Schizophrenia Bulletin vol. 37 no. 1 pp. 1–3, 2011
doi:10.1093/schbul/sbq115
Advance Access publication on October 7, 2010

EDITORIAL

Corollary Discharge, Hallucinations, and Dreaming

Irwin Feinberg*

Department of Psychiatry and Behavioral Sciences, University of California, Davis

*To whom correspondence should be addressed; University of California Davis Sleep Lab, 1712 Picasso Ave, Suite B Davis, CA 95618; tel: 530-752-7216, fax: 530-757-5729, e-mail: ifeinberg@ucdavis.edu

In 1978,[1] a new neuroscience-based model of hallucinations was proposed in this journal. The premise of this model was that hallucinatory experiences are necessarily produced by self-generated neural activity. Their pathology does not lie in the fact that they are self-generated. Much, if not most, of our mental experience results from endogenous or ''spontaneous'' neural activity. Hallucinations are pathological because this self-generated neural activity is perceived as coming from the environment. Consciousness is made up of a stream of internally generated thoughts and images that interact with (or accompany) mental events stimulated by the environment. Within this mix, we readily distinguish the mental events arising from the endogenous neural activity of our brains from the neural activity produced by external sensory stimulation. We can make this distinction even when our brains present us with bizarre or even loathsome thoughts and images. We still recognize that these images or ideas as self-generated, even though they radically depart from what we believe is our ''true'' nature.

These considerations indicate that there must be brain mechanisms that act to distinguish self-generated mental experiences (arising from neural activity initiated by our own brains) from mental experiences stimulated through our sense organs. When this proposition was advanced, it was known that corollary discharge or feed-forward (CDFF) circuits make this distinction at lower levels of the neuraxis, where they distinguish sensory stimulation caused by an organism's motor activity from that originating in the environment. CDFF circuits had been demonstrated in a variety of sensory-motor systems ranging from mammals to invertebrates. The existence of such circuits was postulated in the 19th century by Helmholtz[2]; they were demonstrated experimentally in the 1950s by Sperry[3] and, independently, by von Holst and Mittelstaedt.[4] The basic concept is straightforward: CDFF systems give a ''heads up'' to sensory systems that will be stimulated when the brain of an organism emits motor commands. These signals inform the nervous system that sensory stimulation will be produced by self-initiated motor acts. The messages to the sensory systems are dispatched with the motor commands (hence ''feed-forward'') and typically nullify or otherwise modulate the sensory stimulation produced by the motor activity.

I hypothesized in 1978 that similar circuits operate in the sensory-motor systems of consciousness. Impairment of such systems in schizophrenia could produce hallucinations because neural activity originating in the brain (mind) is attributed to external stimulation. At that time, CDFF mechanisms had been identified only at much lower levels of the nervous system but there were reasons to believe that they should also operate at the level of consciousness. Thus, Hughlings Jackson observed that thinking is simply our most complex motor act. He stated[5] that these ''highest motor centres [of thinking] … can act without producing peripheral reactions on the environment'' (p375). Nevertheless, they would affect the sensory centers (of consciousness) that ''represent the whole organism [i.e. the self] in most complex ways'' (p372). If one defect in schizophrenia causes self-generated conscious events to be experienced as coming from the environment, this could explain hallucinations and other first-rank symptoms of schizophrenia including thought insertion and the general blurring of self-boundaries. Attempts to explain such uncanny experiences could give rise to many types of delusions.

A subsequent paper by Feinberg and Guazzelli[6] developed these ideas further. We noted that obsessive-compulsive patients are often plagued with unceasing intolerable ideas. Nevertheless, obsessive patients recognize these ''unthinkable'' deeply distressing ideas as their own. This demonstrates that even when endogenous neural activity gives rise to involuntary and painful thoughts, obsessive patients recognize these are coming from their own minds. They do not attribute the unbidden and unwelcome thoughts to malign agencies that are inserting thoughts with radio transmitters or infernal devices. Viewed in this light, severe obsessive disorder provides an ''experiment of nature'' that illustrates how CDFF

systems can appropriately identify mental events as self-generated even when they are bizarre, uncontrolled, or even hateful. Guazzelli and I also pointed out that *dreaming is a normally occurring state in which CDFF mechanisms are disabled* (emphasis added), a point made earlier by Feinberg and March.[7] The dreamer regularly experiences visual images and thoughts produced by his sleeping brain as coming from the outside world. The "reality" of these images and ideas is often compelling and can evoke strong emotion. Of course, the similarity of dreams and mental illness has been remarked by observers of human behavior from Aristotle to Jung and Freud. However, the interpretation that the altered conscious experience in both states is produced by an abnormality in a specific kind of brain circuit was new. It may therefore be useful to consider briefly how dream research might advance our knowledge of the neuroscience of psychosis.

