

Received: 12.04.2018  
Accepted: 28.06.2018

A – Study Design  
B – Data Collection  
C – Statistical Analysis  
D – Data Interpretation  
E – Manuscript Preparation  
F – Literature Search  
G – Funds Collection

DOI:10.5604/01.3001.0012.2009

# THE 3 JARGONS OF JARGONAPHASIA: SEMANTIC, PHONEMIC AND JARGON

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## SUMMARY

In this paper the authors will discuss the nature of jargonaphasia. Any dictionary definition of the word “jargon” will indicate that once again the medical science of aphasia usurped what was originally a lay term. Some researchers have gone as far as modifying a neologism in adults with that sort of jargon by called recognizable ones “target related” and “non-target related. Even studies of the so-called “semantic paraphasias,” go way beyond what is need to describe any of the three jargons – much less to describe “semantic jargon.” Finally, as we shall see, it is often the case that jargon samples have been elicited from naming tasks, mostly object naming. Nevertheless, it is typical to engage the jargon subjects in tasks that require spontaneous stretches of speech discourse. The complex nature of Jargonaphasia which involve a heterogeneity that will ultimately drive many subcategories of it is discussed. The authors described the 3 jargons of jargonaphasia.

**Key words:** stroke, communication, medical science of aphasia, aphasic jargon

## INTRODUCTION

The title of this contribution is not without importance. It immediately subdivides the “aphasic jargons” or “the jargons” or “Jargonaphasia” into what have generally come to categorize a bizarre kind of speech/language output, most often secondary to stroke, and most often studied in adults with aphasia (Kinsbourne & Warrington 1963; Brown 1981; 1988; 2014). There have been papers with the title “...aphasic jargon,” where the jargon type is “neologistic.” The study is not directed at what we now, somewhat ambiguous still, “Semantic jargon,” nor at “Phonemic jargon,” thereby seeming to conflate “aphasic jargon” with “neologistic jargon” (Kertesz & Benson 1970). Any historical charting of these three jargons will inevitably show that great variations have accrued in terms of nomenclature (Brown 1981). There have even been suggestions that not all “jargon” talk is actually linked with aphasia: lingering speech, confabulation, the jargon in severe dementias, and the like. Any dictionary definition of the word “jargon” will indicate that once again the medical science of aphasia usurped what was originally a lay term. The same confusion with the word “neologism” has caused no little looseness in its definition, and this, too, is likely due to the fact that the word was in the dictionary for all laymen to see. Some researchers have gone as far as modifying a neologism in adults with that sort of jargon by called recognizable ones “target related” and “non-target related (Hillis 2015). This, itself, has caused interpretive headaches, since many writers in the field have claimed that the sine que non of a neologism is its opaque nature. Books have been published on adult language disorders where the index under the word “semantics” takes us far beyond what has been proposed solely for “semantic jargon.” In fact, some will have no term for “semantic jargon” in an index of extraordinary sophistication concerning semantics in adult aphasia (Rochford 1974; Cloutman, Gottesman, Chaudhry i wsp. 2009). Even studies of the so-called “semantic paraphasias,” go way beyond what is need to describe any of the three jargons – much less to describe “semantic jargon.” The term “para+phasia” related to errors where one word is substituted for another and the associative semantics of the target/error pair are sometimes similarity of meaning or close contextual relations, called “contiguity,” meaning closeness or actually abutting one another. Furthermore, there are vast distinctions in the functional/linguistic descriptions of the three jargons, and there are vast distinctions (mostly) in the lesion locations and extentions, and importantly, vast differences in the way the researchers understand the normal language system is structures. The history of semantic errors and fluent paraphasia shows that in the early work intentionally or unwittingly worked off of association psychological models, and as the neurosciences developed, neurologists, anatomists, psychologists, neuropsychologists, psychiatrists, clinical speech/language pathologists, linguists, and computer scientists have crafted a wealth of models, often developed to account for normalcy and then carefully utilized for accounts, descriptions, and explanations of aphasia, psycho-physically or physically. These in turn have been used as well for the jargon aphasias.

Finally, as we shall see, it is often the case that jargon samples have been elicited from naming tasks, mostly object naming. Nevertheless, it is typical to engage the jargon subjects in tasks that require spontaneous stretches of speech discourse.

