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Psychopathology and Hypnotizability: From a Phenomenological Approach to the Neurobiological Perspective

Antonella Ciaramella[1,2]*

[1]*Psychosomatic Lab, GIFT Institute of Integrative Medicine, Lungarno Simonelli n.1, Pisa, Italy*

[2]*Aplysia APS, Partner in Education Program with University of Pisa, Florence, Turin, MIUR, Italy*

***Corresponding Author:** Antonella Ciaramella, Psychosomatic Lab, GIFT Institute of Integrative Medicine, Lungarno Simonelli n.1, Pisa and Aplysia APS, Partner in Education Program with University of Pisa, Florence, Turin, MIUR, Italy.

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Abstract

The debate about hypnosis as a psychopathological phenomenon dates back to the time of Charcot. Subsequent evidence is often contradictory because the instruments used to measure hypnosis and psychopathology differ. However, some studies have shown that the psychological processes which underlie mental disorders can be induced and reversed through hypnosis in experimental settings. Hypnosis has been used as instrumentally in the laboratory setting to study the nature of psychopathology in conditions responsive to hypnotic treatment. Indeed, hypnosis can elicit clinically compelling delusions that are phenomenologically and functionally similar to their clinical counterparts.

The aim of this study was to investigate a possible relationship between a single hypnotic phenomena and psychopathological dimensions in a clinical setting as a mechanism of modulating specific brain circuits, whose variation in connectivity may underlie certain mental disorders.

This retrospective observational study investigated 126 subjects using the phenomenological perspective in which the relationships between the 12 hypnotic phenomena, investigated via the Stanford hypnotic scale (SSHS), and the nine dimensions for psychopathology on the symptom check list 90-R (SCL90-R), highlight a relationship, between the hypnotic suggestion of amnesia and interpersonal sensitivity and anger/hostility. The ideomotor phenomena and hallucination, on the other, were revealed to be negative predictors of high scores on the depression dimension. Based on our results, we would like to propose several precautionary measures regarding the utilization of ideomotor phenomena and hallucination suggestion in the treatment of depression, instead recommending a predictive approach and intervening on the hypersensitivity to rejection through the suggestion of amnesia. These findings are discussed from a neurobiological perspective.

Keywords: *Psychopathology; Hypnosis; Ideomotor Phenomena; Amnesia; Neurobiology; Depression; Hypersensitivity to Rejection; Phenomenology*

Abbreviations

ACC: Anterior Cingulate Cortex; CRF: Case Report Form; DMN: Default Mode Network; GSI: General Symptom Index; MDD: Major Depressive Disorder; MTL: Medial Temporal Lobe; PST: Positive Symptom Total; SN: Salience Network; SHSS: Stanford Hypnotic Susceptibility Scales; SCL-90-R: Symptom Checklist-Revised; VMPFC: Ventromedial Prefrontal Cortex

Introduction

In the MeSH (Service of the National Library of Medicine and the National Institutes of Health) and PsycINFO Thesaurus, psychopathology is defined as 'the study of significant causes and processes in the development of mental illness'. In other references (e.g. Scopus), 'psychopathology' is used as a synonym for 'symptoms'. Going back to the Heidelberg School and Jaspers, psychopathology is described as 'phenomenology' as it 'provides a concrete description of the psychic states that patients actually experience' [1]. As a discipline, Psychopathology is the study of individual morbid psychological phenomena, irrespective of the various clinical disorders in which they occur. It deals only with a subset of symptoms of semiotic relevance. In this context, brain network patterns of cognitive control have been found to be fundamental to advancing our understanding of psychopathology and cognitive dysfunction in various psychiatric disorders (e.g. schizophrenia, depression, etc.) [2,3]. Thus, the phenomena that emerge in a patient's consciousness can be considered a mirror of dysfunctions in brain circuits.

Charcot [4] and Janet's [5] old controversial belief about the relationship between hypnotizability-that is, the ability to respond to hypnotic suggestions-and psychopathology remains unclear even today. Although Bernheim, in the same period [6], suggested considering susceptibility to hypnosis as a natural phenomenon, the controversy has persisted and continued to condition the clinical applicability and usefulness of hypnosis. Spiegel., *et al.* [7] attempted to settle the debate, showing that subjects with psychopathology had a lower susceptibility to hypnosis than healthy subjects. However, when interpreting such findings, it should be borne in mind that later studies have shown that this relationship is dependent on the hypnotic scale used [8].

