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IS PAIN ASYMBOLIA A DEFICIT OR A SYNDROME? HISTORICAL REFLECTIONS ON AN ONGOING DEBATE

Abstract: Nikola Grahek's influential book *Feeling Pain and Being in Pain* introduced philosophers to the strange phenomenon of pain asymbolia. Subsequent philosophical debate around asymbolia has been partly taxonomic: the deep question is whether it is best understood as a specific neurological deficit or part of a broader syndrome. This paper looks to the history of asymbolia, positioning the origin of the term within broader historical trends. It shows that strange phenomena about pain and motivation have always presented interpretive challenges, and suggests that the current debate mirrors a historical split between German and French traditions. This does not resolve the debate, but does help place it within broader scientific and philosophical contexts.

Keywords: Pain, Pain Asymbolia, History of Science, Neurology

1. An Update on the Past

Nikola Grahek's *Feeling Pain and Being in Pain* (2007) was tremendously influential, not in the least because it introduced the philosophical community to the curious phenomenon of *pain asymbolia*. The term was first introduced in a case study by Schilder and Stengel (1928), writing about a patient who appeared to feel pain yet be curiously indifferent to it. Drawing on this and subsequent cases, Grahek argued convincingly that these patients were not merely insensible to pain, but rather felt pain, appreciated it as such—and yet were entirely unmotivated by it to perform any sort of protective action.

A central claim of Grahek's book is that asymbolia forms one half of a double dissociation between postulated affective and sensory aspects of pain. The idea that pain affect and sensation can doubly dissociate had been proposed in the literature before (Rainville et al 1999), and philosophers had long been captivated by other motivational oddities, such as cases of

patients who, upon receiving a significant dose of morphine, say that they no longer care about their pain (Dennett 1985, Hardcastle 1999). Grahek's lasting influence, however, comes from his philosophically astute, careful discussion of Asymbolia as a case study in favor of the double dissociation claim. Morphine pain in particular was always a tricky case; powerful opiates and introspective accuracy are notoriously hard to combine. Pain asymbolics, by contrast, fit within a well-established literature of dissociation and deficit.

Grahek's analysis has given rise in turn to a substantive debate about how to interpret asymbolics' behavior and what it entails about the nature of pain. Klein (2015a, 2015b) noted that Asymbolics' deficits are not merely limited to pain but appear to cover all manner of bodily protective activities. Asymbolics are also reported to be indifferent to loud noises and oncoming trucks, for example. Klein suggested instead that indifference to pain was only possible as a consequence of a more general lack of care for the body and its integrity. What care for the body might mean has in turn been the subject of further debate (de Vignemont 2015, Klein 2017).

The goal of the present paper is to contextualize these debates by showing that, in an important sense, they were going on even at the time of Schilder and Stengel. Much of this story can be told in part because of the increasing availability of older clinical works through digitization. We offer this as an 'update on the past,' drawing on material that is now more readily available to scholars. Furthermore, as we'll show, Schilder and Stengel were positioning themselves within clinical debates, but these debates are now themselves largely forgotten. Language presents a further barrier: much of the interesting work was done in either French or German, making it less accessible to today's English-dominated philosophical tradition.¹ And Schilder himself was committed to a psychoanalytic reading of phenomena, particularly in his later work; much of his speculation now reads strangely to contemporary scientifically-minded readers. Nevertheless, we think there is an interesting historical story to be told. We do not pretend to have unraveled the whole narrative. But we hope that by telling a part of it, we may cast further light on the complex and interesting work that Grahek brought to our attention.

2. Grahek versus Klein

It will be helpful to start by saying a few words about how Grahek and Klein differ in their explanation of pain asymbolia.

Grahek held that pain is a composite mental state comprising both a sensory component and an affective/motivational component (perhaps along with other elements). On his view, the affective/motivational component of

1 In what follows, unless otherwise noted, translations were done by Google translate with the help of native speakers. Translations from Schilder and Stengel 1928 follow those in Klein (2015a) and were done with the assistance of Mae Liou and Aleks Zarnitsyn.

pain is brought about by the limbic system, and lesions to the posterior insula prevent the integration of the limbic signal with the sensory signal responsible for the sensory component. This is why the main underlying neurological cause of pain asymbolia are lesions to the posterior insula (Berthier et al. 1988). When such lesions occur, the agent's pain possesses only its sensory component, the role of which is to represent something like tissue damage. The pain is thus recognizably pain, but loses its unpleasant character and motivational force.

