

Hypnosis and Modern Frontal-Lobe Concepts - A Sketch for a Review and an Invitation to One Particularly Promising Field

Muzur, Amir

Source / Izvornik: **Collegium antropologicum, 2006, 30, 205 - 211**

Journal article, Published version

Rad u časopisu, Objavljena verzija rada (izdavačev PDF)

Permanent link / Trajna poveznica: <https://urn.nsk.hr/urn:nbn:hr:184:554645>

Rights / Prava: [In copyright](#)/[Zaštićeno autorskim pravom.](#)

Download date / Datum preuzimanja: **2025-01-09**



Repository / Repozitorij:

[Repository of the University of Rijeka, Faculty of Medicine - FMRI Repository](#)



Hypnosis and Modern Frontal-Lobe Concepts – A Sketch for a Review and an Invitation to One Particularly Promising Field

Amir Muzur

Department of Family Medicine, School of Medicine, University of Rijeka, Rijeka, Croatia

ABSTRACT

The present paper intends to briefly review the most important concepts of the modern neuropsychology of the frontal lobes, and to relate these findings to the phenomenology usually encountered in hypnosis research and practice. The frontal lobes have been studied very intensively during the last several years and some of the results, including the syndromes described in frontal-lobe lesions and psychiatric patients, demonstrate striking similarity with hypnotic phenomena. Based on these similarities, an alternative neuropsychophysiological definition of hypnosis/suggestion is proposed, viewing hypnosis/suggestion as the process of external manipulation with frontal-lobe functions with consequent effects upon the entire brain potential of the subject.

Key words: hypnosis, frontal lobe, anterior cingulate, confabulation, memory

Introduction

Hypnosis and hypnosis-like states have been known and used most probably since the beginnings of human race. The principles of their functioning, nevertheless, fall still far from our understanding. At that point, hypnosis results similar to electricity: static charge has been applied very early in human history, but one had to wait for its explanation until the second half of the 19th century. Frustrating for us but true, the age of explicating hypnosis has not come yet. We did come closer, however. Frenetically measuring everyone and everything, we have started experiments demonstrating activations of the various parts of the brain. And so we arrive to the story of the frontal lobes.

Firstly, one should admit that our traditional division of the brain into lobes is fully inappropriate, since it has nothing to do with the function. Therefore, it is much better to speak of the involvement of the prefrontal cortex, which does reduce the neuronal mass we are dealing with but, unfortunately, not the complexity. Since it is characterized by several general qualities, it is justified to consider the prefrontal cortex a functional entity per se, without contradicting the fact that different cortical areas might be servicing different input regions, as Patricia Goldman-Rakic suggests¹, or supporting even different subfunctions, as Michael Petrides wants it². Precisely

this idea, that is, that the prefrontal cortex somehow co-ordinates or employs the functions of more posterior brain areas (which reminds of the function of the hypothalamus within the endocrine system), might have been a starting point for locating the processing of hypnotic phenomena within the frontal lobes. If one understands suggestion as a system of triggers and inhibitions directed towards various cerebral activities, than the frontal lobes might be the right place to look for the mechanisms supporting this system.

A Frontal-Function Primer

Although elementary division of the frontal lobes differentiates also the primary motor area and the premotor cortex, we shall concentrate upon the prefrontal cortex, standing at the top of the organization of behavior. Functional reasons suggest to divide the prefrontal cortex into dorsolateral (DLPFC) and ventromedial (VMPFC) parts.

Dorsolateral prefrontal cortex

For about three decades already, the implication of the prefrontal cortex in working memory (visuospatial;

representational memory) has intensively been studied³. In monkeys, it has been found that »the principal sulcus contains a mechanism for guiding responses on the basis of stored information in the absence of external cues«, the memories for object locations being mapped in visuo-spatial co-ordinates⁴. Lesion experiments suggest the dorsal prefrontal cortex has a role in the temporal order of information retrieval, while temporal-lobe lesions proved to have no influence on this capability (but only on visual recognition)⁵. Some human experiments have indicated that the activation of the same prefrontal area 46 (middle frontal gyrus) was correlated with the performance of a spatial working-memory task⁶. A more recent study of humans by Courtney et al.⁷ described the area specialized for spatial working memory to be located »more superiorly and posteriorly than in the monkey.«

Some psychological theories quite precisely match what we know about dorsolateral prefrontal cortex from experience with patients. So it is with Shallice's Supervisory Attention System (SAS), involved in the choice of non-routine procedures^{8,9}. The routinized choice of behavioral schemata, called contention scheduling, is inhibited by the SAS. A test sensitive to these functions is the Tower of London. Lesion studies confirmed the prolongation of response-initiation reaction times and the appearance of difficulties in response suppression (where the expected response was to complete a sentence)¹⁰. The hypothesis of a Central Executive¹¹, where the outputs from a phonological store and visuospatial scratchpad are supposed to meet, could very likely be nested in the prefrontal cortex as well.

