Available online at www.sciencedirect.com
SCIENCE DIRECT
International Journal of Psychophysiology 58 (2005) 179 – 189
INTERNATIONAL JOURNAL OF PSYCHOPHYSIOLOGY
www.elsevier.com/locate/ijpsycho

Corollary discharge dysfunction in schizophrenia: Can it explain auditory hallucinations?

Judith M. Ford *, Daniel H. Mathalon

Department of Psychiatry, Yale University School of Medicine, New Haven, CT, United States
Psychiatry Service, Veterans Affairs West Haven Health Care System, West Haven, CT, United States

Received 15 November 2004; accepted 20 January 2005
Available online 31 August 2005

Abstract

Failure of corollary discharge, a mechanism for distinguishing self-generated from externally generated percepts, has been posited to underlie certain positive symptoms of schizophrenia, including auditory hallucinations. Although originally described in the visual system, corollary discharge may exist in the auditory system, whereby signals from motor speech commands prepare auditory cortex for self-generated speech. While associated with sensorimotor systems, it might also apply to inner speech or thought, regarded as our most complex motor act. In this paper, we describe the results of a series of studies in which we have shown that: (1) event-related brain potentials (ERPs) can be used to demonstrate the corollary discharge phenomenon during talking, (2) corollary discharge is abnormal in patients with schizophrenia, (3) EEG gamma band coherence between frontal and temporal lobes is greater during talking than listening and is disrupted by distorted feedback during talking in normals, and (4) patients with schizophrenia do not show this pattern for EEG gamma coherence. While these studies have identified ERPs and EEG gamma coherence indices of the efference copy/corollary discharge system and documented abnormalities in these systems in patients with schizophrenia, we have so far had limited success in establishing a relationship between these neurobiologic indicators of corollary discharge abnormality and reports of hallucinations in patients.

Keywords: Schizophrenia; Corollary discharge; N1; EEG coherence

1. Introduction

Auditory hallucinations are a cardinal symptom of schizophrenia, occurring in about 75% of schizophrenic patients (Nayani and David, 1996). They are experienced as voices even though no one is speaking. With hemodynamic and electrophysiological brain imaging, we have the opportunity to understand the neural mechanisms underlying this perplexing symptom. One approach to understanding auditory hallucinations is "symptom capture", a naturalist approach which attempts to image the brain, using electroencephalography (EEG), functional magnetic resonance imaging (fMRI), or positron emission tomography (PET), as patients are experiencing hallucinations. While this approach is conceptually simple, it is extremely difficult in practice because it relies not only on the timely occurrence of an illusive subjective experience but also on the ability of the patient to reliably report its initiation and completion. Symptom capture requires patience from the research team and cooperation and insight from the patient. Nevertheless, a number of investigators have used it successfully, variously reporting that auditory hallucinations are associated with activation of speech production areas (Dierks et al., 1999), primary (Dierks et al., 1999) and secondary auditory cortices, and various polymodal association cortices (Dierks et al., 1999; Shergill et al., 2000; Silbersweig and Stern, 1996).

A more mechanistic approach that does not rely on timing, patience, cooperation and endurance is the "fundamental deficit" approach (see Silbersweig and Stern, 1996). The first step in this approach is to identify a fundamental

* Corresponding author. Department of Psychiatry 116A, VA Connecticut Healthcare System, West Haven, CT 06516, United States.
E-mail address: judith.ford@yale.edu (J.M. Ford).

0167-8760/$ - see front matter © 2005 Elsevier B.V. All rights reserved.
doi:10.1016/j.ijpsycho.2005.01.014

psychological mechanism that when disrupted could cause auditory hallucinations. The second step is to identify the neurobiological process underlying the psychological process, and the third step is to assess the integrity of this neurobiological mechanism. First, following a suggestion of Frith (1987), we identified the self-monitoring deficit as the fundamental dysfunctional psychological mechanism responsible for auditory hallucinations. The underlying assumption is that if voices that come from inside the head (i.e., thoughts) are not identified as self-generated through a failure of self-monitoring, they will be experienced as coming from an external source (i.e., hallucinations). Next, we adopted the proposal of Feinberg (1978) who suggested that self-monitoring deficits in schizophrenia reflect dysfunction of the efference copy/corollary discharge mechanism. Then we sought a neurobiological assay of this efference copy/corollary discharge mechanism. And finally, we attempted to relate abnormalities in this assay to auditory hallucinations.

