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Pragmatic Impairments

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Pragmatic Impairments*

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Abstract
Drawing on two recent books, this article addresses the question: "What, properly speaking, is a pragmatic impairment?" It considers three answers, namely (a) that it involves clinical deficiency in the pragmatics module, (b) that it merely requires reduced performance on pragmatic tasks due to some medical condition, and (c) that pragmatic impairment is a deficit in specifically pragmatic abilities. Though the advantages and disadvantages of the three options are considered, no definitive conclusion is proposed.

Keywords
clinical pragmatics, pragmatic impairment, pragmatics module, pragmatic abilities

1. Introduction

Clinical pragmatics has become a thriving research area of late. Testifying to this is the wealth of results, and the rich controversies, described in two recent books, Michael Perkins' *Pragmatic Impairment* and Louise Cummings' *Clinical*

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Pragmatics. Both serve as excellent introductions to the field: they are exhaustively researched, admirably clear, and survey a wide range of evidence and methods. Both highlight, furthermore, two exciting prospects for philosophers of language and linguists. First, theoretical pragmaticians stand to contribute directly to the diagnosis and treatment of conversational disorders, and thereby to an improved quality of life for patients. Second, drawing an inference from malfunction to "normal" functioning, clinical cases stand to afford a valuable new source of data with respect to traditional issues in philosophical and linguistic pragmatics.

As clinical pragmatics has taken off, a foundational issue has become pressing, namely:

Q: What, properly speaking, is a pragmatic impairment?

This will be my focus in what follows. As will emerge, it is doubtful that we can even come close to answering Q at present. Too few facts are in. Many seeming results are hotly disputed. And, where agreement exists, the findings are complex and do not always coalesce into a coherent overall picture. What's more, as both authors stress, there remains quite a bit of crosstalk in the literature, some of it based on stipulation rather than discovery. Nonetheless, Perkins' and Cummings' books suggest what some of the answers to Q might be, and some of the empirical evidence relevant to deciding among them.

My discussion will proceed as follows. I begin with a "framework" answer to Q. This will also serve as an introduction to pragmatic impairments for readers wholly new to the topic. I then present three competing, more substantive answers to Q: three models (A1–A3) of what makes something a genuine pragmatic impairment. I end by laying out some considerations that bear on the plausibility and implications of the answers, though without offering any definitive conclusion.

Before turning to that, a caveat. What follows is a review of an area of inquiry, not a traditional review of a book (for the latter, see Stainton, 2010). The material is certainly stimulated by insights in Cummings' and Perkins' important books. They jointly serve as my departure point. However, the positions to be presented are not ones that they lay out. More than that, Perkins in particular retains a healthy scepticism towards the very terms of the debate.

2. A "Framework" Answer

A rough and ready answer to Q goes like this. A pragmatic impairment is a clinical difficulty in linguistic social interaction. Spelling this out, first, by

definition, "pragmatic impairment" pertains to natural/spoken language – though there certainly are communicative deficits that do not involve language, and it may be that many of the same causes underlie the linguistic and non-linguistic cases. Second, a pragmatic impairment must result from a medical condition of some kind. Third, pragmatic impairments pertain specifically to the conversational *usage* of language, to linguistic social interaction – they are thus to be contrasted with, say, syntactic or phonological deficits.