Klein offers a different story. According to him,

...asymbolics fail to react to pain because they no longer care about the physical integrity of their bodies. More precisely, they have lost the capacity to care about their bodies in whatever way is relevant to pain. They do not care about cuts and burns and scrapes, because they can no longer conceive of why such events are bad. (Klein 2015a, p. 498)

One main motivation for Klein in adopting this analysis is to defend a view according to which pain is not a composite mental state at all, but rather a *sui generis* mental state with imperative content. For instance, someone's pain caused by smashing their big toe on a hard surface is a mental state with the content such as *Do not put weight on your big toe or bump it anymore*. Klein doesn't see pain asymbolia as arising from a change in the nature of someone's pain (as Grahek does), but rather from a change to that person's affective/motivational structures and those structures' handling of imperative signals. This sort of view predicts that pain asymbolia ought to come with more general deficiencies in reacting to immediate threats to one's body. Moreover, on this account, pain asymbolia turns out to look a lot like (and perhaps *be*) an instance of what is known as *depersonalisation syndrome*, which the DSM IV defines as "a feeling of detachment or estrangement from one's self."

At first pass, this is a debate that aims to use a specific condition (pain asymbolia) to make claims about pains and their nature. But more broadly, Grahek's work and the subsequent debate end up an instance of a general pattern found in many discussions about lesions and their consequences. In the traditional neuropsychological picture, it is only *dissociations* of deficits that matter. *Associations* of deficits, such as Klein's lumping together deficits about reaction to pain with more general problems about bodily care, are suspect because they may reflect mere spatial contiguity of functionally distinct brain regions (Shallice 1988). Yet in practice, characterizing a deficit always involves lumping like symptoms together to form syndromes (Clutton et al 2017). A patient who believes that their wife has been replaced by a robot and a patient who believes that their husband has been replaced by an impostor both suffer from Capgras' delusion, despite the difference in first-order content.

Sometimes lumping is trivial: we group together all failures to write dictated nonwords under the heading of phonological agraphia, rather than postulate many specific deficits. Similar lumping is common across a variety of linguistic tasks. Other times the associated deficits are more complex, and the lumping more difficult: witness debates about whether prosopagnosia constitutes a specific deficit in face recognition or a more general deficit in expert object recognition (Farah 2004). The debate between Klein and Grahek has this character: how one groups symptoms ends up affecting whether one sees Asymbolia as informative about pain or about the sense of about bodily integrity more broadly.

Thus, one way of summing up the disagreement as it pertains to pain asymbolia is that Grahek sees pain asymbolia as a very specific *deficit* related to pain only, whereas Klein conceives the kind of insensitivity to pain involved in pain asymbolia as a symptom of a larger psychiatric condition, i.e., as part of a *syndrome* related to lack of bodily care.

We will argue in the rest of the paper that the debate between Grahek and Klein recapitulates moves that were made within two largely autonomous and distinct traditions of psychiatric thinking that originate at the end of the 19th century, one based in Germany, the other in France. We will do so by defending two main claims: (1) researchers that are part of the German tradition, such as Schilder and Stengel, adopted a *deficit*-based approach to pain asymbolia of the type further developed by Grahek; (2) researchers that are part of the French tradition endorsed a *syndrome*-based approach of the kind defended by Klein.

This historically interesting story also has contemporary relevance for disagreements about how common (or uncommon) pain asymbolia really is. On the one hand, there are people who think that asymbolia is rare and that the details have been over-sold (Coninx 2022; Park 2023). On the other hand, there are those who link it to depersonalisation phenomena (Klein 2015a; Gerrans 2020), with the consequence that some asymbolia-like phenomena might be a relatively common *sequela* of a variety of conditions. Again, and as we'll show, this debate is not new. Terms have shifted, and our sense of the clinical space and its consequences has improved, but the present debate echoes a split that goes back almost to the beginning of rigorous clinical study.

3. The German tradition

As a way of introducing the German tradition, let us pause and reflect for a moment about the word 'asymbolia'. It is now a rare word—so much so that an informal check on google scholar suggests that, since Grahek's book, it is almost exclusively associated with pain asymbolia. There are a few exceptions—the odd discussion of 'tactile asymbolia', some historical discussions—but by and large it is a term that appears to have vanished from the regular clinical lexicon.

