Speech rate as a sticky switch: A multiple lesion case analysis of mutism and hyperlalia

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Abstract

Though it has long been known on the basis of clinical associations and serendipitous observation that speech rate is related to mood and psychomotor baseline, it is less known that speech rate is also related to libido and to immune function. We make the case for a bipolar phenomenon of “psychic tonus,” encompassing all these dimensions. The elated, agitated, libidinal, immunofacilitated, and talkative pole is an “approach” disposition primarily activated by the normal left hemisphere—especially, though not exclusively, its frontal lobe. The dejected, lethargic, delibidinized, immunosuppressed, and mute pole is an “avoidance” disposition primarily activated by the normal right hemisphere—especially, though not exclusively, its frontal lobe. In support of this proposed model, we present new evidence, via meta-analysis of previously published single lesion case reports, of a highly significant association between right hemisphere lesions and non-aphasic hyperlalia, and between left hemisphere lesions and non-aphasic mutism. © 2003 Elsevier Inc. All rights reserved.

Keywords: Speech rate; Sticky switch; Hemisphere; Lesion; Single case; Hyperlalia; Hypolalia; Logorrhea; Mutism

1. Introduction

The purposes of this report are: (1) To develop a concept of speech rate as one component of “psychic tonus” on the basis of research into the normal personality, psychopathology, and clinical neurology. (2) To establish speech rate as a bipolar continuum, with each pole being modulated by its own hemisphere of the brain (as are the other components of “psychic tonus”). (3) To blend speech rate into an ecological (i.e., systems) concept of “psychic tonus,” encompassing mood, psychomotor baseline, libido, and immunity as coherent psychophysiological poles of the “approach-avoidance” dialectic, i.e., the eustress–distress dynamic.

1.1. Speech rate as a component of “psychic tonus”

For the purposes of the present investigation, speech rate will refer to the density of conversation, or expressed words, extending from one extreme consisting of complete silence, i.e., refusal to speak (also termed aki-netic mutism, speech adynamia, etc., e.g., reluctance to speak in a person who is known to be able to speak because he or she has done so under extreme pressure or in special circumstances), to the other extreme consisting of incessant chatter, i.e., refusal to be silent or listen to others (hyperlalia or logorrhea). This construct can easily be operationalized as number of words spontaneously expressed per unit of time.

There are two further specifications of the behaviors we are targeting here for analysis, mutism and hyperlalia: (1) we will be concerned here with hypo and hyperlalia in the context of no linguistic impairment such as agrammatism and/or fluent aphasia. Indeed, it is established in neurolinguistics that Broca’s aphasia can be associated with initial mutism or hypolalia (Martins & Ferro, 1993; Ziegler & Ackermann, 1994) as well as dejection (Carota, Rossetti, Karapanayiotides, & Bogousslavsky, 2001). It is also established that fluent aphasia (Wernicke’s aphasia) can be associated with logorrhea (Joanette et al., 1983; Liederman, Kohn, Wolf, & Goodglass, 1983) and even elation (Baruk, 1980). It should be noted however that a prospective study of large groups of Broca’s and Wernicke’s
aphasics found that both groups had equal risk for depression, i.e., 15% (Damecour & Caplan, 1991). These opposite pathologies of speech rate (hypolalia in Broca’s and hyperlalia in Wernicke’s aphasia) could dissociate as a function of lesion site within the left hemisphere. Broca’s aphasia is most commonly associated with left frontal lesions (Taubner, Raymer, & Heilman, 1999) and Wernicke’s aphasia with left temporal lesions (Hillis et al., 2001). The present investigation will focus on mutism and logorrhea not associated with agrammatism or aphasic fluency, respectively; (2) the mutism we will be considering henceforth is not any type of mutism. The mutism of an unconscious or paralyzed person is excluded. The type of mutism we will address is a reluctance to speak in a person who is otherwise capable of speech. For such a characterization to be made, it is imperative that the subject’s speech be observed and interpreted in those rare occasions when it is manifested.

