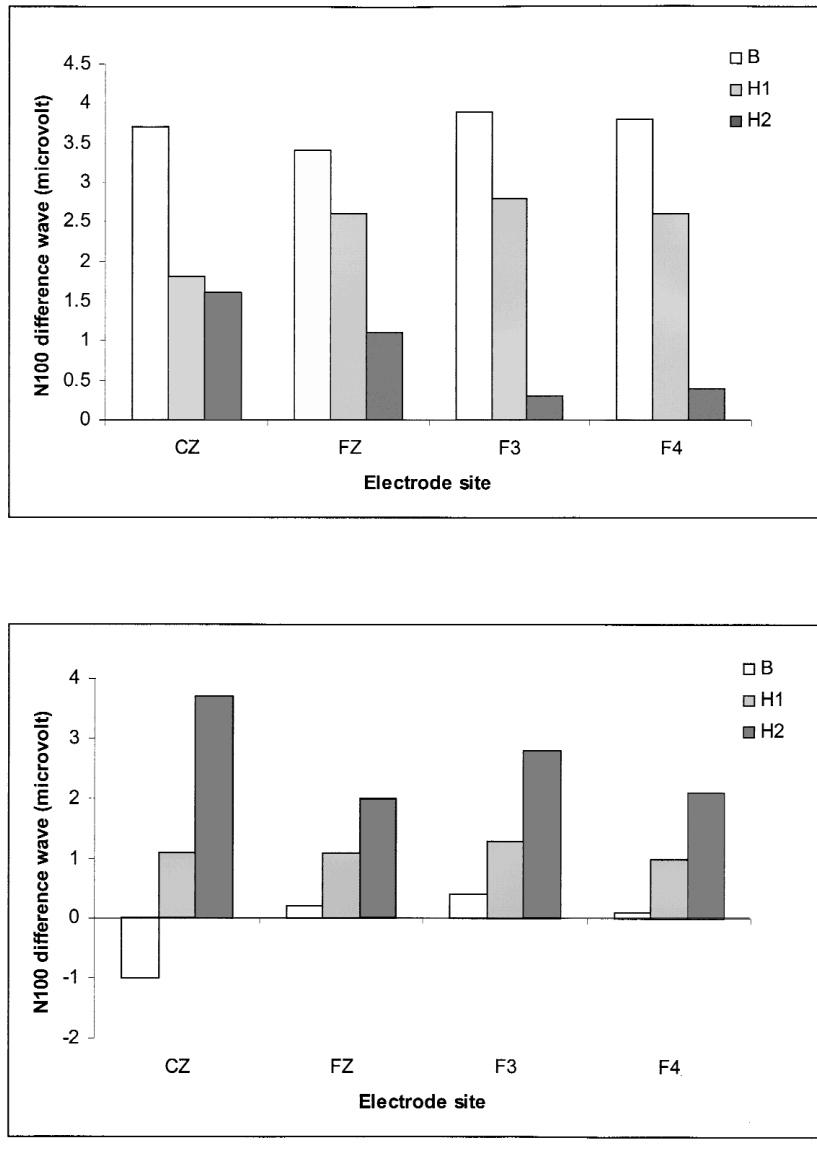

Figure 1. N100 difference wave across the three conditions at the four anterior electrodes showing progressive decreases in the highly susceptible group (upper) and progressive increases (lower) in the group with low hypnotic susceptibility.

baseline to hypnosis (F=11.05, $p<0.012$), but no further reduction during hypnosis. In the subjects low in hypnotic susceptibility there were no systematic changes at Fz or Pz (F=1.73, ns).