Ventromedial prefrontal cortex

According to Cummings^{12,13}, two functional circuits are to be differentiated in the VMPFC: the medial prefrontal circuit includes an anterior cingulate/ventral striatum/mediodorsal thalamic loop plus multiple limbic, brainstem, and cortical inputs; while the orbitofrontal circuit includes an orbital prefrontal cortex/dorsal and ventral striatal area/mediodorsal and ventral anterior thalamic loop plus multiple limbic, brainstem, and cortical inputs.

Along with ventromedial aspects of other prefrontal areas such as the orbitofrontal cortex, the anterior cingulate plays a key role in decision-making, social cognition, and social judgment^{14–16}. This concept has been most clearly enunciated in Damasio's somatic marker hypothesis which ascribes to the ventromedial prefrontal cortex the role of »marking« complex stimuli (such as social situations) with records of past autonomic and emotional responses thereby making possible high level social judgments such as behavioral inhibition and assessment of complex social cues^{16,17}. There is evidence that the capacity to mind-read as well as its more refined sequelae in the social competencies of adult humans is subserved at least in part by ventral and medial portions of the prefrontal cortex^{18,19}.

On the origins of action

The study of patients with frontal damage revealed precious hints on the role of the frontal lobes in the organization and control of action. The »unblocked« behavior (»magnetic apraxia«²⁰, »utilization behavior« or »environmental-dependency syndrome« and »imitation behavior« as the two stages^{21,22}), following some frontal-lobe lesions, was described in details by Lhermitte and collaborators in the 1980s²³. The patients were noted to use knife and plate even if not hungry (immediately after lunch, for instance), to put three pairs of glasses at the top of one another, to write, drink, or urinate when paper, water, or night pot, respectively, had been presented to them. A patient with multiple bilateral infarctions in upper frontal convolution, cingulate gyrus, and right temporal gyri, used to read street- and trade names – practically each letter in the visual field, and to calculate with automobile license numbers so as to reach »special meaning«²⁴. Compulsive counting and eating were noted in a patient with medial and orbital frontal damage, with spared basal ganglia (in addition, the patient had difficulties in recognizing funny situations and demonstrated episodic amnesia and anomia²⁵). Vascular lesion in the territory of pericallosal artery was found to provoke spontaneous grasping of objects that previously had been touched by the hand (tactile stimulus to the palmar surface). The patient, aware of this behavior, could stop it only by starting another movement. The authors suggested this grasp reflex be a cutaneous spinal reflex unlike »other grasping behaviors«²⁶. A lesion of thalamo-cortical pathways (paramedian thalamic region) can result in utilization behavior²⁷, as well as a combined lesion of left anterior cingulum and the head of the right caudate nucleus (which brought some authors to the idea that cingulum and caudatus should be considered as a functional complex)²⁸. Patients with left ventral inferior frontal damage often try to verbally inhibit the action (saying »No!«), but then still perform it²⁹. In another case of utilization behavior, the patient was caught to pick up cards and deal. Goldberg et al.³⁰ describe utilization behavior (although not calling it so) as a symptom of the »alien-hand sign« (beside forced grasp and motor perseveration; all limited to the contralateral hand). Shallice and collaborators³¹ noticed that utilization does not occur only when objects are presented directly to the patient (induced behavior), but even when they are beside the patient and he is ordered another task (incidental behavior). The findings were interpreted as the lack of Supervisory Attention System and the random triggering of action scheme. At any rate, it is justified to view utilization behavior as a pathological (uninhibited) version of visuo-motor priming³². Following the logic of some of the described cases where aneurysm of the anterior communication artery was found (with destroyed head of the caudate nucleus), Lhermitte hypothesized that orbital frontal cortex might be responsible for the supervision of action start (releasing or not parietal lobe activities^{22,23}). The idea that (contralateral) orbitofrontal cortex and/or anterior cingulum might really be in char-

ge of motor inhibition was supported by the finding³³ of their activation in hysterical paralysis (at the same time, there was no activation noted in the DLPFC and premotor cortex). Carmichael and Price³⁴ were more precise, assigning to area 13 a motivational signal which, in normal circumstances, decides whether a plan from the premotor cortex will be started or not (which is in accordance with the finding that sensory-to-motor transformation, i.e., the realization of a plan in the premotor cortex takes place only if the action is connected with reward)³⁵. If area 13 is damaged, logically, an automatic sensory-to-motor transformation occurs (that is, utilization behavior). The parallel of hypnosis with utilization behavior has already been noted and discussed at some length in a recent study by Woody and Szechtman³⁶.