Another form of mutism which will not be considered here is psychogenic mutism, of the sort described by Jane Campion in her film “The Piano Lesson.” Though this form could conceivably, in certain cases, overlap with the adynamic mutism we are targeting here (i.e., a particular expression of depression), it can also simply consist of a highly deliberate interpersonal ploy or tactic, completely unrelated to psychic tonus which is a spontaneous uncontrollable, non deliberate state having no tactical value, and providing no secondary gain. Thus, for mutism to be judged adynamic rather than psychogenic, the overall context has to be evaluated. Inversely, one could construe that hypelalia could also be “psychogenic” rather than spontaneous: a deliberate strategy to irritate an interlocutor for example. We are not aware of such a ploy ever having been used or described, but any case remotely evoking such an eventuality would have been excluded from the current review.

Speech rate is dynamically related to other dimensions of human experience such as mood, psychomotor baseline, libido, and even immunity. We tend to clam up when dejected and to chatter when in happy company. And when we clam up dejected, we stay still and feel listless, and the last thing we are interested in is sex, whereas when we are happy we feel energetic, want to be active and can be interested in sex (given appropriate conditions). What is less known to us is that the immune response participates in this bipolar aspect of our existence, that all these bipolar dimensions are part of an ecologically coherent behavioral system (the approach-avoidance dialectic or eustress-distress dynamic), and that each pole of these varying dimensions is modulated primarily by its own brain hemisphere.

1.2. Evidence from research on normal personality traits

Empirical research on individual differences in normal personality has found associations between euphoria, grandiosity, vigor, and talkativeness (Rapoport, 1983). Likewise, associations have been found between dysphoria, dejection, apathy, and untalkativeness (Ellgring & Scherer, 1996; Hart & Payne, 1973). Immune facilitation is also associated with such traits as optimism, vigor, communicativeness, etc. (Littrell, 1996). As would be expected, immune suppression is linked with such personality traits as withdrawal, loneliness, dejection, despair, listlessness, apathy, etc. (Brennan & Charnefetski, 2000)—especially in the context of extreme stress (Reynaert, Libert, & Janne, 2000; Solomon, Segerstrom, Grohr, Kemeny, & Fahey, 1997).

Functional brain imaging confirms right hemisphere activation in negative mood, sexual indifference, and psychomotor lethargy, and left hemisphere activation in positive mood, sexual excitement, and psychomotor agitation (Baxter et al., 1989; Bench, Friston, Brown, Frackowiak, & Dolan, 1993; Blumberg et al., 1999; Dolan et al., 1993; Martinot et al., 1990; Migliorelli et al., 1993; Stoleru et al., 1999; Tiitonen et al., 1994). Similar dissociations are observed with EEG topography (Cohen, Rosen, & Goldstein, 1976; Drake, 1988; Koek et al., 1999; Rosen, Goldstein, Scoles, & Lazarus, 1986; Tucker & Dawson, 1984). Kang et al. (1991) investigated normal women at the extremes of right or left asymmetry of frontal EEG activation. They found that women with extreme right frontal activation had significantly lower levels of natural killer cell activity (>11:1) than did left frontally activated individuals. Neuropsychological profiles have been found to be indicative of left hemisphere dysfunction in psychomotor and affective forms of “low” psychic tonus and of right hemisphere dysfunction in psychomotor and affective forms of “high” psychic tonus (Braun, Archambault, Daigneault, & Larocque, 2000; Pettigrew & Miller, 1998).