## **SEMANTIC JARGON**

It is the opinion of the present author that this form of Jargonaphasia is the most complex, involving a heterogeneity that will ultimately drive many subcategories of it. To begin with, this type of jargon is the result of deep seated disruptions to semantic memory, and all that that includes. In its dissociated form, it nevertheless can pass the lexical and phonological level with relative normalcy. This is why so much of the descriptions of semantic jargon, although quite bizarre, utilize words in the language. The jargon here is also fluent, and so it has supposedly been run through the phonological/phonetic levels that are recognizable as words and where the words follow the typical phonological/phonetic constraints we all know of. First of all, many words produced here are bizarrely connected to some target (although remote), but with a good degree of surface grammaticality, since arguments and predicates are usually in their normal frames for nouns and verbs, but where the lexical semantics of predicate/argument structure are at best very bizarre.

Many cases involve what look to be ludic behavior, where it appears that the subject is playing with words, confabulating, or revealing some sort of anosognosia. Many of these bizarre and low frequency words may nevertheless be analyzed as (remote) verbal/semantic paraphasias. Accordingly, the sentences produced may reveal word productions that are almost pathologically of lower frequency. We also see subjects described as having semantic jargon as actually producing clever word substitutions approaching the metaphorical. Indeed several workers have had some degree of suspicion that the speakers have more control of their output than might be imagined. Some have actually claimed that patients with this form of what might be called pathological creativity actually have no aphasia at all – presumably casting the cases as some bizarre form of anosognosia that nevertheless seems fairly convincing that there is at least some degree of conscious control of much of what is said.

Importantly, when we consider the neuroanatomical structures that have been included in the possible lesion spots causing this kind of jargon, we see the enormous possibilities of the functional heterogeneity just described. First off, these jargon patients have in most all cases bi-lateral infero-lateral temporal cortex in T2 (the fusiform gyrus) medially with connections with BA 39 as well as with limbic structures. These last connections are more likely connections with the temporal poles in more anterior temporal cortex in closeness with the hippocampal gyri, and again often bilaterally. Some suggest as well that the right hemisphere involvement in the pathology is what gives rise to the semantically bizarre lexical selections. But, we can also imagine that the errors in the right hemisphere have nothing to do with disallowing some degree of word finding in these cases. Even

other types of anomics, without the characterization of jargon, produce lexical items to naming tasks with what some have labelled Luria's/Pavlov's cortical/pathological state as "paradoxical" in that the word frequencies are strangely reversed with more productions of target words of low frequency as compared to other higher frequency words for the target – object name in this case (Paçhalska, MacQueen, Cielebak 2018).

Adding some recent imaging complexity to this picture are findings that the left (only) inferior temporal lobe (area 21) and the temporal pole region (area 38). Here, lesions to these temporal lobes and specific zones of them, unlike with Wernicke's area, severely affect "semantic memory" with observable hypoperfusion with subjects talking in the scanner. And, the lesions here do not seem to require bilateral involvement but do not give rise to agnosia, they nevertheless cause a severe predicate/argument disconnect, such that one might ride a Tennessee Williams but not a Tennessee Walker, or any of the enormous lexical interrelations of, say, verbs and nouns. One can only wonder how a bit more lesion severity further into the limbic system or additional pathological involvement of the other left temporal lobes – fusiform gyrus (area 37) and Wernicke's (area 22).

Not only with the aphasias, but also with severe Korsakoff syndrome, schizophrenia, and the different dementias such as Alzheimer's disease will ultimately give rise to disrupted functions such as the semantic jargon of aphasics (Marshall 2006). Note also that in the study of all the jargons, we must follow any recovery patterns and the model the researchers use must be understood to fully interpret their descriptions (Kohn, Smith & Alexander 1996). Some models, such as microgenesis, work from the depths of the lower structures we have implicated for semantic jargon, but proceed functionally and anatomically to higher structures and more punctate linguistic function until actually uttered words are produced (Brown 2014; Paçhalska, MacQueen, Cielebak 2018). Other models work from top down anatomically from higher conceptual, planning and attentional/focus zones of frontal cortex and work their way down to the limbic. Still other models, such as the ones used by Garrett or Levelt have been used, although with some misgivings, to describe the aphasias. Levelt, for example, has a model built on normal language production and its vicissitudes with strict adherence to experimental design. Some of Levelt's colleagues have tried a bit more to square with the aphasias, while Garrett has rather changed over to some basic belief in connectionism. Needless to say, connectionism has made inroads into the descriptions and accounts of the aphasias, but so far only one basic kind of jargon aphasia: neologistic. Descriptive parallels in connectionism and the fMRI studies would be, for example, "deafferentization" with hypoperfusion. We can now turn to the next form of jargonaphasia, referred to as "neologistic jargonaphasia."