When Schilder and Stengel (1928) propose the term, however, they propose it as an additional kind of asymbolia, to be “added to the other asymbolias.”² That would suggest that they mean pain asymbolia to be understood as comparable to another set of deficits. And indeed, at the time, the notion of asymbolia had a well-understood place in the study of psychological deficits caused by cortical lesions.

The origin of the term asymbolia appears to be due to Finkelnburg (1870/1979). Finkelnburg was concerned with the variety of symptoms which were then grouped under the term *aphasia*, and which included problems with both production and comprehension of words. The correct taxonomy of aphasias was a major debate in the late 19th century, as it was realized that good theories depended on distinguishing different ways in which language production could go wrong.

Against this background, Finkelnburg noted that in many cases the deficits involved went beyond problems merely with words. In such cases, he writes

It seems much more simple and correct to speak of a disruption of the symbolic function of the brain or to choose the comprehensive term, asymbolia. Thus, asymbolia would be that disturbance of function in which there is a partial or complete loss of the ability to comprehend and express concepts by means of acquired signs. Thus both a deaf and mute human being and an animal (which naturally lack word formation from the outset) can suffer from asymbolia. (Finkelnburg 1870/1979, p. 164)

The failure to connect words (or more broadly, ‘acquired signs’) with meaning was the hallmark of asymbolia. Broadly speaking, simple sensations or words need to be invested with symbolic meaning by higher cognitive processes; ‘asymbolia,’ then, would be a breakdown of that meaning-making process.

As Duffy and Liles note in their translation, Finkelnburg’s proposal influenced a number of his contemporaries—notably, the great neurologists John Hughlings Jackson and Henry Head were introduced to the concept via Kussmals’ book on *Aphasia*, which in turn spoke approvingly of Finkelnburg (1870/1979, p. 166).

Also endorsing Finkelnburg’s distinction was Carl Wernicke, whose name is still associated with Wernicke’s (or receptive) aphasia. Writing just four years after Finkelnburg, Wernicke adopts the term and suggests that “Asymbolia might then be described as a reduction or loss of the visual memory images of an object, or as the loss of a memory image essential for the conceptualization of the object.” (Wernicke 1874/1977, pp. 116–117). In a footnote, Wernicke also suggests that one must of course expand the

2 1928 p. 155 “Wit schlagen für die hier beschriebene Störung den Ausdruck Schmerzsymbolie vor und reihen sie den übrigen Asymbolien an.”

notion of asymbolia beyond the words to deficits in appreciating “essential tactual and visual images.” In other words, Wernicke seemed willing to move beyond ‘asymbolia’ as merely a feature of aphasia, considering it as a more general deficit in attaching meaning to sensory impressions. Wernicke argued that several cases of what he called ‘psychic blindness’ (now known as visual agnosia; see Baumann 2011) should instead be considered as instances of asymbolia, albeit in the visual realm (Wernicke 1906/1977, pp. 256 & 267).

Numerous authors wrote of asymbolias in the decades that followed. One important bit for our story is work done by Sigmund Freud. Freud distinguished two sorts of aphasia: *verbal aphasia*, in which “associations between the single elements of the word concept” are disrupted, and *asymbolic aphasia* “in which the association between word concept and object concepts are disturbed.” (Freud 1891/1953 p. 191). Freud argued for a restriction of ‘asymbolia’ to exclude deficits of object recognition, but maintained with Finkelnburg and Wernicke the general concept of asymbolia for a failure to link word and meaning as a distinct clinical entity. Notably, in his figure 2 of the same work, he tentatively assigns asymbolia to what Geschwind (1965) would later call a disconnection syndromes: that is, to the disturbance of the connection between the ‘optic field’ and the ‘acoustic field’, rather than a specific processing deficit as such.

This fleshed-out notion of ‘asymbolia’ as designating a specific failure to connect meaning to stimuli would have been readily apparent to Schilder and Stengel, and presumably to their readers. Notably, Stengel is responsible for the 1953 English translation of Freud’s *Zur Auffassung der Aphasien*, referenced above. And of course, Hemphill and Stengel (1940), which has been discussed for its rich description of pain asymbolia, is primarily a study of a patient with ‘pure word-deafness’—that is, of aphasia. The introduction of this paper is a comprehensive review of the debates on aphasia. Indeed, Hemphill and Stengel make the tentative suggestion that word-deafness and asymbolia may be different aspects of the same syndrome, both with a “common feature seems to be a lack of the ability to make an appropriate response to stimuli reaching the patient from the outer world.” (1940, p. 260). They immediately go on to suggest that asymbolia for pain might be “the more primitive and more general symptom” (p. 260), with word-deafness a special case.