1.3. Evidence from psychopathology

Pathologically excessive talkativeness (hyperlalia or logorrhea) is well known to be associated with mania (Goldberg, Garno, Portera, Leon, & Kocsis, 2000) and with pseudomania (i.e., psychomotor agitation without mood disorder) (Barkley, Cunningham, & Karlsson, 1983). It is also related to hypersexuality (high libido, increased sexual activity) (Howell et al., 1987; Ramrakha, Caspi, Dickson, Moffitt, & Paul, 2000). In opposition to this constellation of associations, reduced speech rate (hypolalia, mutism) is associated with depression (Dantchev & Widlocher, 1998; Kivela & Pahkala, 1988; Lewis, 1975) and pseudodepression (psychomotor lethargy without mood disorder) (Marin, 1990). Hypolalia is also related to hyposexuality (low desire, reduced sexual activity) (Howell et al., 1987; Ramrakha et al., 2000). Depression seems to be associated with an increased risk for immunosuppression.
behavioral repertoire in a person who talked prior to the capacity to talk in the presence of an otherwise normal language areas of the brain. There is one lesion site which presents an exception to this: over a hundred cases of akinetic mutism resulting from cerebellar lesions, nearly always vermal medulloblastomas observed in children, have been published (see Catsman-Berrevoets et al., 1999; Janssen et al., 1998; Nagatani, Waga, & Nakagawa, 1991; Pollack, Polinko, Albright, Towbin, & Fitz, 1995; Siffert et al., 2000). Several adult cases of akinetic mutism following cerebellar lesions have also been reported (Kai, Kuratsu, Suginohara, Marubayashi, & Ushio, 1997). The reason for this remarkable effect of cerebellar lesions remains mysterious.

The next most common causes of neurogenic mutism are a bilateral striatal lesion (Kadota, Kondo, & Sato, 1996; Lechevalier et al., 1996; Ure et al., 1998; Ushida, Fukuda, Endo, & Okada, 1998; Wakabayashi et al., 2000) or a bifrontal lesion (Buge et al., 1975; Dandy, 1946; Faris, 1969; Freemont, 1971; Grossearch, Grossearch-Reider, & Korn, 1991; Kurzt, Autret, & Ruchoux, 1988; Nemeth, Hegedus, & Molnar, 1988; Nielsen & Jacobs, 1951; Poppen, 1939) or a bicingulate lesion (Barris & Schuman, 1953; Buge et al., 1975; Catsman-Berrevoets, van Dongen, & Kros, 1999; Janssen et al., 1998; Nagatani, Waga, & Nakagawa, 1991; Pollack, Polinko, Albright, Towbin, & Fitz, 1995; Siffert et al., 2000). Several adult cases of akinetic mutism following cerebellar lesions have also been reported (Kai, Kuratsu, Suginohara, Marubayashi, & Ushio, 1997). The reason for this remarkable effect of cerebellar lesions remains mysterious.

We present, in Table 1, all the previously reported cases of mutism caused by a unilateral brain lesion that we could find in the literature—devoid of aphasia. For each case we note the lesion location, age at onset, gender, etiology, co-morbidity, presence or absence of seizures or epileptiform signs in the EEG, and the reference. Though aphasia was an exclusion criterion, mood or psychomotor disorder were not.

1.5. Post-lesion mutism

Mutism consists of a pathological reluctance or incapacity to talk in the presence of an otherwise normal behavioral repertoire in a person who talked prior to the morbid onset. Though it can be hard to determine, presence or absence of aphasia can be determined from limited speech segments during the mute phase or after some degree of recovery. The post-lesion form must be distinguished from the elective which is typically best explained in dynamic terms. In the latter case, a person is motivated to maintain silence in a stressful and unpleasant interpersonal environment. However, post-lesion mutism results not at all from interpersonal stressors but typically from a lesion affecting the drive system in a subcomponent located near the expressive system in a subcomponent located near the expressive...
very unusual case. Verstichel, Cambier, Masson, Masson, and Robine (1994) reported an interesting case of a patient who verbalized extensively and compulsively, but only when required to draw or imitate gestures. The patient manifested no agitation in any behavioral domain. The patient had an extensive hemorrhage of the right temporo-parieto-occipital lobes. This case suggests that post-lesion hyperlalia could be of several sorts, destruction of interhemispheric inhibition (Verstichel’s case) or domain-specific overactivation in other cases. In their multiple lesion case review, Ames, Cummings, Wirshing, Quinn, and Mahler (1994) present 28 adult cases of phrase perseveration (meaningless repetition of sentences) of lesional etiology. All the cases had bilateral frontal atrophy and 27 of the 28 also had some bilateral temporal lobe atrophy as well. These cases would be better characterized as compulsive mumblers than logorrheics per se. Arseni and Danaila (1977) developed an argument to the effect that there exists a post-lesional syndrome opposite to akinetic mutism which they termed logorrhea with hyperkinesis. They presented several cases with or without either of the two main symptoms (see Table 2). Other symptoms which they associate with the logorrhea-hyperkinesis syndrome include “affective charging,” “hyperactive attention,” “hypermnesis,” “sharp critical sense.” They even present one case with logorrhea who was not hyperkinetic, a case of post-traumatic subdural hematoma with dilatation and softening of the left anterior temporal lobe and brain stem compression.