Combining data from a long list of experiments and clinical experience, Deecke et al.³⁷ suggested a general strategy of action: 1) WHAT to do (decision to be brought in the orbitofrontal region receiving input from hypothalamus and limbic system; this part should balance motivation, moral judgement, and the inhibitory control of primitive drives); 2) HOW to do (frontolateral cortex receiving afferents from the posterior sensory cortices); 3) WHEN to do (frontomesial cortex, especially SMA with its Bereitschaftspotential – »action timing«, a lesion in this region leaves the possibility of re-acting, but not to act spontaneously).

More complex clinical hypotheses of the frontal lobe function are still waiting to be proved or disconfirmed: attention deficit disorder (ex »hyperkinetic reaction of childhood«) has been linked to the disturbance of frontal-lobe inhibitory functions³⁸; schizophrenia has been linked to deficits of prefrontal dorsolateral surface; obsessive-compulsive disorder was noted to follow a higher activity in orbitofrontal cortex^{39,40}. Posner and Petersen⁴¹ have investigated the role of frontal cortex in resistance to distraction and sustained attention. It is not by chance that neuroscience is searching in the frontal lobes for the underpinnings of the most sophisticated human abilities, since here, the roots of action as well as the roots of thought seem to intermingle.

Frontal Lobes and Hypnosis: A History of Linking

In the mid of the 1970-ies, Pribram and McGuinness⁴² proposed a model of attention explaining that the prefrontal cortex controls the limbic system in the gating of sensory stimuli arriving from the periphery. The model was corroborated by more recent ERP studies demonstrating the involvement of the prefrontal cortex in the inhibition of the conscious perception of pain⁴³ and by neuroimaging studies differentiating certain and uncertain expectation of pain, activating the rostral anterior cingulate cortex and ventromedial prefrontal cortex⁴⁴. Moreover, Helen Crawford's studies revealed an increased cerebral blood flow in the orbito-frontal cortex during hypnotic analgesia⁴⁵, which was explained as an increase in attentional efforts⁴⁶: more precisely and more proba-

bly, what has been activated is an inhibitory loop localized in this region.

The finding that highly-hypnotizable persons have more efficient frontal attentional and inhibitory systems, has been stressed in a series of papers^{47,48}. Parallely, Spiegel & King⁴⁹ found a positive correlation between dopamine activity (as a measure of frontal involvement) and hypnotizability. More difficult, however, has been to localize the activity within the frontal lobes. While Halama⁵⁰, using SPECT, reported an increase in CBF during hypnosis (termed »frontalization«) particularly in the right hemisphere, John Gruzelier's group revealed a shift during hypnotic induction, transferring cerebral activity peaks from the left frontal cortex towards right posterior regions of the brain⁴⁸. This shift, with an expressed inhibition of frontal function occurring as soon as focal attention systems are activated⁵¹, might be a reflex of the task performance in which the subject is engaged after the induction (primarily imagery). The initial increase in frontal perfusion, however, does occur also in »empty« hypnosis experiments, i.e., hypnotic induction without subsequent tasks (»basic hypnotic state« with deep relaxation and no specific suggestion⁵²), as stressed by Walter⁵³. Gruzelier and Warren⁴⁸ ascribe the most prominent hypnotic features, like suspension of reality testing, abdication of planning functions, and reduced attentional monitoring of external cues, to the inhibition of frontal functions following the left to-right and anterior-to-posterior shift of activity. The results of the more recent PET and EEG study by Rainville and collaborators⁵⁴, however, do not agree with those of Gruzelier. Rainville finds an increase in rCBF in the inferior frontal gyri during hypnotic relaxation, but even additional widespread increases in rCBF in the predominantly left frontal cortices during hypnosis with suggestions for altered pain unpleasantness. More recently, there have been experimental attempts at relating hypnosis to frontal-executive dysfunctions. Since the main result was that, in a baseline condition, high susceptible individuals had performed significantly better on the Wisconsin Card Sorting Test⁵⁵, those attempts will still have to wait for a final confirmation⁵⁶.

Anterior Cingulate

A frontal structure that, beside the orbitofrontal cortex, proved to be involved in some of the hypnotic phenomena is the anterior cingulate. The role of this structure in hypnotic analgesia was evidenced by Helen Crawford's group⁵⁷. This finding coincides with the data obtained by Marshall et al.³³, who revealed significant PET activity both in orbitofrontal region and in the anterior cingulate during a movement trial in a patient with hysterical limb paralysis. In this case, the anterior cingulate was found to suppress inappropriate motor responses, triggered, interestingly, by volitional effort only.

The area 32 of the right anterior cingulate was found to be active in a group of hallucinators when they heard an auditory stimulus and when they hallucinated hear-

ing it but not when they merely imagined hearing it⁵⁸. The study, performed with PET, did not detect the same activation in the group of highly hypnotizable nonhallucinators. The authors emphasized the similarity of the described activations with those previously seen in schizophrenic hallucinations.