Three sub-categories merit highlighting.[1] Pragmatic impairments often involve difficulty understanding others, including in particular their communicative intentions. Patients "miss the point". They have trouble understanding non-literal speech: metaphor, conversational implicature, proverbs, irony, sarcasm. They may confuse jokes with lies. Sometimes the difficulty traces, in particular, to a failure to notice non-linguistic clues such as facial expressions, gestures, and the larger non-linguistic context. Another sub-category involves difficulties in making oneself understood. Patients may fail to obey Grice's (1975) maxims, in particular placing an undue interpretive burden on the hearer. For example, they may use pronouns and demonstratives when their reference either cannot be recovered by the interlocutor, or can only be recovered with difficulty. Or again, they may use unfamiliar neologisms and vague terms in ways that obscure what they intend to convey. The third sub-category of disordered conversational interaction concerns discourse and style. People with pragmatic impairments may have trouble building cohesive and coherent texts: they may drift from one topic to another, violate informal rules of turn-taking, issue confusing tangential remarks, etc. They may fail to initiate conversations, or opt out of them unexpectedly. Their speech may exhibit inappropriate levels of formality: too formal, as in recherché vocabulary and sophisticated syntax over coffee; or not formal enough, as in using "equal status" pronouns ("tu" versus "vous" in French, "vos" versus "usted" in Spanish) when speaking with a social superior. Especially noteworthy problems with discourse and style include atypical prosody, echolalia (repeating back the interlocutor's words and phrases), verbosity, the overuse of formulaic expressions, and underrepresentation of certain classes of words (e.g. propositional attitude verbs).

Continuing with our rough and ready framework, there are cases which pretty clearly count as pragmatic impairments, e.g. (1) through (3). The deficits here are highly specific, paradigmatically pragmatic, and have a clinical cause. Equally, there are cases which clearly do not count, e.g. (4)-(6):

[1] These categories are not mutually exclusive, and others could have been chosen. I taxonomize this way merely to render the array of symptoms easier to understand and recall.

(1) Impaired processing of sarcasm corresponding to a stroke-induced lesion in the left middle and inferior frontal gyri (Cummings, 2009: 95).
(2) Impaired processing of indirect requests following a traumatic injury to the middle and inferior frontal, superior and temporal or supramarginal gyri (Cummings, 2009: 95).
(3) Difficulty with nonliteral language – including especially metaphor, proverbs and conversational implicature – in Autism Spectrum Disorder (Cummings, 2009: 57).
(4) Impaired understanding of non-literal language due to temporary drunkenness.
(5) Difficulty conveying ideas in a second language because of limited knowledge of its grammar and lexicon.
(6) Reduced understanding of speakers from a different culture, because of unshared beliefs and desires.

All of this being granted, a detailed and penetrating account of the nature of pragmatic impairments remains elusive. When there is a pragmatic impairment, what kind of thing is impaired? What is the causal structure of such impairments? Related to this, given only what was said above, there remain a host of "difficulties in linguistic social interaction" whose status as pragmatic impairments remains open to debate:

(7) Inability to recognize speakers' intentions due to paranoid delusions in Schizophrenia (Cummings, 2009: 147).
(8) Difficulty conveying ideas due to a global non-fluent aphasia after a stroke (Cummings, 2009: 91).
(9) Boring and/or inappropriate conversational contributions following a traumatic brain injury (Cummings, 2009: 106).

3. Three Contrasting Models

Let us turn to more substantive accounts. I will highlight three. Let me concede immediately that I will be overstressing the contrasts among them. There definitely are intermediate positions, and important commonalities. I mostly set these aside to make the discussion clearer and more stark.

The first model has it that a pragmatic impairment is a clinical dysfunction in the pragmatics module, typically resulting in troubled linguistic social interaction. A pragmatics module is held to be a causal mechanism, specifically an isolable, discrete computational organ. It is function-specific – as opposed, say to Fodor's (1983) Central System. Such a module would move

from inputs to outputs by means of a specialized algorithm, rather than involving a discursive, general-purpose "inference engine". Goes the idea, it is triggered by stimuli which appear to involve ostensive attempts to manipulate another's mental state, e.g. intending to modify a hearer's belief state by making clear that one intends to do just that. The posited module operates unconsciously and intuitively; automatically and quickly. It either evolved phylogenetically (compare colour vision) or was overlearned ontogenetically (compare reading). Importantly, it is not a peripheral "Fodor-module" but is, instead, taken to be a less encapsulated Central System Module (for more on the what and wherefore of pragmatic modules, see Sperber and Wilson, 2002; for the contrast between peripheral and Central System modules, see Barceló, Eraña and Stainton, 2010).

