SHORT REPORT

Anosognosia: examining the disconnection hypothesis

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Abstract

Objective—To test the hypothesis that anosognosia for hemiparesis results from intrahemispheric disconnection.

Methods—Using right carotid barbiturate injection as a model for anosognosia for hemiparesis, systematic attempts were made to modify deficit awareness by providing the left hemisphere with explicit information regarding left upper extremity function.

Results—Experimental interventions failed to modify deficit awareness in 19 of 32 patients. In those patients who discovered their weakness, attempted movement of the weak limb seems more important than explicit observation of the extremity by the left hemisphere.

Conclusions—The results fail to support Geschwind's disconnection hypothesis for anosognosia for hemiparesis.

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Although many theories have been proposed, no current model accommodates the hemispheric asymmetry found with anosognosia for hemiparesis. One group asserts that premorbid personality¹ or intellectual decline² contribute to the development of anosognosia for hemiparesis. Others emphasise interruption of somatosensory feedback³ or defective feedforward from premotor systems⁴ as a critical component. However, none of these theories adequately explains why anosognosia for hemiparesis more commonly follows injury to the right hemisphere.

Geschwind viewed anosognosia for hemiparesis as a consequence of interhemispheric disconnection.⁵ He proposed that patients might fail to verbally report weakness when injury to the non-dominant hemisphere isolates this hemisphere's motor system from the dominant hemisphere's language centres. Without veridical information from the right brain, the undamaged "eloquent brain" fabricates responses to questions regarding functions subserved by the injured brain. In

Geschwind's opinion, "... many of the patient's responses can only be described as confabulating—that is, they are attempts to fill gaps in the information available to the speech area."

The confabulation predicted by Geschwind's hypothesis might account for "productive" phenomena associated with anosognosia for hemiparesis such as somato-paraphrenic delusions.⁶ Furthermore, the disconnection account might also provide a basis for hemispheric asymmetries of anosognosia for hemiparesis, as the inherently verbal nature of confabulatory responses renders them susceptible to the effect of aphasia. Despite its attractive features, no study has ever directly tested the predictions of the disconnection hypothesis.

If anosognosia for hemiparesis results from interhemispheric disconnection, then directly supplying the left hemisphere with information regarding functions normally processed within the right hemisphere should modify the patient's appreciation of deficit. Such a finding would not only help elucidate the neuropsychological basis for anosognosia for hemiparesis but might also provide a practical adjunct to rehabilitation in patients who lack deficit awareness. Using intracarotid barbiturate injection to induce anosognosia for hemiparesis, we attempted to modify deficit awareness through presentation and manipulation of the paretic extremity in the ipsilateral right hemifield.

Methods: part I

Subjects consisted of patients (n=15) from the University of Florida Epilepsy Surgery Program who underwent intracarotid barbiturate injection during preoperative evaluation. As documented through intracarotid barbiturate injection, all subjects were left hemisphere dominant for language. All patients were right handed by self report and their seizure foci were in the temporal lobe. As part of an earlier study,⁷ patients with anosognosia for hemiparesis were asked to identify their extremities during selective right hemisphere anaesthesia. The subject's vision was restricted to about 15° of central vision by a pair of modified safety goggles. This device prohibited visual contact with

the upper extremities as the patients extended them over their chests during barbiturate infusion. After induction of weakness via administration of methohexital (Brevital), the patient was first asked whether they recognised motor disability. Next, examiners passively moved the paretic limb into the centre of the visual field. To verify visual input into the left hemisphere, the patient was required to verbally identify a number attached to their palms. Patients showing accurate self identification were instructed to move the hand under direct visualisation and were then asked to re-evaluate the hand's motor function.

Results and preliminary comment

None of the 15 patients showed initial awareness of left hemiparesis after right hemispheric inactivation. All patients accurately identified the numbers on their hands and made either partial or no movements while viewing the hand that they had accurately identified as their own. Despite this manoeuvre, eight of 15 patients continued to deny motor deficit on repeated questioning. Another five patients discovered their weakness, denying hemiparesis while the left hand was in the left hemisphere (out of the field of view), then acknowledging weakness only after self observation. The other two patients gave no reply when asked to re-evaluate their strength.

The ability to read numbers on hands that they recognised as their own suggests that the left hemisphere received visual feedback about the function of paretic extremities. However, anosognosia for hemiparesis persisted in over half the patients, providing evidence against the disconnection hypothesis. The lack of response in two patients defies characterisation; that they had earlier offered an (inaccurate) assessment of motor function and then properly followed subsequent commands argues against communication disorder as an explanation. The other five patients acknowledged paresis only after they attempted to move their hand while watching it. An alternative account for anosognosia for hemiparesis is that some subjects remain unaware of paresis because they do not attempt to move. Whereas the performance of these five patients is consistent with the interhemispheric disconnection hypothesis, the methods did not allow determination of whether left hemispheric visualisation or attempted movement was the critical factor facilitating deficit awareness.