The involvement of the anterior cingulate in hypnosis might be responsible also for a higher error rate at the Stroop task (reflecting a failure in the inhibition of automatic responses), found in highly hypnotizable subjects⁵⁹. Gruzelier suggests that, what occurs during hypnosis, is a dissociation between cognitive-executive and affective-executive functions of the anterior cingulate, the former being related to response selection while the latter being engaged in the regulation of autonomic and endocrine functions (for new data relating human cingulate cortex to autonomic control, see Critchley et al.⁶⁰). According to Gruzelier⁶¹, cognitive-executive component remains intact during hypnosis, while the affect system ceases to function. Knowing that hypnosis is related to the inhibition of amygdala⁶², which is strongly interconnected with the anterior cingulate, the fact that this component remains unresponsive, does not surprise any more and fits quite well to the image of hypnosis as an emotionally underbalanced state, characterized by a significant and measurable decrease in the emotional impact of pain perception⁶³ or of prosodic imitation⁶⁴, for instance. The involvement of anterior cingulate, in addition, could explain the gating of sensory input arriving via thalamus and the consequent motor response selection. The activity of anterior cingulate during imagery tasks, as revealed by the study of Stephan et al.⁶⁵, again fits well to hypnosis, especially to its more advanced phases.

The Lateralization Problems, Solutions, and New Problems

The lateralization issue has been a particularly vividly discussed problem during the last several decades already. The earlier widely accepted idea of the predominance of the right hemisphere activity in hypnotic processing⁶⁶ recently was challenged by a series of studies finding no lateralization at all. Crawford and collaborators⁴⁵ supported such a view by a xenon-rCBF experiment, and the first PET study of hypnosis⁶⁷ agreed with them. Therefore, although no final decision can be made, at the present state of knowledge, it seems far more reasonable to avoid speaking of hypnosis as a »right-hemisphere task.«

Recent advances at the lateralization issue, nevertheless, open new problems on the other side. If conscious processing (whatever this might be) is connected with the left (dominant) prefrontal cortex, as suggested by certain studies⁶⁸, and if the same left prefrontal cortex is able to modulate the ipsilateral amygdala being in charge of »positive« emotions, and if the involvement of the left hemisphere results in the up-regulation of the immune system⁶⁹, than what is occurring with all those parameters during post-induction hypnosis, when the left pre-

frontal cortex becomes inactivated? Does it mean that only the phase of hypnotic induction can procure benefits for the health of the subject, while the continuation of the session down-regulates the immune-system activity level and stirs up negative emotions? Common experience does not agree with this theoretical logic. On the contrary, hypnosis and self-hypnosis have been used to increase creativity, enthusiasm, and health in general. Oppositely to theoretical predictions that hypnosis shifts the cerebral activity from left to right, it has been demonstrated that, during hypnosis, an increase in vagotonus occurs⁶³, and it is known that parasympathetic hyperactivation is connected with the stimulation of left human-brain structures (insula)⁷⁰ and corresponds to the effects of some other non-hypnotic relaxation techniques like yoga or meditation⁷¹. Maybe the key for these paradoxes is the inactivation of amygdala, which is supposed to occur during hypnosis⁶²: this fact could at least explain the different emotion logic. At any rate, one will have to know much more not only about hypnosis, but about emotions, consciousness, and the autonomic system in order to be able to disentangle this knot.

Hypnotic Memory and Confabulation

Easier than anywhere else, the pathways of hypnosis and the frontal lobes can be crossed over the field of memory. The acquisition of data has been brought in connection with the left dorsolateral prefrontal cortex and retrosplenial area, while retrieval has been found to activate particularly the right prefrontal cortex and the precuneus⁷². Encoding is thought to involve executive system and the left-frontal control of hippocampus⁷³, while retrieval seems to use slightly different loops within the right prefrontal cortex, depending on whether it was internally or externally specified. When internally controlled, retrieval process predominantly activates the dorsolateral region; when externally cued (by the experimenter, like in hypnosis), ventrolateral activity augments⁷⁴. A broad array of memory deficits has been related to frontal lesions: impairment of free recall, metamemory (knowledge about retrieval strategies), source memory (factual information without the knowledge of the context in which the information was encountered), etc.¹⁵. One possible basis for these defects is an inefficient inhibition (»filtering« or »gating«) of irrelevant information or irrelevant posterior areas. If we accept Gruzelier's suggestion that, what follows the initial frontal activation, is frontal inhibition, then this »inhibition of frontal inhibitory structures« could be compared to the effects of frontal lesions. According to McConkey⁴⁷, three major phenomena regarding memory might be encountered after hypnotic induction: hypermnesia, memory distortion, and pseudomemory (»false memories«)⁷⁵. Hereby, hypermnesia denotes the uncritical delivering of the material encoded in the long-term memory; memory distortions imply mixing of encoded material and the elements of imagination; while pseudomemory consists of the hypnotizer's suggestions which are accepted as the