It will help to bring out the nature of this first answer to Q if we very briefly consider what motivates the postulation of such a module. Exegesis not being important in the present context, I will not pin the opposing view on anyone in particular, but will rest content to say that a very longstanding idea in philosophy of language, in both the Analytic and Continental traditions, is that all hermeneutics is cognitively "of a piece". The psychological processes at work in understanding ordinary talk, it has been presumed, are the very ones at work when scrutinizing biblical texts or interpreting poetic metaphors. It may be added that such interpretation is like scientific theorizing: forming hypotheses about what a text means, and testing them against all available evidence. Put in contemporary cognitive scientific terminology, hermeneutics is a full-blown, non-modular, Central System activity. A fundamental problem with this picture is that, whereas interpreting a sophisticated literary work requires effort and time, understanding, say, a request for coffee is seamless, rapid and easy. Put otherwise, unlike textual exegesis, not to mention scientific inquiry, ordinary linguistic interpretation looks far more like (conceptually rich) perception than like full-on intellectual reflection (that is, ordinary speech comprehension is comparable to face recognition and mental state attribution). Recognizing this, a natural thought is that, psychologically speaking, there are two very different kinds of linguistic interpretation. There is the effortless, automatic, unconscious kind, and the effortful, reflective, conscious kind. Equally natural is the idea that the former is subserved by a module in just the way that perceptual processes generally are.

According to our first answer to Q, then, a pragmatic impairment involves a clinical deficit in this causal mechanism – just as a coronary impairment involves a clinical deficit in the heart. Setting aside details for the sake of brevity, this is very roughly the view of Cummings: "…pragmatics is not a

type of conversational performance, but a rational competence that makes conversation possible" (2009: 245).

Notice that this first answer (A1) postulates properly pragmatic causes. A second answer (A2) rejects the very idea: just as there are funny behaviours, but no "funniness-causes", delicious meals but no "deliciousness-causes", there are pragmatic behaviours but no such thing as properly pragmatic causes. A pragmatic impairment, on this second model, is nothing more than reduced performance on pragmatic tasks, whatever the medical condition underlying said reduction. That is, whereas on the first answer, poor conversational performance is merely *evidence* of a pragmatic impairment, a mere symptom, on the second answer poor performance *constitutes* pragmatic impairment (as long as the cause is some kind of medical condition, e.g. shortness of breath due to asthma, slowed phonological processing due to memory loss, or a stutter). On this view, the "ontology" of, say, syntactic and phonological impairment is very different: these involve a deficit in a specific mental competence, whereas pragmatic impairment does not.

A useful analogy here is the contrast between diseases such as AIDS, on the one hand, and mobility impairments on the other. A person whose symptomatology is identical to a patient with AIDS may nevertheless not have AIDS, if the observable symptoms are not in fact caused by the HIV virus. In contrast, what makes something a mobility impairment is simply the nature of the reduced task performance: such reduction may arise from any clinical cause. Our first answer to Q conceives of pragmatic impairment as disease-like in this way, whereas the second conceives of it as a cluster of clinically induced symptoms.[2]

Before moving on, consider the predictions of each with respect to (1)-(3) and (4)-(6). In the former, lesions to various gyri and ASD yield reduced performance in sarcasm, indirect requests and tropes respectively. Each of these is a medical condition. According to A2, this is sufficient for being a pragmatic impairment. On both views, (4)-(6) do not involve clinical damage, and hence are not pragmatic impairments. What of (7)-(9)? A2 gives a clear cut verdict: there is reduced task performance in social-linguistic interaction, and there are