Tables 1 and 2 are neuropsychologically limpid: post-lesion mutism without aphasia, when unilateral, is a matter of left hemisphere lesions (8 cases/10), and post-lesion hyperlalia without aphasia, when unilateral, is a matter of right hemisphere lesions (8 cases/9). Because certain cells of the implementation of our hypothesis of a double dissociation (between speech rate and hemisphere lesioned) did not comprise five replicates, we were compelled to test the double dissociation (lesion side by speech disorder) with the conservative Fisher Exact test. The double dissociation was highly significant ($p < .002$). A small proportion of normal subjects are believed to present atypical hemispheric specialization for expressive language (Marien, Engelborghs, Vignolo, & De Deyn, 2000). Whether spontaneous speech rate would conform to this pattern remains to be investigated, but appears plausible to us. Unfortunately, we could not determine from the published reports cited whether the atypical cases reviewed indeed had atypical specialization for language.

2. Discussion

2.1. Intrahemispheric locus of psychic tonus

Could it be that psychic tonus is not particularly a matter of hemispheric asymmetry, but rather a matter of intrahemispherically organized brain systems? For example, one could make a case for a double dissociation of speech rate by lesion site within the left hemisphere. Aphemic (hypolalic) Broca’s aphasics have predominantly frontal lesions while logorrheic Wernicke’s aphasics have predominantly left temporal lobe lesions. However, this anatomical dissociation (limited to the left hemisphere) does not apply to mood, sexual drive, or immune function, nor to aberration of speech rate in the absence of aphasia. Unilateral lesions most likely to produce dysphoria are located primarily (though the tendency is slight) in the frontal lobes (Robinson, Ku-
Psychic tonus is a dynamic mixture of a diversity of human experiences and behaviors. This “tonus” includes language, motor, emotional, sexual and even immune dimensions, and most of these are reflected in, of course, (and could perhaps be partially “caused” by) intellectual representations. However, we think that the mainspring of psychic tonus, as defined here, must be primarily a matter of appetitiveness or aversiveness of a situation. Psychic tonus may be “cold,” “down,” “blue,” “exhausted,” “dull,” “flat” or “constricted.” On the other hand it may be “hot,” “high,” “pink,” “expansive.” The two hemispheres of the brain exert opposed modulation on this ensemble of “tonuses,” in a coherent manner. A concept which encompasses all of these “tonuses” without surpassing their boundaries, i.e., which delineates a necessary and sufficient definition, is the approach-avoidance disposition. We propose that the two hemispheres play opposed roles in the alarm or “stress” response, particularly in its approach-avoidance aspect. There are situations in which it is important for speech and the sexual response to be inhibited, and these are, of course, situations of great danger (real or perceived or “felt”), i.e., that call for avoidance and flight or inaction. These are situations of “distress,” i.e., which activate the alarm response described by Selye and successors (Rosch, 1999). It is also known that immunosuppression is required, and occurs, in situations of extreme acute stress, with the brain mobilizing all the body’s resources for fight or flight (adrenalin secretion) and energy expenditure (cortisol) (Rosch, 1999).

2.2. Hemispheric modulation of psychic tonus

In short, there seems to be some association between frontal lobe lesions and low psychic tonus and between postero-rolandic cortex lesions and high psychic tonus. However, the hemisphere damaged seems to be more important in modulating psychic tonus than the intrahemispheric locus of the lesion. In other words, though the frontal lobes are important in modulating psychic tonus, psychic tonus derives primarily from the balance of influence of the right and left hemispheres.