subject's own memories. Actually, all of those three hypnotic-memory phenomena might be reduced to defective inhibition. Hypermnesia can be viewed as an »unblocking« of memory, a release of stored material without or with significantly lowered moral, social, and biological inhibitions. Memory distortions and pseudomemories, on the other hand, might be explained as a consequence of a deficient filtering of true data versus imagination constructs or external information, respectively, with the inefficient inhibition of the latter ones. It would be difficult to precise whether the inhibition of always the same inhibitory loop is responsible for the appearance of all three hypnotic memory phenomena: more probably, one loop evades moral/social barriers (hypermnesia), a second one filters reality from imagination (memory distortion), while a third one inhibits pseudo-perception (pseudomemory). This would also be in accordance with the scheme proposed by Goldman-Rakic¹, claiming that different prefrontal cortical areas render the same service (inhibition, filtering) to different posterior regions. Let us stress at this point that there is growing evidence^{76–79} against the old position that hypnosis can yield meaningful increase in memory. That the so called »temporal regression« (i.e., conforming to childhood norms) during hypnosis might actually be only topographic regression (i.e., eliciting of more imagistic and affect-laden material), has been proposed by the studies of Michael Nash^{80,81}. Moreover, precisely because of the unfortunate and inextricable blend of really stored data and imaginative or heterosuggestive elements, it becomes clear how dangerous and futile are all too strict confidings in depositions under hypnosis.

What is more interesting for our comparisons is a disturbance known as confabulation, encounterable in certain frontal pathologies. Confabulation means »false statements that are not made to deceive, are typically more coherent than thoughts produced during delirium, and do not reflect underlying psychopathology.«⁸². Amnesia is not sufficient condition for confabulation⁸³ and, on the other hand, not all »frontal syndrome« patients confabulate⁸³. Specific deficits described in confabulating patients consider the recall of autobiographical memories that rivals the detailed report of imagined events, and source monitoring (for instance, the identification of speaker⁸⁴). Obviously, there is a striking similarity between hypnotic memory phenomena and confabulation: hypermnesia as criticism lowering, memory distortion as blending facts with imagination, and pseudomemory as a mess in source attribution, all these elements are to be traced both during hypnosis and in confabulating patients. One would dare to suggest that hypnosis is comparable to a reversible inactivation of the area supplied

by the anterior communicating artery, the pathology of which usually results in confabulation. Woody and Bowers⁸⁵ have, already discussed the idea that hypnotic effects on memory may be likened to the effects of frontal lesions, in a work.

Conclusions

If one takes the prefrontal cortex as a complex system of control/inhibitory loops and accepts that suggestion implies the inhibition of one's own internal »motors,« then it is more than logical to expect that the prefrontal cortex will play the crucial role in hypnotic induction and an obligatory supportive role in all post-induction tasks processing. Hypnotic dissociation has already been suggested to result from partial suppression of frontal functions^{85–87}. This idea brings us to an alternative neuropsychophysiological definition of hypnosis (or suggestion) as the process of external manipulation with frontal-lobe functions with consequent effects upon the entire brain potential of the subject. The normal behavioral paradigm implies parietal preparation for action and a subsequent premotor selection of appropriate motor pattern, which is constantly monitored by the prefrontal cortical inhibitory system. In hypnosis, the prefrontal inhibitory system is inhibited, and the parietal incentive to action is partly replaced by external suggestions that can be transformed into actions without the prefrontal inhibitory monitoring. This new loop is characterized by a significant degree of automaticity⁸⁸. It has been suggested that the crucial phenomena characterizing hypnosis are the lost of insight into the causes of one's own action (similarly to what occurs in schizophrenic patients with a defective »internal monitor«⁸⁹) and/or the weakening of volition⁹⁰. The frontal lobes might be the ideal site to support both phenomena, since both the monitoring of action and volitional control have been related to frontal structures^{39,22}).

Although our definition agrees with the suggestion by Kirsch & Lynn⁹¹ advocating the abandoning of »hypnotic state hypothesis« and favoring theories based on the concept of hierarchical control systems, we are fully aware of its »provisionality and the necessary incompleteness«⁹⁰. Therefore, it would be erroneous to think that this paper tries to »solve« the problem of neurophysiological basis of hypnosis by attributing the location of processes to the frontal lobes: this paper only speculates upon the growing evidence indicating the central role of the prefrontal cortex in triggering the most or even all hypnotic phenomena.