Finally, the idea that pain asymbolia is the result of a particular kind of disconnection between sensation and meaning reaches its canonical formulation in Geschwind’s seminal discussion of disconnection syndromes (1965; see Mesulam 2015 for a review). We note that Geschwind’s discussion comes as part of a quite general discussion of agnosias and language deficits, many of which he considers as disconnection syndromes. Pain asymbolia is offered as a potential case of disconnection between sensory and limbic systems (1965, p. 269ff). And in a passage that prefigures the later use to which Grahek would put the phenomenon, he notes that

This distinction between pain as a sensation and the emotional response to pain has long figured in the literature of research on pain. Denny-Brown (1962) has stated this distinction well, “. . . for such patients (i.e. those with asymbolia for pain) that we have seen can feel pain and can discuss it, though it is not of any biological importance to them.” (1965, p. 270)

Note, however, that by the time Geschwind writes, the term ‘asymbolia’ appears to be already dropping out of common use in favor of more specific terms; indeed, while one can still find references to asymbolias in the 1960s, pain asymbolia is the only sort that Geschwind himself discusses.

Summing up, we suggest the following story about the origin of ‘pain asymbolia’ as a clinical entity. It arises within a literature heavily influenced by German-language studies of neurology, one that focuses on deficits produced by brain damage. Though not uniform, there is even a broadly localizationist flavor—the literature is full of careful discussion of postmortem examination of lesions, for example, and both Schilder and Geschwind consistently attempt to use their observations to ground functional attributions to brain regions. Pain asymbolia is seen, not uniquely, as a kind of failure to attach the appropriate meaning to sensations. The notion of ‘meaning’ is understood in various ways (Schilder via Freud-inflected considerations of the body image, for example, Geschwind via the limbic system), but the idea that a lesion might disconnect a percept from meaning clearly harkens back to older discussions of asymbolias in aphasia and associated agnosias. Indeed, the link is tight enough that discussions like Hemphill and Stengel’s take seriously the possibility that aphasias and pain asymbolia may be aspects of the same phenomenon. And this is, of course, the picture inherited by later discussions which cite Schilder and Stengel, culminating in the picture presented by Grahek.

The general outlines of a deficit-based approach are familiar and compelling to modern ears, even if the details may sound rusty. It may come as some surprise, therefore, to learn that it didn’t need to develop that way. For that story we must turn to a less well-known, but equally interesting, French tradition.

4. The French tradition

The French neurologist Jules Cotard is today associated with an eponymous monothematic delusion, the core of which is the belief that one is no longer alive. Yet Cotard’s original report (1880/1999) on what he came to call *le délire des négations* presents a considerably richer and more complex clinical picture. The patients he discusses are affected with a form of ‘anxious melancholia’ that gives rise to a variety of ‘hypochondriacal delusions’ surrounding the internal experience of ‘abnormal bodily sensations’³ known to be common in those with severe melancholy.

3 See, p. 278 in the translation. “*sensations malades*” in the original.

The patients he describes have a variety of beliefs about their bodies. Some believe they have no organs. Others believe that they are effectively immortal, or that they remain alive despite the rotting of internal organs. His own patient, Miss X, believed that the state of her own body was such that she could not die a natural death but rather must be burned alive, and attempted to end her own life several times.

As Berrios and Luque (1995a; Cotard, Berrios, and Luque 1999) have discussed in their thorough history, Cotard's original picture was not one of a simple, monothematic delusion. They note that the French *délire* is not readily translated by the English 'delusion' or 'delirium', writing that:

These terms only manage to convey fragments of its French meaning. *Délire* is not a state of delirium or organic confusion (in French, *délire aigu* and *confusion mentale*) or a delusion (in French, *idée* or *thème délirante* [sic])—it is more like a syndrome that may include symptoms from the intellectual, emotional, or volitional spheres. Hence, translating *délire des négations* as nihilistic delusion gives the wrong impression (caused by the intellectualistic semantics attached to the term delusion in English) that it exclusively refers to a thought. As clearly described in his ... report, Cotard never meant it to be a thought, but instead a symptom cluster. So, to talk about the delusion of being dead as Cotard's delusion makes little sense, for *délire des négations* also entails the presence of anxiety, severe depression, and other attending delusions. (1995a, p. 219)