2. Efference copy/corollary discharge

Von Holst and Mittelstaedt (1950) and Sperry (1950) suggested that motor actions are accompanied by an efference copy of the action which sends a "corollary discharge" signal to sensory cortex, signaling that impending sensations are self-initiated or self-generated. In the visual system, this system may serve to stabilize the visual image during eye movements, maintaining visuo-spatial constancy. In the somatosensory system, it may explain why we cannot tickle ourselves (Blakemore et al., 1998). In its simplest form, the efference copy/corollary discharge mechanism works to suppress perception of events that result from a self-generated action. Thus, in addition to serving as a mechanism for learning and fine-tuning our actions, it may allow an automatic distinction between internally and externally generated percepts.

3. Efference copy/corollary discharge in the auditory system

A similar mechanism may exist in the auditory system: corollary discharges from motor speech producing regions in the frontal lobes prepare the auditory cortex for perceiving resulting speech as self-generated (Creutzfeldt et al., 1989). Support for this mechanism comes from a study (Creutzfeldt et al., 1989) in which recordings were made during a pre-surgical planning procedure from the exposed surface of the right and left temporal cortices while patients talked and listened to others talking. Different populations of neurons in both the superior (STG) and middle temporal gyri (MTG) responded when the patients were hearing their own speech than when hearing the speech of others. About one third of MTG neurons and some STG neurons showed reduced responsiveness to self-produced speech. Another study (Muller-Preuss and Ploog, 1981), this time with monkeys, also described differential responses of STG to self- and other-generated vocalizations. Of particular interest was the suppression of on-going cortical activity during self-vocalization. Further, more than half of the STG neurons were reduced in responsiveness during vocalization.

Although corollary discharge is typically associated with sensorimotor systems, its application to thinking,

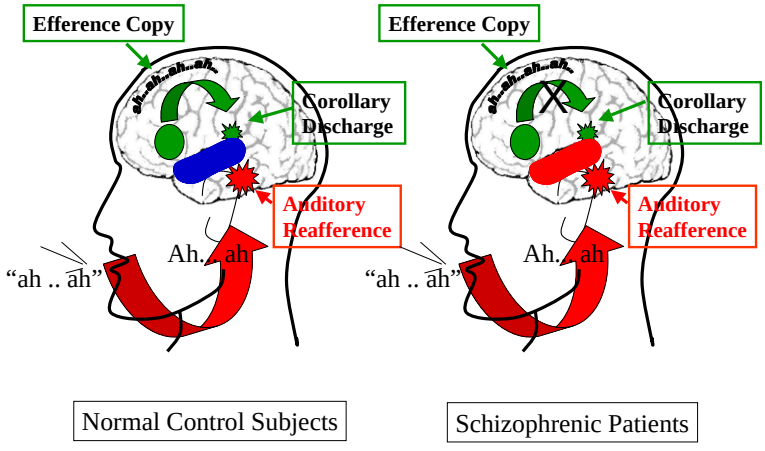

Fig. 1. Two schematics showing normal operation of the hypothesized efference copy/corollary discharge mechanism during talking (left) and its possible dysfunction in schizophrenia (right). The plan to speak originates in the frontal lobes and is shown as a green circle near Broca's area. It sends an efference copy (green ribbon) of the thought or planned sounds to the auditory cortex where it becomes a corollary discharge (green splash). At the same time, or perhaps milliseconds later, talking is initiated and the speech sounds arrive (red ribbon) at auditory cortex as the auditory reafferent (red splash). If the corollary discharge matches the auditory reafferent the sensory experience is cancelled or reduced in its impact. The auditory cortex is colored blue (left) to represent normal suppressed responsiveness to the self-produced sound when it matches the corollary discharge. In the schematic on the right, the schizophrenic patient has an X through the efference copy, and marks the auditory cortex in red to demonstrate that activity of auditory cortex is not suppressed during talking.