Before discussing how the neuroscience of dreaming might be applied to understanding the pathology of the mind in schizophrenia, it is important to acknowledge the creative investigations of Ford, Mathelon, and their coworkers into abnormal CDFF mechanisms in schizophrenia (c.f. Ford et al[8,9]). They have been at the forefront of these challenging studies. However, their work employs perceptual and cognitive tasks below the level of "mind" as conceived by Hughlings Jackson. It may still turn out that experiments at the level of mental experience will be required to test the CDFF hallucinations hypothesis conclusively. One function of CDFF circuits is to serve neural integration. In this regard, "the enormous evolutionary growth of the human cerebral cortex may have posed challenges to integrative mechanisms that were not always successfully met. If so schizophrenia might be a peculiarly human disorder, a disease that is literally *sui generis*."[10(p242)]

In both dreams and psychosis, self-generated images produced by endogenous neural activity are interpreted as coming from the external environment. Guazzelli and I emphasized another similarity that is usually overlooked. This concerns the peculiar way that meaning is often conveyed in both brain states. A single image in a dream sometimes often conveys an immediate emotionally compelling meaning that is not related to the image in any obvious way. Thus, a dreamer might report, "Then my son came into the room and I *knew* he was there to tell he had totaled my car." This knowledge is somehow immanent in the percept but not explained by it. A strikingly similar phenomenon, which is sometimes called an "autochthonous delusion," occurs in schizophrenia. Thus, schizophrenic patients might say, "I saw the way the pens were arranged on your desk and I *knew* that meant I was to be killed." The patient cannot explain the relationship between the pen arrangement and their ominous significance any more than the dreamer can explain how he knows the import of his son's appearance. In both states, compelling ideas are linked to a percept and emerge in consciousness without intermediate steps or connections and without the (postulated) CDFF information that identifies them as the product of the subject's own mind or ego. Of course, there are also important differences between dreams and psychosis. The dreamer is asleep and the hallucinating schizophrenic patient is awake. In addition, the hallucinations of dreaming are predominantly visual, whereas the hallucinations of schizophrenia are usually auditory.

If CDFF brain circuits are temporarily disabled in the normal dreaming brain, sleep research could provide a useful arena for identifying these circuits. Successful identification might shed light on the neural mechanisms of psychosis at the level of "mind" and might even provide new drug targets. Sleep studies could be particularly useful for isolating CDFF circuits because there are phenotypic gradations in the degree to which CDFF circuits are disabled. Near total shutdown occurs when the dreamer is fully involved in and dominated by mental events in a dream that seems unequivocally "real." Partial shutdown occurs in lucid dreaming where the dreamer experiences hallucinatory events but recognizes that he is dreaming and may even be able to control or direct the dream experiences. In addition to hallucinatory dreams, an enormous amount of thought-like mental activity occurs during sleep. Although these thoughts can be strange, the sleeping subject typically experiences them as her own mentation. Studying these different degrees of CDFF impairment during sleep with advanced imaging techniques and sophisticated interviewing methods could help delineate the CDFF circuits of consciousness upon which "agency" depend. Such studies will be challenging, especially because rapid eye movement sleep is not a marker of dreaming.[11] It seems to me a challenge worth taking on, not least because of the esthetic pleasure that would come from successfully verifying the age-old connection between dreams and psychosis.

References

1. Feinberg I. Efference copy and corollary discharge: implications for thinking and its disorders. *Schizophr Bull*. 1978;4: 636–640.
2. von Helmholtz H. In: Southall JPC ed. *Physiological Optics.* 3rd ed Menasha, WI: Banta; Vol. 3 (1925)243–246.
3. Sperry RW. Neural basis of the spontaneous optokinetic response produced by visual inversion. *J Comp Physiol Psychol*. 1950;43:482–489.
4. von Holst VE, Mittelstaedt H. Das reafferenzprinzip. *Naturwissenschaften*. 1950;37:464–476.
5. Jackson JH. *Selected Writings of John Hughlings Jackson.* Vol. 1. New York, NY: Basic Books; 1958.
6. Feinberg I, Guazzelli M. Schizophrenia—a disorder of the corollary discharge systems that integrate the motor systems of thought with the sensory systems of consciousness. *Br J Psychiatry*. 1999;174:196–204.

7. Feinberg I, March JD. Observations on delta homeostasis, the one-stimulus model of NREM-REM alternation and the neurobiologic implications of experimental dream studies. *Behav Brain Res*. 1995;69:97–108.
8. Ford JM, Gray M, Faustman WO, Roach BJ, Mathalon DH. Dissecting corollary discharge dysfunction in schizophrenia. *Psychophysiology*. 2007;44:522–529.
9. Ford JM, Mathalon DH, Heinks T, Kalba S, Faustman WO, Roth WT. Neurophysiological evidence of corollary discharge dysfunction in schizophrenia. *Am J Psychiatry*. 2001; 158:2069–2071.
10. Feinberg I. Schizophrenia as an emergent disorder of late brain maturation. In: Keshavan MS, Murray R, eds. *Neurodevelopmental & Adult Psychopathology*. Cambridge, UK: Cambridge University Press; 1997:237–252.
11. Cavallero C, Foulkes D, Hollifield M, Terry R. Memory sources of REM and NREM dreams. *Sleep*. 1990;13: 449–455.