That being said, an old but very important paper by Kihlstrom [9] reported that the psychological processes that underlie cognitive disorders, namely dissociation, hallucinations, delusions and pain, can be induced and reversed through hypnosis in normal human subjects with no history of trauma and/or psychiatric disorders in several rigorous experimental settings. Hence, hypnosis has been used instrumentally in the laboratory setting to study the nature of psychopathology in conditions responsive to hypnotic treatment [10]. Indeed, hypnosis elicits clinically compelling delusions that are phenomenologically and functionally similar to their clinical counterparts. We could therefore think that a hypnotic phenomenon, being able to generate a psychopathological symptom, can at the same time act selectively on that symptom and, when present, modulate it and perhaps extinguish it. This hypothesis is based on evidence relating to neurobiological mechanisms, generating psychopathological symptoms, can be regulated by specific hypnotic phenomena.

Aim of the Study

The aim of this study was to investigate a possible relationship between individual hypnotic phenomena and dimensions of psychopathology as a mechanism of accessing the modulation of specific brain circuits whose variation in connectivity may underlie mental disorders.

Materials and Methods

The data for this retrospective observational study was collected in the period between 2020 and 2024 at the of Pisa Gift Institute of Integrative Medicine (GIIM) Psychosomatics Laboratory, Italy. The sociodemographic information was collected using a Case Report Form (CRF), and mental health, psychosomatic problems and hypnotic susceptibility were also assessed at first contact using the tools detailed below.

Stanford hypnotic susceptibility scales, form A

The Stanford hypnotic susceptibility scales (SHSS), Form A is still used today to determine the extent to which a patient responds to hypnosis. The SHSS Form A consists of a series of 12 activities in response to specific suggestions delivered under hypnosis; scoring on the SHSS ranges from 0 (no response to any of the hypnotic suggestions) to 12 (a response to all 12 hypnotic suggestions) [11]. The items correspond to 12 phenomena: 1. Postural sway; 2. Eye closure; 3. Hand lowering (left); 4. Immobilization (right arm); 5. Finger lock; 6.

Arm rigidity (left arm); 7. Hands moving together; 8. Verbal inhibition (name); 9. Hallucination (fly); 10. Eye catalepsy; 11. Post-hypnotic (changes chairs); and 12. Amnesia.

The symptom checklist-revised (SCL-90-R)

The SCL-90-R is a self-administered questionnaire designed to measure psychopathology [12]. It consists of 90 items scored on a Likert Scale from 0 (not at all) to 4 (very much). Nine symptom dimensions are assessed via the relevant subscales: Somatization, obsession-compulsion, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation and psychoticism. The SCL-90-R also features three global indices used to provide measures of overall psychological distress: the global severity index (GSI), the positive symptom total (PST or SCL90-R total score) and the positive symptom distress index (PSDI).

Statistical analyses

Data were analyzed using IBM SPSS statistics 26. The sample was divided into two groups, respectively comprising subjects who presented psychopathology according to the SCL-90 global severity index and those that did not (GSI ≤ .70) [13]. Application of the Kolmogorov-Smirnov test revealed the non-normal distribution of our data, so non-parametric analyses were performed. Spearman correlation was used to investigate the relationships between psychopathology and hypnotizability. Subjects were subdivided based on their degree of hypnotizability using Thompson., *et al.*'s evidences [14]: Lows (0 - 4), mediums (5 - 7), and highs (8 - 12), and Mann-Whitney and Kruskal-Wallis tests were applied to compare psychopathological dimensions between two and three groups, respectively. Chi-squared analyses were performed to investigate differences in hypnotic phenomena among the three subject groups. Logistic Regression analysis was also performed to investigate the strength of each association and the predictiveness of each hypnotic phenomenon for each psychopathological dimension.

In the tables, the means are reported instead of ranks and the median for a clearer understanding of the data.