Cotard's *délire des négations* seems as if it were familiar enough to French psychologists. By 1893 Régis referred to it as "Cotard's Syndrome," and the term was then popularised by Ségla's 1897 book (Cotard, Berrios and Luque 1999, p. 271). Régis influentially argued that Cotard meant to describe a syndrome—that is, a cluster of symptoms—that was primarily associated with severe melancholy but could be found in other conditions (Berrios and Luque 1995a, 220). This was the position of Ségla, who argued that Cotard's syndrome did not represent a "distinct clinical entity" and that it ought to be classified in terms of its etiology rather than the particular content of the beliefs.⁴

We recount this debate at some length to emphasize the fact that Cotard's description—adopted by the syndrome camp—was in terms of a cluster of symptoms. He originally postulates six co-occurring symptoms: melancholic anxiety, ideas of damnation or possession, tendency to suicide or self-harm, delusions of non-existence of bodily organs or soul, delusions of immortality, and analgesia.

4 See Berrios and Luque (1999, p. 271). The issue of whether delusions ought to be categorized by content or etiology remains a live one; see (Clutton et al 2017). For a meta-analytic study of Cotard's syndrome and the suggestion that it might cover several subtypes, see (Berrios and Luque 1995b)

The finding of analgesia is what concerns us. Cotard's patient, Miss X, herself "showed bilateral reduced sensibility to pain in most areas of her body: for example, she would not show any reaction when pricked by a pin." (1880/1990 p. 275). This was not due to a general numbness. He reiterates that most such patients do not show pain when pinched or pricked, and that this enables self-harm (p. 278). Cotard includes anesthesia as the most striking of the alterations of sensibility in his 1882 report.

Now here, we pause to note an obvious interpretive puzzle—one that won't entirely be resolved, nor need it be—about the meaning of 'anesthesia'. Our context is pain asymbolia, and we would want to know whether Cotard's patients are truly anesthetic or feel pain but are not motivated by it. The distinction is not obvious even to examiners; the modern debate over whether some schizophrenics have asymbolia-like deficits (Singh et al 2006, Virit et al 2008, Bonnot et al 2009) for example, arises precisely because a motivational deficit with felt pain and a lack of pain entirely often result in the same behavior. Cotard's primary aim in discussing alterations of sensibility seems to be to distinguish *délire des négations* from delusions of persecution; in the former alterations of sensation are common (he claims), while in the latter they are not.

What is obvious, however, is that many of Cotard's patients have profound motivational deficits that extend to many areas of their lives. He notes a finding of general emotional deadening, and how this is tied up with their belief that they lack organs; quoting Falret, he claims that "They claim that they have no more love, no more affection for their parents and their friends, even for their children."⁵ And in a later description of severe melancholy more generally, his recounting of first-person accounts of the anesthetic state makes it sound more like depersonalisation than strict lack of pain as such:

Some melancholics are aware of this state and complain that their sensitivity is dulled, they declare that their various senses no longer perceive the external world except as through a veil. Some even complain that they no longer suffer.⁶

This distancing has an obvious parallel to later accounts of depersonalisation, to which we will return in later sections.

What also appears clear is that the link between lack of affect and abnormal sensations came to be recognised as a clinical syndrome in its own right. As an example, consider a 1905 case report from the French psychiatrist Gabriel Revault d'Allonnes. It is notable in part for its appearance

5 (Cotard 1882, p. 159) "Ils prétendent qu'ils n'ont plus de cœur, plus d'affection pour leurs parents et leurs amis, ni même pour leurs enfants."

6 (Cotard 1891, p. 280) "Quelques mélancoliques ont conscience de cet état et se plaignent que leur sensibilité est émoussée, ils déclarent que leurs divers sens ne perçoivent plus le monde extérieur que comme à travers un voile. Quelques-uns même se plaignent de ne plus souffrir."

in a philosophical journal—d’Allonnes’ primary interest in the case is his patient Alexandrine’s impaired sense of time. However, Alexandrine herself shows a curious indifference both to her emotions and bodily sensations.

As a parallel to Cotard’s discussion above, Alexandrine expresses great distress at the idea that she no longer feels emotions with respect to her husband or child. She also claims that she no longer feels hunger or the desire to urinate, nor is she bothered by heat and cold. And, d’Allonnes notes, she does not respond to pinprick over most of her body.