## **NEOLOGISTIC JARGON**

Of the three jargonaphasias, "neologistic" jargonaphasia appears to have been subject to the most widely studied. Unlike Semantic and Phonemic jargon, this

second form of jargon is somewhat more tractable and therefore facilitates the closer analyses that we have seen in the past 45 years or so. This has not so much to do with the anatomy assigned to the lesion parameters that when damaged “cause” it (quotes intentional), but to the type of language production models used for its description and explanation. An important account for at least one type of neologism is that it is the result of phonological alterations to a previously (of quite simultaneously) semantic lexical substituted word then being further compromised by sound type errors. Pick’s 1931 book shows this kind of translucency when he discusses complex paraphasia and perseveration for “jargon aphasias,” the ambiguous term that still lives with us today, and which we have tried to sub-categorized in this essay.

The anatomical locations, when narrowed, and when specifically for neologistic jargon are usually: Wernicke’s area, the supramarginal gyrus (SMG) the infero-parietal lobule (plenum temporale, the posterior parietal operculum, the posterior portion of T1, the posterior temporal operculum, the BA 39 (Angular Gyrus) and the underlying white fiber system (arcuate fasciculus, with parts of some other fiber systems as shown by diffuse tensor imaging), more likely than not the posterior part of the tract, since that region importantly subtends the BA 40 area of the SMG. Most obviously, many lesions of the middle cerebral artery will not be limited by mother nature to these and only these areas, and in those cases the clinical/pathology will be more complex, often therefore masking some features say of the “purer” form of neologistic jargon. Most researchers have found much less bi-lateral involvement for this form, but others, too, have indeed noticed some inclusion in the non-dominant hemisphere – often in similar regions – but not necessarily. We recall that semantic jargon has bi-lateral damage almost as a sine que non; this is not the case for neologistic jargon.

In the last 30 years of so, language production models have helped in correlating lesions with language functions – but still in many cases these functions were more specific response types to different aspects of clinical battery testing. Nevertheless, repetition, naming, reading print aloud, and in other cases writing test have revealed much. Nevertheless, spontaneous speech outputs of longer ranges of uninterrupted production have been analyzed, but with the constant caution that with neologism in many instances the glimpsing of a target may not be forthcoming. Many aphasiologists have observed that there is quite often a good degree of sentential control, with functors often in place giving a handle on much of the grammatical patterning. Recently, we have observed a sort of higher modalizing syntax which reflects what the speaker feels about the embedding proposition to come. Accordingly, some fascinating work has been done on this quite old distinction between the syntax of modalizing and the syntax of propositionalizing. The control of the two structures has revealed almost a chunk or a sort of holistic structure with the higher modalizing, while neologistic forms wait for the production of the propositional part. This dissociation in neologistic jargon aphasia is extremely powerful evidence for modal logic vs. propositional logic. To our knowledge, there have been at most two studies with this logical

difference in mind; one by a French psycholinguist and the other by a theoretical semantician – of the MIT ilk.

Recovery patters have been extremely useful in neologistic jargon, and have often provided evidence – although never pristine – of one or another “theory” of how neologism could be produced. For instance, it is widely known that initially, neologisms were felt to stem from what might be and has been referred to a severe conduction aphasia, where left-to-right, and right-to-left phoneme transposition may be additive or substitutive, non-place-shift substitutions, and deletions are legion, and where there is much perseveration. Naturally, a left-to-right movement is a post-activation (perseverative), which a right-to-left movement is anticipatory (coming from the future, not the past). Early forms of the BDEA appeared to unwittingly cling to this process for neology, since the score the errors it said that more than 50% errors in some word would render it for scoring as a neologism, thus seeming to have proclaimed that there could be a “target related” neologism (not enough phonemic errors) and a “non-target related” neologism. Some researchers still used this distinction for all neology, but seem to therefore be calling phonemic error continua of complexity are all that is needed to explain the genesis of neologisms. It came to be called “the conduction theory,” since the analogy was to the overarching appreciating of the principle production breakdown in that aphasia category. This, too, will often, demonstrate that many conduction aphasics can produce the enigmatic non-target-related neologism. In both types, there is much perseveration.