Results

Presence of psychopathology and hypnotizability

The sample was divided based on the GSI cut-off (.70) into those with low psychopathology (n. 36) and those with high psychopathology (n. 90). Subjects with psychopathology were older (mean 41.41 years, SD: 15.51) than those with a GSI ≤.70 (mean 31.05 years, SD: 9.74) (z = 3.47, p = .001). Conversely, the GSI>.70 group had had less education (mean 15.84 months; SD: 3.21) than the GSI≤.70 group (mean 17.99 months; SD: 4.95) (z = 3.09, p = .002). However, we found no differences in age and education when the group was divided according to the degree of hypnotizability. Subjects without psychopathology according to the SCL-90-R were more prevalent in the low hypnotizability group, while only three of the subjects with high GSI of hypnotizability (7%) had no psychopathology (Figure 1).

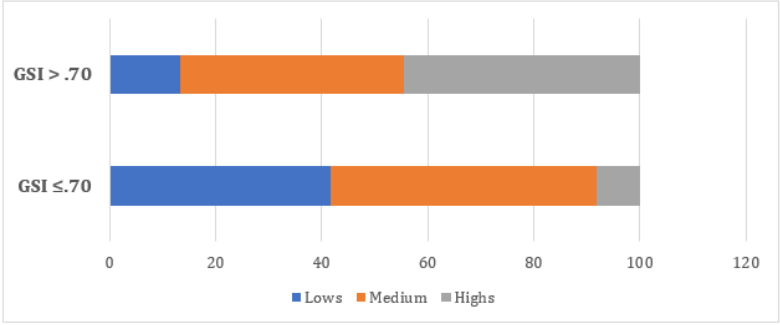

Figure 1: *Difference (in %) in the degree of hypnotizability between subjects with presence (GSI >. 70) and absence (GSI ≤ .70) of psychopathology. Lows are more prevalent in the group with no psychopathology (χ2 = 19.80; p = .000; ϕ = .397).*

Difference in hypnotic phenomena in the sample distinguished based on degree of hypnotizability

A difference in the prevalence of ideomotor phenomena was found when subjects were distinguished based on degree of hypnotizability. There was a lower prevalence of Eye Catalepsy (item 10) in the Lows group as compared to the Highs, a difference that is statistically significant but only reached a medium-high effect size (Table 1). In the Lows, item 10 was the least prevalent hypnotic phenomenon, whereas in the Highs the Finger Lock (item 5) was the most prevalent. This was a statistically significance difference, even though the phenomenon was also frequent, on average, in the lows.

	Lows		Medium		Highs		χ^2	p	ϕ
	n	%	n	%	n	%			
Item 1	12	44.4	38	67.9	40	93	19.80	.000	.397
Item 2	9	33.3	32	57.1	34	77.3	13.51	.001	.326
Item 3	14	53.8	32	57.1	32	72.7	3.44	ns	.165
Item 4	4	14.8	29	52.7	31	70.5	20.87	.000	.407
Item 5	9	34.6	40	71.4	40	90.9	24.99	.000	.445
Item 6	7	25.9	31	55.4	35	79.5	19.86	.000	.359
Item 7	11	40.7	38	67.9	36	81.8	12.79	.002	.317
Item 8	3	11.1	26	46.4	32	72.7	25.55	.000	.449
Item 9	3	11.1	8	14.3	20	45.5	18.26	.000	.358
Item 10	2	7.4	26	46.4	38	86.4	43.02	.000	.582
Item 11	7	25.9	26	46.4	26	59.1	7.39	.025	.241
Item 12	6	22.2	20	35.7	18	40.9	2,63	ns	.144

Table 1: *Prevalence of hypnotic phenomena in the sample distinguished BY degree of hypnotizability.*

Table 1: 1. Postural Sway; 2. Eye Closure; 3. Hand Lowering (left hand); 4. Immobilization (right arm); 5. Finger Lock; 6. Arm Rigidity (left arm); 7. Hands Moving Together; 8. Verbal Inhibition; 9. Hallucination; 10. Eye Catalepsy; 11. Post-hypnotic suggestion; 12. Amnesia.

Psychopathological dimensions and hypnotic phenomena

Distinguishing the sample into three groups based on the degree of hypnotizability revealed no differences among them in scores for each dimension of psychopathology. The sum obtained from grouping all the ideomotor phenomena was, however, correlated with the scores for each psychopathology dimension on the SCL90-R. Nonetheless, no statistically significant correlation was found.