[2] There are two interesting consequences of this contrast. On A2, there is relatively little doubt that pragmatic impairments exist: its controversial commitments are a distinction between linguistic and non-linguistic behaviours, and between medically-induced malfunction and mere difference. In contrast, A1 opens up as a very live and pressing possibility that there simply are no pragmatic impairments properly so called. Moreover, the contrasting notions of "cure" will tend to differ. On A2, successful treatment requires only remediation of task performance. On A1, however, one naturally thinks of a cure as singling out the cause of the deficit and fixing it.

medical causes. Therefore, these are *ipso facto* pragmatic impairments (Perkins also discusses the surgical removal of parts of the tongue (2007: 52) and cleft palate (2007: 115); on A2, these would be pragmatic impairments as well). A1 leaves the issue open. Whether these really are pragmatic impairments depends upon their etiology: if Schizophrenia, the blood clot and the brain injury impacted upon the pragmatics module, and this in turn gave rise to the symptoms described, then there is a genuine pragmatic impairment, otherwise not. Indeed, though I said above that (1)-(3) "pretty clearly count", according to A1 insofar as these genuinely are pragmatic impairments, this too must be because lesions to various gyri and ASD damage the pragmatics module, which then yields reduced performance in sarcasm, indirect requests and tropes respectively.

The third and final model (A3) pursues a middle ground. When there is a pragmatic impairment, what is impaired are linguistic-social-interactive *abilities*. Crucially, these are properly pragmatic causes. But there is no single cognitive mechanism dedicated to pragmatics, and no commitment to one shared neurocognitive substrate. Instead of one pragmatics module, various abilities emerge from the interaction of more basic mechanisms (I note in passing that there are two fundamental differences here: A1 introduces just one causal mechanism, whereas A3 posits numerous ones; and A1, given the language of "modules" and "mechanisms", tends to bring in its wake a commitment to a single neurocognitive substrate, whereas A3 does not. These commitments are separable, and separating them would yield one of the intermediate positions mentioned earlier). Not by accident, the third model also sounds a bit like the second: it likewise treats pragmatic impairments as emerging from *interactions*; however, A2 took them to be merely interaction *effects*.

Model three seems to promise the best of both worlds. It is for this reason that I introduce it. One main difficulty, however, is how sketchy it remains. In particular, as I will stress below, it is hard to know how to individuate pragmatic abilities. To give a better idea of the main thrust of the proposal, however, let me mention some sample abilities, focussing on linguistic comprehension. They might include: identifying a stimulus as communicative; detecting eye gaze in particular and establishing joint attention in general; recognizing a linguistic expression as context sensitive; correctly assigning illocutionary force to a speech act; realizing when a speech act, if taken wholly literally, would be uncooperative. The analogy in this instance is hearing impairment. There are a number of higher-order, multiply realizable, specifically auditory abilities: to detect frequency, pitch and amplitude; to identify the directional origin of sounds; to dampen background noise, etc. A hearing impairment may be understood as a clinical defect in any one of these causes of successful hearing.

A final word about the contrasting models. Both A2 and A3 are inspired by Perkins' *Pragmatic Impairment*. He writes, for instance:

> Pragmatics is defined as the emergent consequence of interaction between cognitive, semiotic and sensorimotor systems within, and between, communicating individuals. In accounting for pragmatic ability and disability, the burden of explanation thus shifts (…) to the constitutive elements and interactions from which it emerges. (2007: 5; See also §4.4.1)

Perkins himself, however, does not endorse either answer to Q. More than that, he is wary of the terms in which I have couched and pursued it: its top-down nature, its search for the theory-independent "essence" of the pragmatic, its locating of pragmatic impairment within the agent herself (as opposed to within an interacting group of interlocutors). Equally, he is suspicious of medical/clinical dysfunctions forming a genuine kind, and of my sharp contrasts among linguistic competence, other semiotic systems, and communicative performance. Thus, revisiting a point from footnote 3, the title of his book notwithstanding, Perkins himself recognizes that, ironically, his work may entail that there is no single thing that constitutes pragmatic impairment: to his mind it may be, to an important degree, an illusory diagnostic category.