While there were no effects of condition on P300 latency (F=1.12), there was a significant interaction between Susceptibility and Electrode (F=5.94, $p<0.013$). Subjects low in susceptibility had significantly longer latencies at Pz than highly susceptible subjects ($p<0.02$), and their Pz latencies were longer than at Fz ($p<0.042$), whereas at Fz

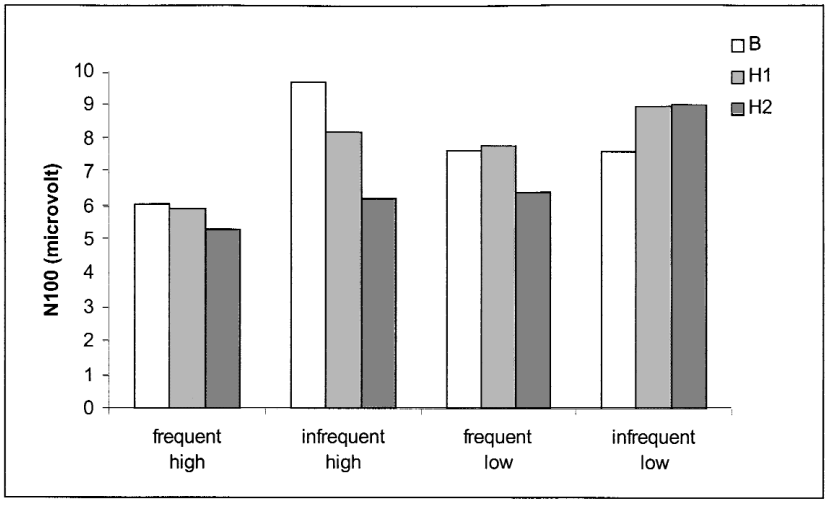

Figure 2. Comparisons between N100 amplitudes to infrequent and frequent tones.

there was no group difference (F=0.56). This result held across conditions and was not influenced by hypnosis.

Discussion

The results supported the hypothesized involvement of anterior brain processes in hypnosis. This was shown in the subjects with high hypnotic susceptibility by the attenuation of the N100d wave from the pre-hypnosis baseline to the 20 minutes of hypnotic induction, followed by a further reduction after approximately another 30 minutes of hypnosis. At this final hypnosis stage the N100d wave was virtually absent. Here the effects could not be attributed to order because they were not seen in the low hypnotic susceptibility group. Nor could they be attributed to a progressive reduction in generalized arousal as may occur with relaxation because there was not even a marginal reduction in the response amplitudes to the frequent stimuli in the high susceptibility group, shown in Figure 2. On the other hand, there was a reduction in the parietal P300 amplitude with hypnosis, which may signify a reduction in arousal and which did not vary between the earlier and later stages of hypnosis; relaxation was interpreted to have influenced brain rhythms in the posterior derivations in another study in the laboratory (Williams and Gruzelier, 2001).

Clearly by discounting arousal or order effects the effect of hypnosis was on attention, via progressive reduction in frontal activation which mediates processes of novelty detection. Firstly the decline in the N100d across the frontal chain was due to a progressive reduction in the response to the rare tone and not the frequent tone, which remained invariant in amplitude. Secondly there was a reduction in the P300 to the rare tone at the frontal placement; the frontal P300 is regarded predominantly as a response to novelty. Thirdly there was no progressive reduction between the earlier and later stages of hypnosis in the P300 amplitude at the posterior parietal placement; this reflects a temporo-parietal response to stimulus significance with typically a longer latency than the frontal P300.