The same analysis was performed to investigate the relationship between psychopathological dimensions, hallucination and memory. Only Amnesia appears to show statistically significant correlations with Interpersonal Sensitivity ($r = .189$; $p = .037$); Anger/Hostility ($r = .204$; $p = .021$), and the General Symptom Index (GSI) ($r = .191$; $p = .032$), albeit all with a low effect size.

Hypnotic phenomena as predictors of psychopathology

Performing binary logistic regression using the items on the SSHS as dependent variables and the SCL 90-R dimensions as independent variables revealed no hypnotic phenomena are positive predictors of psychopathology; instead several items appear to negatively predict increasing levels of depression. Specifically, items 1, 5, 9 and 10 were found to be negative predictors of higher depression scores. The same was true for anxiety, as there was a negative association between the finger lock phenomenon (item 5) and high scores for anxiety (Table 2).

Phase 1								95% C.I. for Exp(β)	
		B	S.E.	Wald	df	p	Exp(β)	Lower	Higher
Item 1	Depression	-2.90	1.09	7.12	1	.008	.055	.006	.042
Item 5	Depression	-4.32	1.82	5.61	1	.018	.013	.000	.473
	Anxiety	-2.92	1.11	6.86	1	.009	.054	.006	.479
Item 9	Depression	-3.31	1.63	4.12	1	.042	.036	.001	.891
Item 10	Depression	-2.14	.857	6.29	1	.012	.117	.022	.625

Table 2: *Binary logistic regression using SSHS items as dependent variables and SCL 90-R dimensions as independent variables.*

Table 2: 1. Postural Sway; 5. Finger Lock; 9. Hallucination; 10. Eye Catalepsy.

Discussion

The observational nature of this study allowed us to observe phenomena as they appear, without any tampering. If we consider the Global Severity Index (GSI) of the SCL-90-R, our findings appear to diverge from the established literature [7], as a lower GSI was more frequently observed in the group with low hypnotic susceptibility. However, the results of our logistic regression reveal that both the ideomotor and perceptual items of the Stanford Scale act as negative predictors for anxiety and depression.

This nuanced relationship is further underscored when analyzing individual hypnotic phenomena: no significant correlations emerged with psychopathological dimensions, with the notable exception of Amnesia (Item 12). This specific item correlates positively with Interpersonal Sensitivity, Anger, and GSI scores, albeit with a small effect size. Consequently, it appears that subjects responsive to amnesia suggestions may exhibit a heightened sensitivity to judgment and rejection, alongside a predisposition toward anger and hostility. Taken together, our results demonstrate that the hypnosis-psychopathology nexus is not monolithic; rather, it varies significantly depending on the specific psychopathological dimension explored and the nature of the hypnotic suggestion induced.

Neurobiological studies make us aware that social rejection-a condition predisposing individuals to major depressive disorder (MDD)-is associated with an increased response by the amygdala to negative social appraisal. The increased amygdala activity during social rejection is associated with greater functional connectivity between the insula and anterior cingulate cortex (ACC) and ventral medial prefrontal cortex (VMPFC) [15]. These studies therefore also inform us that there is a relationship between amnesia and amygdala hyperactivity during temporary (< 1h) epilepsy of the medial temporal lobe (MTL) [16]. Hence, amnesia is a phenomenon related to an alteration in the amygdala's activity in a condition of temporary epilepsy.

Drawing on Kihlstrom's laboratory studies [9], and neurobiological research on the connectivity of the amygdala and the MTL, the latter can be modulated using a specific hypnotic suggestion like amnesia. As described by Holmes., *et al*. [17] amnesia is a dissociative phenomenon explained by psychological mechanisms of compartmentalization and detachment. In this context, amnesia may act either through detachment, reducing (regulating) hypersensitivity to rejection, or through compartmentalization, separating and disabling certain control functions (e.g. judgment). Neurobiological studies demonstrate an involvement and deactivation of the Default Mode Network (DMN) in abnormal distress following situations that require social interaction (i.e. Social phobia) [18]. Furthermore, an increased within-network connectivity of posterior/lateral DMN regions (precuneus, angular gyrus, superior/middle temporal cortex) is more strongly and positively correlated with symptom improvement in adults with depression [3]. Therefore, we can use amnesia to modulate a psychological factor (hypersensitivity to social rejection) that predisposes to depression. Hence, we could exploit the compartmentalization and detachment mechanism of hypnotic amnesia to act on factors predisposing to depression (e.g. rejection hypersensitivity), specifically, by modulating the activity of the amygdala and thereby preventing the dysfunctional connectivity of the DMN that underlies the onset of depression.