REFERENCES

1. MOUNTCASTLE, V. B., F. PLUM (Eds.): Handbook of Physiology, section 1, The nervous system, vol. 5, Higher functions of the brain, part 1. (American Physiological Society, Bethesda, 1987). — 2. PETRIDES, M., Curr. Opin. Neurobiol., 4 (1994) 207. — 3. BADDELEY, A. D., Nature Rev. Neurosci., 4 (2003) 829. — 4. GOLDMAN-RAKIC, P., J. F. BATES, M.

V. CHAFEE, Curr. Opin. Neurobiol., 2 (1992) 830. — 5. CABEZA, R., J. MANGELS, L. NYBERG, R. HABIB, S. HOULE, A. R. MCINTOSH, E. TULVING, Neuron, 19 (1997) 865. — 6. MCCARTHY, G., A. M. BLA-MIRE, A. PUCE, A. C. NOBRE, G. BLOCH, F. HYDER, P. GOLDMAN-RAKIC, R. G. SHULMAN, P. N. A. S., 91 (1994) 8690. — 7. COURTNEY,

- S. M., L. PETTIT, J. M. MAISOG, L. G. UNGERLEIDER, J. V. HAXBY, Science, 279 (1998) 1347. — 8. SHALLICE, T., Phil. Trans. Roy. Soc. Lond. B., 298 (1982) 199. — 9. DAVIDSON, R. J., G. E. SCHWARTZ, D. SHAPIRO (Eds.): Consciousness and self-regulation, Vol. 4. (Plenum Press, New York, 1986). — 10. BURGESS, P. W., T. SHALLICE, Neuropsychologia, 34 (1996) 263. — 11. BADDELEY, A. D.: Working memory. (Clarendon Press, Oxford, 1986). — 12. CUMMINGS, J. L., Arch. Neurol., 50 (1993) 873. — 13. GRAFMAN, J., K. J. HOLYOAK, F. BOLLER (Eds.): Structure and function of the human prefrontal cortex. (New York Academy of Sciences, New York, 1995). — 14. ADOLPHS, R., T. I. C. S., 3 (1999) 469. — 15. BUSH, G., P. LUU, M. I. POSNER, T. I. C. S., 4 (2000) 215. — 16. DAMASIO, A. R., Phil. Trans. Roy. Soc. Lond., 351 (1996) 1413. — 17. GAZZANIGA, M. (Ed.): The new cognitive neurosciences. (MIT Press, Cambridge, 2000). — 18. FLETCHER, P. C., F. HAPPE, U. FRITH, S. C. BAKER, R. J. DOLAN, R. S. J. FRACKOWIAK, C. FRITH, Cognition, 57 (1995) 109. — 19. GOEL, V., J. GRAFFMAN, N. SADATO, M. HALLETT, Neuroreport, 6 (1995) 1741. — 20. DENNY-BROWN, D., J. Nerv. Ment. Dis., 126 (1958) 9. — 21. LHERMITTE, F., Brain, 106 (1983) 237. — 22. LHERMITTE, F., Ann. Neurol., 19 (1986) 335. — 23. LHERMITTE, F., B. PILLON, M. SERDARU, Ann. Neurol., 19 (1986) 326. — 24. ASSAL, G., Rev. Neurol., 141 (1985) 493. — 25. BRAZZELLI, M., N. COLOMBO, S. DELLA SALA, H. SPINLER, Cortex, 30 (1994) 27. — 26. SHAHANI, B., P. BURROWS, C. W. M. WHITTY, Brain, 93 (1970) 181. — 27. ESLINGER, P. J., G. C. WARNER, L. M. GRATAN, J. D. EASTON, Neurology, 41 (1991) 450. — 28. DEGOS, J. D., N. DA FONSECA, F. GRAY, P. CESARO, Brain, 116 (1993) 1541. — 29. ROLLS, E. T., J. HORNAK, D. WADE, J. MCGRATH, J. Neurol. Neurosurg. Psychiat., 57 (1994) 1518. — 30. GOLDBERG, G., N. H. MAYER, J. U. TOGLIA, Arch. Neurol., 38 (1981) 683. — 31. SHALLICE, T., P. W. BURGESS, F. SCHON, D. M. BAXTER, Brain, 112 (1989) 1587. — 32. CRAIGHERO, L., L. FADIGA, G. RIZZOLATTI, C. UMILTÀ, Visual Cogn., 5 (1998) 109. — 33. MARSHALL, J. C., P. W. HALLIGAN, G. R. FINK, D. T. WADE, R. S. J. FRACKOWIAK, Cognition, 64 (1997) B1. — 34. CARMICHAEL, S. T., J. L. PRICE, J. Comp. Neurol., 363 (1995) 642. — 35. RIZZOLATTI, G., R. CAMARDA, L. FOGASSI, M. GENTILUCCI, G. LUPPINO, M. MATELLI, Exp. Brain Res., 