# How the Brain Integrates Affective and Propositional Language Into a Unified Behavioral Function

# Hypothesis Based on Clinicoanatomic Evidence

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 Recent publications suggest that the right hemisphere dominates in modulating the affective components of language. Disorders of language from right-sided focal brain lesions have been called "aprosodias" and can be classified in a manner similar to the aphasias. We describe a patient with motor aprosodia who subsequently died and underwent neuropathologic examination. From the neuropathologic findings and recent observations concerning the neurology of depression, we hypothesize that the motor integration of propositional and affective language takes place in the brainstem, whereas their higher-order integration takes place via the callosal connections between Wernicke's area on the left and its homologue on the right. Direct application of these functional and anatomic relations can help clinicians to properly interpret the often incongruous and disparate behavioral and language responses encountered in brain-damaged patients.

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Motor aprosodia is a disorder of affective language consequent to right frontoparietal opercular damage.<sup>1</sup> It is characterized by monotone speech, poor ability to repeat sentences with prosodic-affective variation, and excellent comprehension of the affective components of speech and gesturing; propositional language, a dominant function of the left hemisphere.<sup>2</sup> is intact. The syndrome is one of six recently described disorders of language called the "aprosodias,"1 secondary to focal lesions of the right hemisphere, which provides further evidence to support the hypothesis that the right hemisphere is dominant for modulating the affective components of language.3-5 This dominant language contribution by the right hemisphere seems to be anatomically and functionally organized along the lines of propositional language in the left hemisphere.<sup>1</sup> Depending on the specific combination of affective language disturbances, the disorders of affective language appear to be classifiable in a manner similar to the aphasias. Six varieties of aprosodia, motor, sensory, global, transcortical motor, transcortical sensory, and mixed, have been described and correlated to lesions observed on computed tomography (CT).<sup>1</sup>

In this report, we describe a patient with motor aprosodia who subsequently died and underwent neuropathologic examination. Although his lesion proved to be in an unusual location when compared with those of previously described patients with motor aprosodia,<sup>1</sup> the functional-anatomic correlations in this patient, in conjunction with recent observations concerning the neurology of depression,<sup>6</sup> provide insights into how the brain anatomically integrates the propositional and affective components of language into a unified behavioral function.

### REPORT OF A CASE Clinical Examination

A 47-year-old, right-handed man was transferred to the neurology service at the Dallas Veterans Administration Hospital on Aug 30, 1980, after suffering a sudden left hemiplegia with dysarthria ten days prior to transfer. His medical history was significant only for non-insulin-dependent diabetes mellitus and hypertension, controlled by appropriate medication. There was no history consistent with a previous stroke, transient ischemic attack, or focal neurologic deficit.

Examination on admission disclosed a moderately obese man who was alert, attentive, and fully cooperative. Results of his physical examination were normal. Immediate recall, recent and remote memory, similarities, proverb interpretation, and judgment were intact. He had a flattened affect but denied depression. There

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Fig 1.—Computed tomographic scan sections of patient. Arrows point to infarctions.

was implicit denial of his left hemiplegia in that he was aware of his deficit but thought unrealistically that he would have full recovery in a few days. He was mildly dysarthric but not aphasic. Propositional speech, comprehension, repetition, object naming, writing, and reading were completely normal. He had no difficulty with finger recognition, right-left orientation, or calculations. Constructions were good with no evidence of neglect.

Cranial nerve examination demonstrated full visual fields and normal fundi. Bilateral simultaneous visual or auditory stimuli were perceived normally. There was moderate left-sided facial weakness but no other deficits in cranial nerve functions. Specifically, there was no evidence for pseudobulbar palsy or dysphagia on examination; he could protrude and wiggle his tongue and elevate his palate on request without difficulty. He had a dense left hemiplegia with hyperreflexia and a left-sided Babinski's sign. Results of somatosensory examination were entirely normal, including graphesthesia, two-point discrimination, stereognosis, and bilateral simultaneous stimulation.

#### Evaluation of Affective Components of Language

**Spontaneous Prosody and Gesturing.**—His voice was flat and monotone, lacking the alterations in intonation, pitch, and rhythm that normally color speech with emotions. There was no discernible difference in his voice when discussing his sadness over his illness, his prolonged absence from his family, or his excitement and anticipation about his upcoming weekend pass. There was a marked paucity of spontaneous gesturing and facial animation, which caused the patient to have a flat affect.