The Pick-like possibility of a “two stage error” mechanism for neology suggested that a viable alternative would involve some sort of anomic, and that response Pick suggested was a semantic substitute, which was subsequently altered by phonemic error. This set up a prediction for endstage recovery (Eaton, Marshall & Pring 2011). The conduction theory is not a two stage theory, so recovery from that type of neologism would resolve to correct outputs, or perhaps, this could be the endstage speech output monitoring ability as recently suggested and that was not to be confused with anomia. In fact, this type of reasoning seems to make sense. Two other endstages may appear for an “anomia” theory account. One, the patient would discontinue improving, thereby ending with the semantic lexical error type, without further phonemic disruption. That picture would buttress the 2 stage anomia theory. The further improving a second endstage production would be either an anomia, which we now would need to show that it was not simply that the patient was monitoring production better, or it could also support a conduction theory once recovery was reached. A problem was not early on here, since if the theory is “conduction” why would an endstage by an anomia? No logic here, since the conduction theory is not a two-stage theory, but one where a single phonological mechanism could account for all the neologisms...perhaps the first to disappear would be the non-target related, often called “frank” or some other mechanism such as that of a “semi-random generator” swinging into action where no word is forthcoming.” But, that would entail

the anomia theory. This division has still not garnered 100% acceptance, but the present writer sees no alternative.

Another very significant aspect has been uncovered by Dell, Martin and colleagues in their connectionist cascading studies. Only recently has it been observed and written down that slips-of-the-tongue in non-brain damaged speakers, who are very fortunate, tend to be anticipatory in nature. Many fewer perseverations have now been appreciated for these slips. It is also known that the observance of perseverating in all modalities indicates some pathological involvement. That then would correlated with an “anticipation ratio” developed by Dell and associates, such that with no coexisting impediment, such as speaking too fast, being shown fuzzy pictures, and other “noise” for normal speakers, the anticipation ratio” would lower. Surely severe neologistic jargon will and does reveal much perseveration, and so it would be expected that as recovery took place, that ration would then favor anticipation errors. One other observation, it has been shown that not all neologistic subjects have less perseverative at recovery. That would need also to show that there had been on increase in anticipatory errors, since then the recovered ratio would still show an increase in the ratio.

There is yet another anatomical argument that now comes from magnetic resonance imaging of T1, T2 and especially lobule edges of T2. These are fMRI with measurements of degree of perfusion (roughly “hypo” and “hyper” – “perfusion by weighted imaging). Three patterns of anatomical specificity (correlated with previously purely functional model description) have shown that with typical Wernicke’s aphasics, lesions in BA 22 (fittingly called Wernicke’s area by the coinage of eponymy) in the left superior temporal (T1) lobe. These Wernicke’s subjects had multiple associative semantic lexical paraphasias on the typical Aristotelian coordinates of “similarity” and “contiguity.” Jakobson would have added that the similarity dimension was “paradigmatic,” while the contiguity dimension was “syntagmatic.” Hypoperfusion for these BA 22 lesioned patients was occasionally extended to areas 40 (SMG) and 39 (AG). Very importantly, this set of patients had clearly marked semantic trouble on non-verbal tests of comprehension and also had the phonemic paraphasias (associative) typical of Wernicke’s aphasics with the T1 lesions (here, the hypoperfused zones). It turns out though that second set of imaged patients showed hypoperfusion not in 22 but rather in T2 in area 37. T2 is crucially divided into the fusiform gyrus posteriorly in this lobe, extending even to the anterior zones of the occipital lobe, and also extends to the anterior temporal pole region. This 2<sup>nd</sup> set of T2 hypoperfusion subject had absolutely NO semantic disruption on non-verbal comprehension in terms of word-matching. When naming objects, they did produce semantic paraphasia, but their problems also showed blockage but without phonemic paraphasia. In fact, what was noticed was that they constantly complained that the “knew” the word, but could not retrieve it. Some would have put it earlier that they could not access the “phonological output lexicon.” Recall that Wernicke’s subjects had a great deal of trouble with the “form lexicon,” but the trouble would be a how the phonemic strings were processed for utterance, no doubt due to

the inclusion of fiber track disruptions in arcuate connections (and some others now that we have diffusion tensor imaging), subjacent to the SMG. The fusiform gyrus hypoperfusion put the subjects, as we would expect, in the T.O.T stage. This is quite typical of many neologisms of that form of jargon, whereby investigators have had to go beyond typical “conduction” theories of neology, and suggest other processes that could constitute much of a neologism – such as the metaphor of the random (not so random) generator, and a host of perseverative processing – all of which, of course, had to have some constraint at the output exit so as to assure that the paraphasias, or here, the neologism, abided by the long observed phonotactics of the speaker’s language.