In addition, this study highlights several hypnotic phenomena as negative predictors of depression, namely ideomotor phenomena, and more specifically catalepsy (e.g. of the eyelids). As Yapko has reported in a recent editorial [19], the treatment of depression has many facets. Until a few years ago, hypnosis was discouraged as a therapy, because it impairs a person's ability to focus (main mechanism of hypnosis), or due to unfounded fear that hypnosis would strip the defenses of individuals with depression, making them more vulnerable to psychosis or suicide. Nonetheless, difficulties in treating depression do not only concern hypnosis, but also pharmacological treatments, which frequently seem ineffective, leading to the growing use in recent years of substances that modify the normal state of consciousness and have dissociative properties [20].

The results of our study give indications on which phenomena fail to act on the Depression dimension, and therefore would be unhelpful during hypnotic treatment of depression. From a neurobiological perspective, our results also tell us that these phenomena do not directly modulate the circuits that underlie depression. The motor circuits involved in motor functional disorders have been demonstrated [21], in which the ventromedial prefrontal cortex (VMPFC), precuneus (a key region for accessing internal representations about the self) and, perhaps, the amygdala are frequently found to be hyperactivated in paralysis due to conversion disorders. In parallel with this is impaired recruitment of primary motor and/or sensory pathways at the cortical or subcortical basal ganglia [22]. Hypnotic paralysis increases activity of the precuneus-generally included in the DMN even though recent studies consider it part of a different network (parietal memory network or precuneus network) [23]-but not the VMPFC. Thus, if we consider DMN dysfunction as the basis of depression, our data suggest that ideomotor phenomena are not able to directly modulate this circuit. We can make the same consideration for anxiety. In fact, our data show that ideomotor phenomena are negative predictors of anxiety. Recent neurobiological studies have highlighted dissociable roles for the salience network (SN) and DMN in the pathophysiology of anxiety symptoms, with psychological symptoms being associated with DMN hyperactivity and somatic symptoms with SN hyper function [24].

Another negative predictor of depression was found to be item 9, that is, the suggestion of a hallucination of a fly. In depression, anomalies of the suggested imagination are reported due to the persistence of intrusive negative mental images; the positive image is impoverished and there is a sort of bias for the image of the observer's perspective and a tendency to general images in which specific images are missing. Therefore, we can consider that in as part of depressive psychopathology there is a dysfunction of the image [25]. In the hallucination of the fly, the suggestion includes the sensation of the fly touching a part of the body. There is not much evidence on imagination from neurobiological studies but it seems that the imagination of touch is encoded by our brain differently than the imagination of movement [26].

Conclusion

The functional change in brain activity following psychotherapy is well documented [27,28]. Furthermore, non-pharmacological treatments that modify the normal state of consciousness, such as hypnosis and meditation, appear to be associated with changes in brain activity [29]. This study addresses this issue starting from phenomena observed during clinical practice, with a view to their possible neurobiological implications. Our results lend weight to hypnotic phenomena being able to direct us to personalize treatments in the field of mental disorders from a phenomenological perspective, with specific hypnotic phenomena being used to selectively modulate the function of specific brain networks that underlie mental disorders.

Although in our sample a greater number of subjects with low psychopathology were less hypnotizable, no significant relationships were found between susceptibility to hypnotic ideomotor phenomena as a whole and any individual psychopathology dimension. While single ideomotor phenomena were found to be negative predictors of high levels of depression. Based on our results we recommend the adoption of specific precautions in the use of ideomotor phenomena and Hallucination suggestion in the treatment of depression, instead recommending a predictive approach and intervening on the hypersensitivity to rejection through the suggestion of Amnesia.

The results of this study also highlight that the response to single suggestions (i.e. ideomotor, hallucinatory or amnesic) differs according to the degree of hypnotizability; for example: eye catalepsy seems to be less elicitable in subjects with low hypnotic susceptibility.

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Conflict of Interest

No conflict of interest to declare.

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