It is noteworthy that cerebral blood flow and metabolism studies invoke the anterior cingulate in the frontal novelty detection process (Gabriel, 1990; Tzourio et al., 1997). The anterior cingulate has been widely implicated in the hypnotic process. Blood flow studies of hypnotic analgesia have implicated the anterior cingulate (Crawford et al., 1993; Rainville et al., 1999; Faymonville et al., 2000). The application of low resolution source estimation to the frontal gamma oscillation-pain relation disrupted by hypnosis (Croft et al., 2002) localized the effect to the anterior cingulate, which is in keeping with the fact that this structure has been strongly implicated in the subjective experience of pain. PET imaging of hysterical limb paralysis has disclosed anterior cingulate involvement (Halligan, Athwal, Oakley and Frackowiak 2000) providing the basis for a model of alterations of volitional effort in hypnosis (Oakley, 1999a, 1999b). Hypnotically induced hallucination has also been associated with the anterior cingulate (Szechtman et al., 1998) as has the revivification of pleasant memories (Maquet et al., 1999). Error detection as depicted by the error-related positivity and negativity waves, the former of which was abolished by hypnosis (Kaiser et al., 1997), has been localized to anterior cingulate (Dehaene, Posner and Tucker 1994). Furthermore, the higher error rate in the Stroop-like task of Kaiser et al. (1997) requiring inhibition of competing responses is consistent with compromise to the anterior cingulate, which has been implicated in the production of errors in blood flow imaging studies of the Stroop task (Pardo, Pardo, Janer and Raichle 1990; Bench, Frith, Grasby, Friston, Paulescu, Frackowiack and Dolan 1993).

The pattern of response in subjects with low hypnotic susceptibility disclosed several noteworthy characteristics. Firstly there was the opposite pattern of change in the N100d to the one found in the highly susceptible subjects, but more specifically there was no differentiation in the N100 between the rare and frequent stimuli at baseline. Interpreted in the light of resource models of attention this is compatible with a dispersion of attention away from the task at hand. It has been proposed that hypnosis requires engagement of frontal activation in the first place, which is achieved by strategies aimed at narrowing the participant's attention through fixation and so on (Gruzelier, 1990, 1998). This is accomplished by highly susceptible subjects, but is one factor underpinning the lack of susceptibility in low scoring subjects (Gruzelier, 1998, 2000b). The absence of the N100d provides evidence of this lack of frontal engagement in the service of focused attention in the subjects with low hypnotic susceptibility. Here this was not a trait marker for the less hypnotically susceptible subjects with practice progressively achieved the frontal activation signified by the N100d in the course of the experiment.

The second feature of note in the subjects with low hypnotic susceptibility was the longer latency of the parietal P300 compared with their frontal P300 and with both frontal and parietal P300s in the highly susceptible group. The conventional P300 amplitude is largest at the parietal location where its amplitude is associated with processing requirements; typically the latency of the P300 lengthens in normal subjects as the processing demands of the task increase (Donchin and Coles, 1988). Accordingly this may signify more prolonged processing of the rare stimulus, perhaps in compensation for the attenuated frontal involvement. The fact that this effect was stable across conditions suggests that it may signify a posterior processing bias trait in subjects with low hypnotic susceptibility and one which may militate against the hypnotic process that requires frontal engagement in the initial stages.

In conclusion support was found for the hypothesized progressive reduction with hypnosis in frontally modulated attention to auditory stimuli incidental to hypnosis in highly hypnotically susceptible subjects. This is in contrast to the view that attentional resources are increased in hypnosis (see for example, Crawford, 1994), a feature that was

found to characterize only the subjects with low hypnotic susceptibility in this study. The reduction in electrophysiological indices of attention was due to the attenuated responding to the infrequent stimulus, both in the N100 and the frontal P300, both of which signify frontal activation in the service of novelty detection. The frequent stimuli showed no progressive changes indicating (1) that the generators of exogenous components of the N100 of temporal lobe origin (Nataanen and Picton, 1987) were not altered by hypnosis, and (2) that a generalized reduction in arousal cannot account for the compromise in the novelty detection waves. The unexpected result was the opposite pattern of change in the N100 in the subjects low in hypnotic susceptibility, particularly their absence of the N100d in the pre-hypnosis baseline. This suggested a dispersion of attentional resources away from the experimental task and a lack of engagement of frontal novelty detection processes. Their longer latency of the posterior P300 may imply a posterior bias in processing, one that may militate against the induction of hypnosis and its frontal engagement, an engagement supported here by the results with the hypnotically highly susceptible participants.

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