4. Evidence and Implications

Our question, recall, was "What, properly speaking, is a pragmatic impairment?". I began by presenting a rough and ready, "framework" answer: a pragmatic impairment is a difficulty in linguistic social interaction resulting from a medical condition of some kind. I then explored three more substantive views:

A1: *Pragmatics Module*: A pragmatic impairment is a clinical dysfunction in the pragmatics module, typically resulting in troubled linguistic social interaction (compare a coronary impairment).

A2: *Not-Specifically-Pragmatic Causes*: A pragmatic impairment is a breakdown in linguistic social interaction, whatever the clinical cause (compare mobility impairments).

A3: *Pragmatic Abilities*: A pragmatic impairment is a clinical dysfunction in one or more specifically pragmatic abilities (compare hearing impairments).

We may sum things up in terms of a simple diagram:

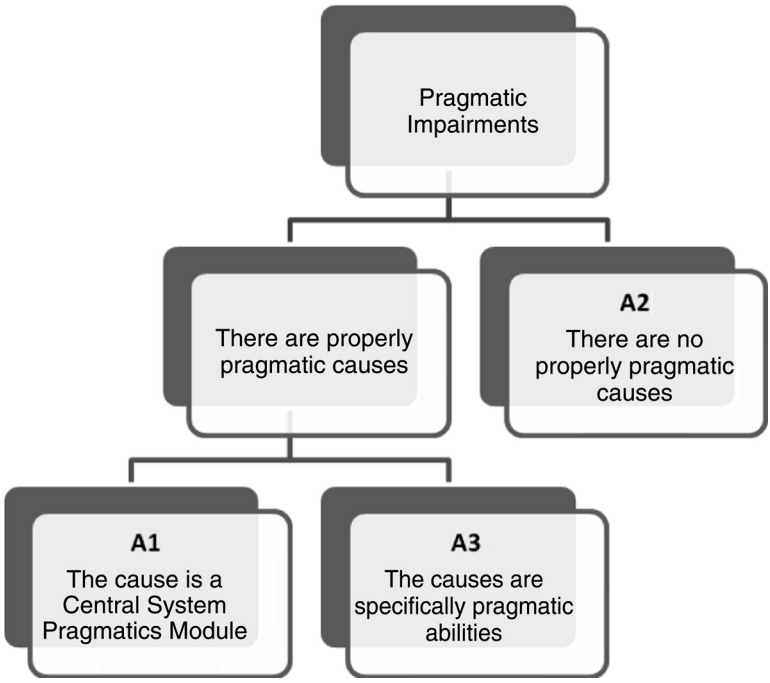

Figure 1. Three views answering Q.

In this final section, I consider the plausibility and implications of each. In doing so, I will address not just the descriptive adequacy of the model (e.g. whether it applies to all the sub-varieties of pragmatic impairments), but also its clinical usefulness.

In terms of "clinical adequacy", A1, were it true, would permit more effective diagnosis and treatment of pragmatic impairments. It would allow us to contrast these with other related but different phenomena, specifically in terms of their distinctive causal structures. Related to this, A1 promises a natural causal explanation of pragmatic impairments. The model also allows one to retain a useful clinical distinction between "primary" and "secondary" pragmatic impairments. In the literature generally, one finds a contrast between reduced functioning that derives directly from a pragmatic deficit, versus reduced functioning which either derives as an indirect consequence of compensating for a pragmatic cause, or derives from some not-properly-pragmatic cause. Importantly, A1 has the conceptual resources to make sense of this contrast. Another point in A1's favour is this: it is clinically useful to describe certain patients as having pragmatic impairments even when their language

use, in casual conversation, has become indistinguishable from the general population. These are cases in which the individual compensates entirely for the deficit, whether by deploying "canned" rules, general purpose intelligence, or what-have-you. A1 can make ready sense of such a diagnosis: though the negative effects are masked, the pragmatics module is not functioning normally, and hence there is (unseen) impairment.