Table 2

Previously published cases of non-aphasic hyperlalia or logorrhea subsequent to a unilateral brain lesion

<table>
<thead>
<tr>
<th>Age at onset and sex of patient</th>
<th>Localization of the lesion</th>
<th>Symptomatological considerations</th>
<th>Etiological considerations and complications</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>31 Female</td>
<td>Right temporo-insular</td>
<td>Logorrhea, lability, aggressiveness, persecution complex, and amnesia</td>
<td>Oligoastrocytoma, seizures</td>
<td>Giovagnoli, Strada, Pollo, and Boiardi (1992)</td>
</tr>
<tr>
<td>47 Female</td>
<td>Right temporal</td>
<td>Hyperlalia, hyperphagia, and hypersexuality</td>
<td>Infarct, seizure activity</td>
<td>Monga et al. (1986)</td>
</tr>
<tr>
<td>74 Female</td>
<td>Right temporo-parietal</td>
<td>Logorrhea, apraxia, autotopagnosia, and neglect</td>
<td>Infarct, no mention of EEG or seizures</td>
<td>Verstichel et al. (1994)</td>
</tr>
<tr>
<td>13 Female</td>
<td>Right temporo-parietal</td>
<td>Hyperlalia</td>
<td>Contusions due to head trauma, no mention of EEG or seizures</td>
<td>Rourke, Bakker, Fisk, and Strang (1983)</td>
</tr>
<tr>
<td>69 Female</td>
<td>Right temporo-parieto-occipital</td>
<td>Logorrhea, somatoparaphrenia, neglect, anosodiaphoria, and without hyperkinesis</td>
<td>Infarct, no mention of EEG or seizures</td>
<td>Rode et al. (1992)</td>
</tr>
<tr>
<td>31 Female</td>
<td>Right thalamic, hypothalamic</td>
<td>Logorrhea, hypersexuality, and hyperphagia</td>
<td>Stroke, no mention of EEG or seizures</td>
<td>Miller et al. (1986)</td>
</tr>
<tr>
<td>46 Female</td>
<td>Right acoustic neurinoma and right caudate nucleus</td>
<td>Logorrhea and hyperkinesis</td>
<td>Tumor, diffuse EEG slowing</td>
<td>Arseni and Danaila (1977)</td>
</tr>
<tr>
<td>81 Female</td>
<td>Right thalamus</td>
<td>Logorrhea, hemiballismus, behavioral disinhibition, and euphoria</td>
<td>Infarct, no mention of EEG or seizures</td>
<td>Kulisevsky, Berthier, and Pujol (1993)</td>
</tr>
<tr>
<td>71 Male</td>
<td>Left sub-thalamic nucleus</td>
<td>Logorrhea, right hemiballismus, behavioral disinhibition, and euphoria</td>
<td>Hematoma, no mention of EEG or seizures</td>
<td>Trillet et al. (1995)</td>
</tr>
</tbody>
</table>
associated with “eustress,” i.e., positive stress leading to approach behavior. Such situations (for example comfortable rest and/or sleep) are conducive to immunofacilitation, a time during which it is ecologically advantageous for the body to mobilize large resources (lymphocytes) for combating antigens. Inhibition or enhancement of speech or of the sexual response, viewed in this simple neurodynamic and ecological context (which is, need we recall, only a small part of the overall determination of human sexuality or speech), align themselves according to the human stress response. Hemispheric modulation of the human stress response consists of a dynamic balancing of opposed behavioral toning effects of the two hemispheres. Lesions of one or the other hemisphere thus may grossly pervert the human sex drive or mood or psychomotor baseline or speech rate, in opposed manners, depending on the hemisphere lesioned (see Benson, 1984; Davidson, Jackson, & Kalin, 2000; Heilman, 1997, for compatible neuropsychological models).