71 (1988) 491. — 36. WOODY, E. Z., H. SZECHTMAN, Int. J. Clin. Exp. Hypn., 51 (2003) 234. — 37. DEECKE, L., H. H. KORNHUBER, W. LANG, M. LANG, H. SCHREIBER, Hum. Neurobiol., 4 (1985) 143. — 38. CHELUNE, G. J., W. FERGUSON, R. KOON, T. O. DICKEY, Child Psychiat. Hum. Develop., 16 (1986) 221. — 39. ABBRUZZESE, M., L. BELLODI, S. FERRI, S. SCARONE, Brain & Cogn., 27 (1995) 202. — 40. DAMASIO, A. R., D. TRANEL, H. DAMASIO, Behav. Brain Res., 41 (1990) 81. — 41. POSNER, M. I., S. E. PETERSEN, Ann. Rev. Neurosci., 13 (1990) 23. — 42. PRIBRAM, K. H., D. MCGUINNESS, Psychol. Rev., 82 (1975) 116. — 43. CRAWFORD, H. J., K. PRIBRAM, P. KUGLER, M. XIE, B. ZHANG, T. KNEBEL, Int. J. Psychophysiol., 14 (1993) 118. — 44. PLOGHAUS, A., L. BECERRA, C. BORRAS, D. BORSOOK, T. I. C. S., 7 (2003) 197. — 45. CRAWFORD, H. J., R. C. GUR, B. SKOLNICK, R. E. GUR, D. BENSON, Int. J. Psychophysiol., 15 (1993) 181. — 46. CRAWFORD, H. J., Int. J. Clin. Exp. Hypn., 42 (1994) 204. — 47. FROMM, E., M. R. NASH (Eds.): Contemporary hypnosis research. (Guilford Press, New York, 1992). — 48. GRUZELIER, J., K. WARREN, Psychol. Med., 23 (1993) 93. — 49. SPIEGEL, D., R. KING, Biol. Psychiat., 31 (1992) 95. — 50. HALAMA, P., Exp. Klin. Hypn., 1 (1989) 19. — 51. GRUZELIER, J., Quart. J. Med., 89 (1996) 313. — 52. WALTER, H., I. PODREKA, M. HAJJI, M. MUSALEK, V. PASSWEG, E. SUESS, M. STEINER, O. M. LESCH, Com. Sci. Psic. Gen., 12 (1994) 41. — 53. WALTER, H.: Hypnose: Theorien, neuropsychologische Korrelate und praktische Hinweise zur Hypnosetherapie. (Thieme Verlag, Stuttgart, 1992). — 54. RAINVILLE, P., R. K. HOFBAUER, T. PAUS, G. H. DUNCAN, M. C. BUSHNELL, D. D. PRICE, J. Cogn. Neurosci., 11 (1999) 110. — 55. AIKINS, D., W. J. RAY, Int. J. Clin. Exp. Hypn., 49 (2001) 320. — 56. KALLIO, S., A. REVEN-SUO, H. HAMALAINEN, J. MARKELA, J. GRUZELIER, Int. J. Clin. Exp. Hypn., 49 (2001) 95. — 57. BURROWS, G. D., R. O. STANLEY, P. B. BLOOM (Eds.): Advances in clinical hypnosis. (Wiley, New York, 1996). — 58. SZECHTMAN, H., E. WOODY, K. S. BOWERS, C. NAHMAS, P. N. A. S., 95 (1998) 1956. — 59. KAISER, J., R. BARKER, C. HAENSCHER, T. BALDEWEG, J. H. GRUZELIER, Int. J. Psychophysiol., 27 (1997) 215. — 60. CRITCHLEY, H. D., C. J. MATHIAS, O. JOSEPHS, J. O'DOHERTY, S. ZANINI, B. K. DEWAR, L. CIPOLOTTI, T. SHALLICE, R. J. DOLAN, Brain, 126 (2003) 2139. — 61. GRUZELIER, J., Contemp. Hypn., 15 (1998) 5. — 62. DE BENEDITTIS, G., V. A. SIRONI, Int. J. Clin. Exp. Hypn., 36 (1988) 96. — 63. GRÜNBERGER, J., L. LINZMEYER, H. WALTER, C. HÖFER, K. GUTIERREZ-LOBOS, H. STÖHR, Wien. Med. Wochenschr., 145 (1995) 646. — 64. FABBRO, F., A. CLARICI, A. MUZUR, A. BAVA, Perc. Mot. Skill., 82 (1996) 571. — 65. STEPHAN, K. M., G. R. FINK, R. E. PASSINGHAM, D. SILBERSWEIG, A. O. CEBALLOS-BAUMANN, C. D. FRITH, R. S. J. FRACKOWIAK, J. Neurophysiol., 73 (1995) 373. — 66. WAXMAN, D., P. C. MISRA, M. GIBSON, M. A. BAHER (Eds.): Modern trends in hypnosis. (Plenum Press, New York, 1985). — 67. GROND, M., G. PAWLIK, H. WALTER, O. M. LESCH, W. D. HEISS, Psychiat. Res. Neuroimaging, 