The main challenge to A1 pertains to descriptive adequacy, in particular from the variety of pragmatic impairments. On the one hand, there exists a wide range of syndromes, both developmental and acquired. Development disorders include Autism Spectrum Disorder, emotional-behavioural disorders (such as ADHD, conduct disorder and selective mutism), and various forms of mental retardation (such as Down's Syndrome, Fetal Alcohol Syndrome, Prader-Willi Syndrome, and Williams Syndrome). Disorders acquired in adulthood include Alzheimer's, brain lesions due to stroke, whether in the right or left hemisphere, Schizophrenia, and traumatic brain injury. A1 requires that the same mechanism, namely the pragmatics module, be compromised in all of these. On the other hand, as noted in Section 2, there are a host of symptoms to be explained. If A1 is correct, then all the various varieties of pragmatic impairments – e.g. echolalia, inability to spot sarcasm, abnormal prosody, troubles with pronouns – trace to the same "organ". Finally, and too seldom stressed, A1 is committed to a single explanation for difficulties not just in pragmatic comprehension, but also production. These come in at least two varieties: meaning the wrong thing (e.g. stating what is already obvious, or perseverating on a topic), and expressing a meaning inappropriately. But, at present, no positive proposals exist to explain the nature of the module's malfunction in production.

There are, of course, several strategies for defending A1 in the face of all this variety. Most obviously, it might be suggested that there is a feature of the pragmatics module which allows any number of illnesses to attack its functioning, or to be co-morbid with it. Recall the analogy of the heart: any number of diseases can damage it, and thereby reduce its proper functioning. This might succeed, but it carries a very strong empirical commitment.

Another strategy is to individuate the pragmatics module functionally, in terms of the tasks performed. Thus Cummings begins with a list of (what she deems) genuinely pragmatic phenomena. She highlights in particular speech acts, including especially indirect ones; particular and generalized conversational implicature; pragmatic presupposition; deixis and context-sensitive expressions generally; narrative structure, including cohesion/coherence and the given-versus-new contrast; politeness markers; and tropes. Based on these, she abstracts out four core criteria for the genuinely pragmatic: language

use, reasoning/inference, intention to communicate, and "putting oneself in another's shoes". Finally, she urges that the pragmatics module, the competence at work, is whatever mental mechanism realizes these. This strategy strikes me as unfortunate. Any pre-set list of the "truly pragmatic" is likely to be arbitrary and stipulative. What's more, there is no guarantee that the "realizer" will obey counterfactual-supporting laws: described neurologically, that-which-performs-these-functions may prove too disjunctive.

A more promising way to make sure that all pragmatic tasks trace to the pragmatics module is to insist that a task counts as genuinely pragmatic only if it is performed by the pragmatics module. This sounds question begging. It shouldn't. Taking a leaf from Kripke (1980) and Putnam (1975), we may imagine the theorist beginning with a set of pre-theoretical examples of the properly pragmatic; she looks for a natural kind that gives rise to many of them; having found it, she investigates empirically what this entity does and does not do; she may thus discover that some of the seemingly pragmatic tasks aren't pragmatic after all.[3] Compare again AIDS. Initially it was called "Kaposi's Sarcoma and Opportunistic Infections" and "Gay-Related Immune Deficiency" ("Gay Cancer"), because of its consequences and epidemiological prevalence. Later, other symptoms and other affected groups were identified, and a broader diagnosis emerged: Acquired Immune Deficiency Syndrome. Finally, the underlying cause was singled out, namely the HIV virus. At this point, it became perfectly sensible to say, about a patient with most or even all of the observable characteristics of AIDS, that she nevertheless did not really have AIDS.