**Prosodic-Affective Repetition.**—This was markedly impaired. For instance, when asked to mimic a statement void of emotional words in an angry, tearful, indifferent, or happy voice, the patient was totally unable to repeat the sentence with affective intonation. The only prosodic variation he displayed was minimal lowering or raising of the loudness of his voice at the end of a sentence.

Prosodic-Affective Comprehension.-His



Fig 2.—Drawings of coronal brain slices involved by infarctions. Black areas indicate complete necrosis; hatched areas, demyelination without necrosis. Rostral-caudal sequence is indicated by numbers under each drawing.

prosodic comprehension, however, was completely normal. He was easily able to identify various affective tones, including surprise, anger, disinterest, sadness, or elation, when incorporated into declarative statements that were void of emotional words.

**Comprehension of Emotional Gesturing.**– This was also intact. The patient could identify all the aforementioned emotional tones by name when they were expressed as a pantomime involving face and limbs.

Results of routine laboratory studies, including a lumbar puncture and EEG, were normal. Computed tomography done on Sept 10, 1980, showed an area of low absorption in the deep portion of the corona radiata and rostral internal capsule on the right and a small round lucency in the left mid-internal capsule that were consistent with vascular infarctions (Fig 1).

During hospitalization, repeated testing of affective language revealed a persistent motor aprosodia with no notable change from the initial examination. On Sept 23, 1980, the patient sustained a massive pulmonary embolism that precipitated a cardiopulmonary arrest; he could not be resucitated.

#### METHODS

The brain was removed at autopsy and placed in a 4% formaldehyde solution. Four weeks later the leptomeninges were removed and the brain embedded in a gelatin mixture under negative followed by positive pressure (deLacoste-Utamsing



Fig 3.—Horizontal reconstruction displaying the extent of involvement in right and left internal capsules by infarctions.

et al, unpublished technique). After the gelatin was hardened in a 4% formaldehyde solution for two days, the brain was coronally sectioned at 5-mm intervals on a rotary slicer. The gelatin surrounding each slice was gently removed. The slices were then stained with a trichrome staining procedure (deLacoste-Utamsing et al, unpublished technique). Appropriate sections were also taken for routine histopathologic examination, using hemotoxylin-eosin and Loyez (myelin) stains, which confirmed that the lesions were ischemic infarctions and that their distributions were accurately reflected by the trichrome staining procedure. The stained brain slices were then photographed and the slides back projected onto a glass table. Each brain slice involved by the infarctions was outlined on polyester film tracing paper (Fig 2) so that the extent of the lesions could be accurately reconstructed (Fig 3).

## RESULTS

The right-sided infarction involved the posterior two thirds of the anterior limb, the entire genu, and the anterior third of the posterior limb of the rostral internal capsule (Fig 3). The left-sided lesion was much smaller, partially cystic, and at least two months older than the right-sided lesion by pathologic examination. It involved only the midcentral portion of the rostral genu of the internal capsule (Fig 3). Both lesions were confined to the white matter and located within the internal capsule below the corticocortical transcallosal connections. In terms of clinicopathologic correlations, the right-sided lesion effectively destroyed almost the entire motor output of the right hemisphere with sparing of the sensory radiations," which correlated well with the patient's severe and persistent pure motor (left) hemiplegia. The leftsided lesion was small and clinically

"silent" since there were no rightsided or pseudobulbar findings by examination or symptoms consistent with a previous stroke by history. Therefore, for discussion purposes the left-sided lesion will be ignored although it is conceivable that it could have contributed to the patient's syndrome even though the patient's family agreed that his change in voice ("no life in it") was a direct consequence of the left hemiplegia.

#### COMMENT

If we accept the hypothesis that the left hemisphere is dominant for modulating the propositional components of language and the right hemisphere is dominant for modulating the affective components of language,<sup>1.5</sup> then an important issue to resolve is how the brain anatomically integrates these two parallel and distinct language functions.

Our case would indicate that the actual motor integration of the language outputs from each hemisphere takes place in a subcortical site, below the level of the deep telencephalic nuclei. Were the motor integration of affective and propositional language transcallosal, our patient should not have manifested a motor aprosodia as his right-sided lesion was entirely confined to the internal capsule and did not involve the transcallosal connections. In keeping with current knowledge concerning the organization and termination of the pyramidal and nonpyramidal motor systems,<sup>8.9</sup> the patient's right-sided lesion essentially prevented the entire upper motor neuron output of the right hemisphere from innervating the right and left medial and lateral reticular formation and the right and left motor nuclear groups in the brainstem. Thus, the motor integration of the propositional and affective components of language appears to take place in the brainstem, in the aforementioned areas.