61 (1995) 173. — 68. MLOT, C., Science, 280 (1998) 1005. — 69. GRUZELIER, J., A. CLOW, P. EVANS, I. LAZAR, L. WALKER, L., Mind-body influences on immunity: lateralised control, stress, individual difference predictors and prophylaxis. In: Proceedings. (International Congress of Stress, Budapest, 1997). — 70. OPPENHEIMER, S. M., A. GELB, J. P. GIRVIN, V. C. HACHINSKI, Neurology, 42 (1992) 1727. — 71. HOLROYD, J., Am. J. Clin. Hypn., 46 (2003) 109. — 72. SHALLICE, T., P. FLETCHER, C. D. FRITH, R. GRASBY, R. S. J. FRACKOWIAK, R. J. DOLAN, Nature, 368 (1994) 633. — 73. FLETCHER, P. C., T. SHALLICE, R. J. DOLAN, Brain, 121 (1998) 1239. — 74. FLETCHER, P. C., T. SHALLICE, C. D. FRITH, R. S. J. FRACKOWIAK, R. J. DOLAN, Brain, 121 (1998) 1249. — 75. LOFTUS, E. F., Sci. Am., 277 (1997) 50. — 76. KIHLSSTROM, J. F., Ann. Rev. Psychol., 36 (1985) 385. — 77. MUZUR, A., F. FABBRO, A. CLARICI, S. BRAUN, A. BAVA, Perc. Mot. Skill., 87 (1998) 963. — 78. FRENCH, C. C., J. Consc. Stud., 10 (2003) 153. — 79. NASH, M., Int. J. Clin. Exp. Hypn., 52 (2004) 82. — 80. NASH, M., Psychol. Bull., 102 (1987) 42. — 81. NASH, M., Bull. Menninger Clin., 52 (1988) 383. — 82. PRIGATANO, G. P., D. L. SCHACTER (Eds.): Awareness of deficit after brain injury: clinical and theoretical issues. (Oxford University Press, New York, 1991). — 83. STUSS, D. T., D. F. BENSON: The frontal lobes. (Raven Press, New York, 1986). — 84. JOHNSON, M. K., M. O'CONNOR, J. CANTOR, Brain Cogn., 34 (1997) 189. — 85. LYNN, S. J., J. W. RHUE (Eds.): Dissociation: clinical and theoretical perspectives. (Guilford, New York, 1994). — 86. DIETRICH, A., Consc. Cogn., 12 (2003) 231. — 87. FARVOLDEN, P., E. Z. WOODY, Int. J. Clin. Exp. Hypn., 52 (2004) 3. — 88. KIRSCH, I., S. J. LYNN, Am. J. Clin. Hypn., 40 (1997), 329. — 89. FRITH, C.: The cognitive neuropsychology of schizophrenia. (Erlbaum, Hove, 1992). — 90. WOODY, E., P. SADLER, Psychol. Bull., 123 (1998), 192. — 91. KIRSCH, I., S. J. LYNN, Psychol. Bull., 123 (1998) 198.

A. Muzur

Department of Family Medicine, School of Medicine, University of Rijeka, B. Branchetta 20, 51000 Rijeka, Croatia
e-mail: amirmuzur@yahoo.com

HIPNOZA I MODERNI KONCEPTI ČEONOG REŽNJA – SKICA ZA PREGLED I POZIV K TEMI KOJA OSOBITO OBEĆAVA

S A Ž E T A K

Članak nastoji sažeto prikazati najznačajnije koncepte moderne neuropsihologije čeonih režnjeva i povezati ta otkrića s fenomenologijom koju obično susrećemo u istraživanju i prakticiranju hipnoze. Čeoni režanj istražuje se proteklih godina vrlo intenzivno, a rezultati tih istraživanja, uključujući sindrome opisane pri oštećenjima čeonih režnjeva i u psihijatrijskih bolesnika, otkrivaju zapanjuću podudarnost s pojavama u hipnozi. Temeljeći se na tim sličnostima, u članku se predlaže alternativna neuropsihofiziološka definicija hipnoze odnosno sugestije, koja na ove pojave gleda kao na proces vanjske manipulacije funkcijama čeonog režnja, s posljedičnim učincima na cjelokupni moždani odnosno mentalni potencijal.