Let us turn now to the plausibility and implications of A2. Several of A1's strengths point directly to A2's weaknesses. Because pragmatic impairments, on this model, turn out to be interaction effects – indeed, they will typically be *massive* interaction effects – it becomes far harder to explain them. Still less is there likely to be a global explanation of pragmatic impairment – as opposed to explanations of specific pragmatic impairments. Nor does A2 allow us to distinguish primary pragmatic impairments from secondary ones: if there are no "properly pragmatic causes", one cannot distinguish effects which trace directly to pragmatic causes from those which do not (cf. Perkins 2007: 107ff). A2 also lacks the descriptive vocabulary necessary to capture asymptomatic

[3] Put in terms of philosophy of mind, this strategy combines Functionalism with Identity Theory. First, we sort out, in a rough and ready way, the function corresponding to pragmatics. Then we find the actual "realizer". Finally, we identify the properly pragmatic with what *that thing*, in our brain/mind, in fact does.

pragmatic impairments. The other side of the coin, of course, is that A1's weaknesses highlight A2's strengths. In particular, a wide variety of pragmatic impairments is just what one would expect, insofar as there exists an extraordinary range of medical conditions that impairments emerge from.

In some ways, A3 may seem to offer the best of both worlds. *Modulo* some worries to emerge immediately below, the theorist can proffer a causal explanation of pragmatic impairments rather than merely describing them, viz. by appealing to lost or reduced pragmatic abilities. Descriptive adequacy is more easily achieved because there is no need to trace the grand variety of pragmatic impairments to a single source. A case in point, not mentioned above, is owed to Kasher 1991. There appear to be some pragmatic abilities which are language specific. These include mastery of illocutionary force markers, explicitly context-sensitive lexical items, politeness indicators, genres/registers, and conventional implicatures. These inhabit the space between competence and performance in that, though they involve knowledge about the language, that knowledge pertains specifically to use-theoretic contents. Other pragmatic abilities transcend particular languages, including pragmatic presupposition, particularized conversational implicatures, and metaphor. A3 can easily accommodate this important distinction in terms of "decoding" pragmatic abilities, which are language-specific, versus "inferential" pragmatic abilities which are not. Finally, allowing pragmatic causes, A3 permits the distinction between primary and secondary deficits, and can describe asymptomatic pragmatic impairments in terms of tasks which usually employ abilities *w* and *x* being achieved in this case by deploying abilities *y* and *z*.

There are two fundamental problems with A3. First, one must be careful not to multiply pragmatic abilities at will. Doing so runs the risk of rendering them non-explanatory, in the way that Molière's dormitive virtue is. (To be clear, the worry is not that all explanatory power would be lost. Thus, one can explain why someone has abandoned their study of literature by pointing out that she has lost the ability to process non-literal language. But it will not do as an explanation of why a speaker can no longer understand non-literal language to say that he has lost the ability to understand non-literal language!) Related to this, second, the more freely one posits pragmatic abilities, the more A3 threatens to collapse into a terminological variant on A2. Specifically, it cannot be sufficient for "loss of ability" that there be reduced task performance tracing to some kind of clinical cause. What's needed to sustain A3, then, is a principled means of taxonomizing abilities, and deciding whether they are genuinely pragmatic – and this is something we collectively lack. To reinforce the point, recall the "sample abilities" listed in Section 2: Which are stand-alone causally efficacious capacities? Which are truly pragmatic?

Or again, recall (7)-(9). How would a proponent of A3 determine which, if any, is genuinely a pragmatic impairment?

5. Concluding Remarks

My aims in this review article have been modest: to introduce a question about the nature of pragmatic impairments, to survey three answers to it, and to consider, in a preliminary way, the plausibility and implications of each. It has been no part of my aim to definitively adjudicate among them, for reasons noted at the outset. Along the way, I hope to have highlighted the sharply contrasting background philosophical commitments which underlie these answers, and to have brought home why clinical pragmatics has indeed become an exciting area for philosophers of language and linguists.

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