Since the present essay is indeed about the three types of jargon, we must add that these perfusion studies had an important 3<sup>rd</sup> group of subjects with hypoperfusion in regions of T2 in BA 21, anterior to the fusiform gyrus and others with hypoperfusion at the poles of T2, (BA 38). Hypoperfusion in these basal temporal lobe regions affect hippocampal regions and thus compromise more than associative semantics. Rather, the whole slate of memory function is compromised, thereby damaging much of what is understood to be “semantic memory.” Although, these subjects do not show bi-lateral lesions necessarily, have no agnosia, but importantly show breakdowns we mentioned in our section of semantic jargon, whereby the arguments and predicates may be syntactically situated in noun and verb frames, the actually semantic errors lacks the knowledge that puts a predicate, such as “drink” with a verb of drinking and not with verbs of hitting and breaking. These semantic errors are more typical in the semantic jargons, not being unlike what we see in the dementias, in schizophrenia, where many of the semantic errors are bizarre and rather unexpected. We only mentioned these more recent perfusion imaging studies because they may with a degree of anatomical severity result in a semantic jargon. As well, neologistic jargon is also perhaps either a full blown and severe Wernicke’s aphasia, or even with some fusiform gyrus involvement which would only complicate further the semantic paraphasic component.

## **PHONEMIC JARGON**

Originally, this kind of jargon was labeled “undifferentiated.” It is still said to consist of incomprehensible and meaningless concatenations of phonemes, often consisting of certain by chance stereotypical utterances and a good deal of perseveration – obviously at the segmental level, since the segmentation of words is practically impossible; the utterance string is simply too opaque for word recognition. This same opacity prevents much syntactic isolation based upon anything else but the suprasegmental envelopes scaffolded not abnormally across the jargon strings. It has been claimed that a “semblance” of syntax may indeed be gleaned by close acoustic analyses of the Fo patterns over different ranges that appear to be phrases and sentences. No doubt that there remains a great need for further acoustic phonetic analyses of the prosody, which never-



theless appears through the fog of this kind of jargon. In the end, it is generally felt that phonemic jargon is essentially a disconnected phonology from the rest of the speaker's language (Hanlon & Edmondson 1996), or the disconnected phonologies of a bilingual, which as well needs much more analysis by those who research "bilingual aphasia."

If we look at the few acoustic studies of phonemic jargon strings, we do find some interesting prosodic patterning, presumably the patterning of the speaker's language – the bilinguals, notwithstanding. The encapsulated phonology of phonemic jargon has consistently demonstrated that prosody peaks through, on the assumption that normally is driven by higher levels such as the semantics of the given/new information, choice of speech act and its commitments, selection of implicature maxims and other pragmatic elements the underlie Fo patterning and its suprasegmental fluctuations. Unfortunately, prosody has not been the subject of many studies of this jargon form. Some few have been able to uncover patterns however, and the patterns appear to hold to the prosodic constraints for the subject's language: (1) Fo declination throughout a sentential, phrasal or lexical tone group that has been isolated from within larger ranges of phoneme strings, (2) vocalic increased lengthening through the intonational envelope, from initial to medial and to final syllables in the group – with longer vowel durations from beginning to end – normal in most languages, (3) breaks between the tone groups, (4) normal phonetic units through the tone groups, and even (5) ideolectal dialect features of the speaker. A southern accent has been charted in one monolingual patient of a study of a very severe phonemic jargon with monophthongization ("five" to "fahv) vowel breaking (/blch/ -à bíuhch – now to the parodic and less PC, "bee ach") and the lowering/backing of the /ei/ ("bake" to "bike"). There is fertile ground here for more modern acoustic phonetic studies of the interactions between stress, tonic accents, Fo direction, especially at the point of tonic accent, where English has a noticeable pitch rise, referred to as the major pitch change – most often over the new information, which often comes at the latter parts of a sentence envelope, or further front for contrastive stress. Again, this prosodic segmentation is down where recognizable words, phrases and sentences are opaque. Nonetheless, these are important areas for further analyses as part of the frustrating study of an encapsulated phonology fluently uttered with essentially the phoneme set of the speaker's language, the phonotactic patterns as well as normal co-articulatory allophones. It is not perfect, but it is a beginning of the search for language outside the disconnected phonology. It is painfully true that this form of jargon is rare and intriguing in its opacity.