Methods: part II

Another group of patients ($n=17$) with intractable epilepsy was recruited for experimental observation during right intracarotid barbiturate injection. All but one were right handed and all but one had a temporal lobe seizure focus. This time, awareness of deficit was assessed in three stages immediately after induction of hemiparesis. Firstly, patients were asked whether they felt weak while paretic extremity dangled on the contralateral side. An initial hand held dynamometer reading quantified the degree of early weakness. Secondly, if anosognosia for hemiparesis was present, the

extremity was passively moved into the centre over the patient's face but below the effective viewing "window" afforded by the goggles. Patients were next instructed to squeeze the examiner's fingers with the paretic hand and then questions were repeated about whether they detected weakness in the extremity. Finally, if anosognosia for hemiparesis persisted, the weak limb was shifted rostrally into the visual aperture while maintaining its position with respect to the body's sagittal plane. Again, patients were asked to identify a number in the palm and squeeze the examiner's fingers, this time while visually monitoring the act, and then to make a final judgment about the hand's motor function. To determine whether patients were still paretic at the time they made their last assessment, strength measurement was repeated with the hand held dynamometer.

Results

Despite repeated interrogation about left hand weakness, anosognosia for hemiparesis persisted through all three stages in 11 of 17 patients. All of these patients showed weakness on the second dynamometry reading, indicating that denial of deficit did not result from recovery of weakness. By contrast, two subjects recognised left hand paresis at stage 1, never showing anosognosia for hemiparesis. The other four patients discovered their deficit after initially denying hemiparesis. In three of four patients, awareness occurred in the second stage after attempting to move the hand without visual observation. Recognition of deficit in the last patient did not occur with attempted movement alone but with combined movement and visual feedback about performance.

General discussion

In both experiments, we intended to provide visual feedback about the function of the left extremity to the unanaesthetised left hemisphere. The design controlled for the possible confounding influence of a visual field defect, a communication impairment, and asomatognosia. None the less, focusing the patient's left hemisphere on the weak left limb failed to alter deficit awareness in most patients (19 of 32) in this series, thereby failing to support the disconnection hypothesis of anosognosia for hemiparesis in these 19 patients. A subset of patients (nine of 32) discovered their hemiparesis after identical experimental manipulation. There were no clear clinical or demographic differences between the group with persistent anosognosia for hemiparesis and the group whose anosognosia improved (table).

Before accepting that the results do not confirm the disconnection account of anosognosia for hemiparesis, some limitations of the experimental design require consideration. One limit pertains to the extent to which any investigator can claim that intracarotid barbiturate injection accurately models the behavioural consequences of brain lesions. For a clinical perspective, experience with numerous patients who had epilepsy surgery at multiple centres

Clinical and demographic information

	Persistent AHP (n = 19)	Deficit discovery (n = 9)	Statistical tests
Age	36.4 (10.4)	32.6 (9.7)	$t=0.95$, $p=0.36$
Gender			
Male	7	4	$\chi^2=0.148$, $p=0.70$
Female	12	5	
Seizure focus			
Left	9	3	$\chi^2=0.87$, $p=0.65$
Right	8	4	
Bilateral	2	2	
Amytal dose (mg)	3.5 (0.8)	4.3 (1.3)	$t=1.65$, $p=0.11$
Full score IQ	77.9 (12.7)	85.7 (12.7)	$t=-1.52$, $p=0.14$

Values in parentheses are SEM.

supports the general validity of the intracarotid barbiturate injection test for language and memory functions.⁸ With regard specifically to anosognosia, the intracarotid barbiturate injection test reproduces features of anosognosia for hemiparesis after brain injury, including a greater frequency of occurrence after right hemispheric anaesthesia.^{9,10} Another limitation of the design of the present study pertains to the use of a device that limits visual feedback. Although it is plausible that induction of deficit without complete visual experience might make patients more susceptible to anosognosia, earlier experiments using the same paradigm found a comparable incidence of anosognosia for hemiparesis even without visual restriction.^{7,9}

As the first experiment failed to distinguish whether hand movement or visual feedback was the critical factor for those who recognised weakness, anosognosia for hemiparesis was assessed in stages during the second experiment. Whereas only four patients developed awareness, sharply limiting additional generalisations, three patients developed deficit awareness merely through attempts at motor activation of the weak limb. This suggests that underactivity of “intentional” or premotor processes in the lesioned hemisphere may contribute to anosognosia for hemiparesis, a finding made previously in a patient with stroke induced anosognosia for hemiparesis.⁴ As attempted movement reduced the anosognosia in either stage 2 or stage 3, but not stage 1, there may be additional, complex interactions between movement related processes, body space and kinesthetic feedback that are inadequately considered by this study.

Using a strict interpretation, we found only one patient whose test results clearly supported the concept of anosognosia for hemiparesis as an interhemispheric disconnection syndrome. Although other authors⁸ have asserted that providing the left hemisphere with information about the weak limbs does not attenuate

anosognosia for hemiparesis, these claims were based on informal clinical experience.¹¹ Bisiach *et al*¹² provide a different argument against disconnection: if anosognosia for hemiparesis resulted from disconnection alone, then patients who verbally deny their deficit should retain the capacity to express deficit awareness non-verbally. To our knowledge, dissociations between verbal and non-verbal report of deficit have not been established. Hence, it is perhaps not surprising that our study found few instances in which disconnection could be entertained as an explanation for anosognosia for hemiparesis.

The present results can be tentatively placed within the context of alternative hypotheses for anosognosia for hemiparesis. For theories that view deficit awareness as dependent on “discovery” of abnormal function,² our findings begin to specify exactly which mental processes must be inadequate to produce the anosognosia. Similarly, the findings are consistent with the “feed-forward” hypothesis of anosognosia for hemiparesis,⁴ an explanation that posits that defective premotor processing results in failure to update an internal comparator about impending movement.

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