2.3. Psychic tonus as a “sticky switch”

Arguing from neurological, experimental psychological, electrophysiological, and functional imaging evidence, Pettigrew and Miller (1998) proposed an abnormal interhemispheric neurochemical balance mechanism observed in endogenous bipolar disorder, reproduced in lesion effects, which they called a “sticky switch.” The model proposes that bipolar patients are susceptible to periods during which neurotransmitter concentrations (noradrenaline and serotonin) become markedly asymmetric in the hemispheres, one asymmetry producing mania and the opposite one producing depression. When the patient enters into one of these asymmetric states, he or she is caught in the “sticky switch.” Pettigrew and Miller (1998) demonstrated that the cognitive profile of bipolar patients leans toward left hemisphere dominance when the patient is manic and right hemisphere dominance when the patient is depressed. EEG topography confirms that a reversal of pathological asymmetry indeed occurs in the bipolar syndrome (Drake, 1988). Even basic brain chemistry, as adduced by MRI-S, has since been shown to depart from normality in the depressive and manic phases of bipolar disorder, in opposed hemispheres for each (Hamakawa, Kato, Shioiri, Inubushi, & Kato, 1999). Furthermore, lesions of the right hemisphere, in several mammalian species including man, result in a massive drop of the biogenic amines noradrenaline and serotonin in that hemisphere—as well as concomitant behavioral agitation. This is far less often observed after left hemisphere lesions (Bryer, Starkstein, & Richardson, 1990; Mayberg, Moran, & Robinson, 1990; Mayberg et al., 1988; Robinson, 1979; Robinson & Starkstein, 1997; Starkstein & Robinson, 1999). We think there must indeed exist a complex chemical mediator, not only of mood, as envisioned by Pettigrew and Miller (1998), but of “psychic tonus” as a whole. Understanding the “sticky switch” presents itself as one of the great remaining challenges of neuropsychiatry, with remarkable implications for treatment of patients. Also, it provides speech scientists in general and speech therapists in particular a window through which to envisage the multifaceted phenomenon of speech rate, as a social, psychomotor, affective, brain based and ecologically meaningful, variable.

2.4. Caveat

The metaphor of the “sticky switch” is based on extreme, rare and highly artificial conditions: manic-depressive psychosis, a small subset of patients with focal lesions, complex brain imaging and electrographic findings in normals, weak associations between behavioral traits, and poorly measured constructs (e.g., mania, depression, mutism without aphasia, etc.). There is counter-evidence for each of the associations made in the present report. However, we contend that for each association postulated the evidence is greater than the counter-evidence. Consider the following two cases in point. The relation between side of lesion and depression is the association (among many reviewed above) which has been most investigated empirically. We are aware of 13 distinct group investigations which found a significant relationship between lesion side and depression indicative of a preponderance of left lesions. We are also aware of eight reports of negative findings, consisting of no association between lesion side and depression. No group study has ever found a predominance or right sided lesions leading to depression. As for the association between mania and right hemisphere lesions, we are aware of seven studies of groups of patients. Every one of these studies reported significantly higher prevalence of right than left hemisphere lesions. In short, the association between hemisphere lesioned and valence of the ensuing mood disorder consists of a moderate variance explained, but is sufficiently coherent in the literature to serve as a basis for model building. The dissociation is not picked up in every investigation, but it is nevertheless more plausible than a null model and far less than the opposite model. The model proposed here, of an ecologically meaningful construct of psychic tonus, with a brain systems infrastructure, is thus essentially a heuristic proposal, i.e., a partial explanation of a subtle ensemble of brain–behavior relations. The model attempts to explain how the brain modulates delicate variations in homeostasy, and thus how these modulations can get stuck in an extreme mode after brain lesions.

Presently, there is only piecemeal evidence to the effect that the psychic tonus construct might apply to other
species. For example, immune effects of right and left cortical lesions clearly dissociate in several mammalian species besides humans (Neveu, Betancour, Barneoud, Vitiello, & Le Moal, 1991; Vlajkovic et al., 1994). Right handed chimpanzees approach novel objects more readily than left handed chimpanzees (Hopkins & Bennett, 1994). Vocalization behavior in certain bird species emanates from the left hemisphere, never the right (Floody & Arnold, 1997). We suspect that our proposed model of psychic tonus should apply to several mammalian species (certain more social ones in which vocalization behavior could serve as an analog for speech rate). However, a great deal of research will be required before the concept of psychic tonus can be fully delineated for subhuman species.

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