## CONCLUSIONS

The three jargons we have considered in this broad outline have been observed since 1833, and often in non-committal categorizations under the term "jargon," – most often distinguished from confabulation, schizophasia, glossolalia, and endstage progressive dementias, although each has a jargon ring to it. I have

seen few if any studies of the so-called progressive aphasias that would unfold into one or another of these aphasic jargon types. Nevertheless, there is a good deal of paraphasia in all these symptomatologies.

Very crucially, our three jargon types can often be further evaluated as they worsen or recover. The jargon of neologisms has had the most extensive research since there is at the beginning a good deal of transparency vis-à-vis what is left of language and speech. Recovery studies of neologistic jargon are legion, and those studies often provide ultimate answers to what may have been the recondite processing of neologisms in the acute stages of the disease. Recovery studies of neologistic jargonaphasia had their beginnings in the early 70's and much has been uncovered since as to where they come from – in acute and chronic stages. Most studies have found lesions here in Wernicke's area, the arcuate fasciculus, most often the posterior portion, with areas 40 and 39 also involved. Some neologistic subjects have also been found to have bi-lateral involvement, which expectedly may be why we see many of these kinds of jargon patients have more bizarre semantic paraphasias and other sorts of psycho/emotional compromises.

Semantic jargon is often found late in life in dementia, but may follow strokes that damage a combination of Wernicke's area as well as important zones of T2, both posterior and anterior damage in differing degrees. When "semantic memory" is compromised, the paraphasias may be paradoxical, indeed, with normal associative strengths reversed, where by lower frequency connections are enhanced hyponymically so that "furniture" will just as likely or even more so activate "ottoman" before "foot rest" and other sorts of pathological "artistry."

Phonemic jargon is rare and most often follows bi lateral temporal-parietal lesions. And, many bilinguals have been reported in this group for some reason. This, too, needs, much further research as a lesser appreciated area of "bilingual aphasia." I am not aware of much by way of recovery studies of this bizarre and bewildering jargon type. It has been pointed out that this form is much less observed than the other two we have considered. Obviously, a slow unfolding of the otherwise disconnected phonological language here would be intriguing indeed. Were it to actually worsen, one can only wonder what might be the next elements of the encapsulated system to penetrate it.

The last point in my conclusion would be to consider if and to what extent there are continuities – both anatomical and functional – among the jargons. It would appear that there are indeed model specific commitments that suggest continuity. Weaker sorts of models for this would be those of top-down architectures, largely constructed on slips-of-the-tongue, which eventually were shown to also describe aphasic derailments. Stronger models would be those constructed by strict experimental research, with extentions into connectionist architecture. Also in this middle of the road modeling for continua with the jargons are straight forward connectionist models of interactive activation. To this point, none of these models rarely if ever were committed to neuroanatomy and any serious brain extentions indicating jargon continua. Newer results of architectural

brain metabolic studies by fMRI of different sorts, clearly have a psychophysical commitment to hypo and hyper-metabolic observations with normal or compromised functions of different types. For human language, the parallel descriptions of regions of metabolic interest ‘run’ in parallel with the description of the function being performed by subjects in the scanner. Jargon continuities are becoming increasingly possible with the fMRI work, and obviously the continuities are a “mind/brain” concomitance. Finally, the work developed in cognitive microgenesis, at this point in the consideration of the jargons, has the most convincing architecture for “mind-brain” functional-neuroanatomical continua among the Jargonaphasias, to my way of thinking. We are still in need of much more thought on these issues, and there can be no doubt that mind-brain philosophical reasoning must play a crucial role at this perplexing inter-face of the mental and the physical. These complexities must not be eschewed merely because of the belief of some that the chasm will never be crossed – by some miraculous “bridge” or even more astonishingly if the chasm gently crashes into a byzantine dual-aspect monism.

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