There is evidence, however, that a "higher" order integration takes place between the hemispheres, via the corpus callosum, so that the propositional and affective components are correctly matched prior to their motor integration in the brainstem. Gazzaniga and Sperry<sup>10</sup> published a study concerning language capacities in patients undergoing transection of the cerebral commissures. No significant changes in personality or language behavior were observed. However, they did report that one patient would occasionally display mismatching of emotional behavior to the propositional content of her conversations during

the first two postoperative weeks, thus suggesting that commissural mechanisms do, in fact, exist for unifying propositional and affective language at cortical levels. For instance, when conversing in a normal manner, the patient would suddenly become sad and be on the verge of tears for "no apparent reason." This dissociative type of behavior, where the prosodic-affective components of language are mismatched to propositional content, has also been observed in patients with right-sided brain damage who have sensory and transcortical sensory forms of aprosodia.<sup>1</sup>

In a publication dealing with the neurology of depression, Ross and Rush<sup>6</sup> provide evidence that the synthesis of an appropriate verbal-cognitive set-which depends on the propositional language areas in the left hemisphere to accurately reflect a patient's inner affective state (mood), which in turn is apparently generated by the right hemisphere-requires the posterior-superior temporoposteriorinferior parietal lobes on the right, Wernicke's area on the left, and the callosal connections between both areas to be intact. (The previously mentioned area in the right hemisphere seems to have homologous functions to Wernicke's area as lesions involving this region produce comprehension deficits for the affective components of language.<sup>1</sup>) Thus, whereas proper matching of the affective and propositional components of language appears to be the result of transcallosal interactions between the "sensory" language areas in each hemisphere, the actual motor integration of these components of language appears to be accomplished subcortically, in the brainstem.

Knowledge of the different levels and proposed anatomic pathways for the integration of affective and propositional language is useful to both neurologists and psychiatrists so that when they deal with brain-damaged patients they can properly interpret the patient's affective and propositional language responses since brain lesions may dissociate these functions, causing disparate, confusing, and often incongruous behaviors.<sup>1</sup> A direct clinical application of the issues discussed here can be found in an article by Ross and Rush<sup>6</sup> in which the vexing problems of diagnosing depression in brain-damaged patients are addressed. For instance, a patient with motor aprosodia may be improperly diagnosed as being depressed because of the flat character of his speech and behavior; conversely, if he becomes depressed, the clinician may overlook his propositional language statements concerning dysphoria and suicide since they will be delivered without affective intonation. Some patients with large right-hemisphere lesions may verbally deny all signs and symptoms of depression, including vegetative symptoms, and yet be severely depressed as evidenced by the presence of vegetative signs, irritability, psychomotor retardation, and a positive dexamethasone suppression test, all of which respond to treatment with tricyclic antidepressants. The recent book by Gazzaniga and LeDoux" should also be consulted for further elucidation of these particular issues in split-brain patients.

A final note concerns the anatomic localization of motor aprosodia. As

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indicated in the introduction, previously described patients with motor aprosodia have had right frontoparietal opercular damage,1 whereas our patient's infarction was confined to the internal capsule, destroying almost the entire motor output of the right hemisphere. Although the infarction was subcortical, the cell bodies of the destroyed motor axons resided in cortex; thus, functionally, the lesion can be considered as having я "cortical" component. Whether there are differences between motor aprosodias resulting from frontoparietal opercular damage, which also entails disruption of callosal and intrahemispheric connections, and motor aprosodias resulting from deep subcortical lesions that spare callosal intrahemispheric connections and

cannot be answered at this time. Recent evidence in patients who have aphasia with preserved comprehension from left capsular-putaminal damage, however, has shown that their speech output is grammatically correct although produced with a severe and painfully slow dysarthria.12 This is in contrast to patients with Broca's aphasia secondary to left frontoparietal opercular damage, whose speech output is nonfluent and agrammatical. Whether similar differences will hold true for motor aprosodias from opercular vs diencephalic lesions remains to be determined.

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