Yet d’Allonnes notes obliquely that Alexandrine is not possessed by a “fixed idea of negation”⁷—she is fully aware of herself as alive and human. Furthermore, she lacks obvious perceptual or motor deficits, and she shows the outward signs of emotion—her heartbeat accelerates when she anticipates the arrival of her beloved son, for example. Much of her behavior continues as before, albeit in conventionalised way, and d’Allonnes is primarily concerned with her disrupted sense of time rather than her anesthesia—though in a footnote (602, n. 1) he says that he is not talking about sensations being completely extinguished so much as having a higher threshold for appreciation.

Most notable for our purposes, though, is d’Allonnes’ postulation that the core of Alexandrine’s problem is the absence of cenesthetic sensations (*sensations cénesthésique*; p. 610). ‘Cenesthesia’ was introduced by Reil to refer to the special sense by which the mind was informed as to the state of the body (Jenkins and Röhrich 2007, p. 362)—interoception in modern terms.⁸ The idea that certain disorders intrinsically involved disordered cenesthetic sensations was popularized in France by Dupre and Camus. It was for a time quite a common topic of discussion in psychiatric literature, though it has again become a rare term (Jenkins and Röhrich 2007, pp. 362–3).

The idea of cenesthesia as affecting pain was also remarked upon, and in language that further suggests something like pain asymbolia. In Sollier’s 1907 English-language review of cenesthetic disturbances, for example, he remarks of one patient that her “pain sense is singularly dulled, and when she is pricked she says the effect upon her is as if it were being done to another person, although her tactile sense is preserved.” (p. 3); of another, he writes that he has become analgesic and that “[h]e cannot distinguish what hurts from what does not” (p. 4). These again suggest an ambiguous relationship between anesthesia strictly speaking and a lack of appreciation for pain.

In sum, there is a French-dominated tradition of thinking about severe depression and a collection of symptoms defined by, but apparently dissociable from, Cotard’s *délire des négations*, which includes something like a lack of appreciation of pain as one of the notable (but not invariable) features of the syndrome.

7 (d’Allonnes 1905, p. 610) “n’est point le jouet d’une idée fixe de négation”

8 Note that while there is a similarity with the term ‘kinesthesia’, the idea of cenesthesia is much broader, encompassing all manner of visceral sensations.

This is, to emphasize, a very different *approach* to the neuropsychological, proto-dissociationist tradition which introduces the term ‘asymbolia’. The logic of dissociation, as it would come to be elaborated in the 20th century, focuses on specific deficits at the individual level. By contrast, lack of motivational force from pain is just one of many symptoms discussed in this alternative tradition, and often less striking than the delusional beliefs possessed by the patients discussed. Hence it does not appear to have been systematically investigated, or entered into the clinical literature in the same way.

5. Some complications: The rise of depersonalisation and the lingering effects of taxonomic choices

We have barely scratched the surface of the clinical literature in the late 19th and early 20th centuries, of course. Nor is the subsequent development easy to trace, given the explosion in the relevant literature. However, we think a few themes are worth noting.

The first is the shift of discussion of cenesthetic feelings and cenesthopathies away from negative symptoms towards a focus on positive ones. In Dupré’s (1913/1974)’s “Les Cénesthopathies” for example, the elaborate lists of cenesthopathic symptoms focus on abnormal positive sensations. We hear that the patient might complain of his body that

Different parts are felt to be narrowed, widened, flattened, swallowed, dried, knotted, displaced, or perhaps changed in shape, temperature, weight, secretory activity, movement, and rigidity; they may seem to be held down and compressed by crampons, strings, or tongs; foreign bodies may be felt to be inserted, gases to seep in, and air to circulate... (1913/1974, p. 389)

Insofar as pain is mentioned in this literature, *hyperalgesic* syndromes are more notable. And indeed, the term ‘cenesthesia’ itself became less and less common, and mostly associated with a particular subtype of schizophrenia; the ICD-10, for example, includes it in the ‘other schizophrenia’ category (F20.8). Abnormal positive sensations remain primary focus of that literature (see e.g. Rörich and Priebe 2002; Jimeno and Vargas 2018).

In the same manner, Cotard’s *délire des négations* has become more focused as Cotard’s delusion. This clinical narrowing in turn emphasizes the bizarre beliefs involved, rather than a broader synoptic context.

Second, and similarly, we find a shift in discussion of aphasias, such that more complex and more detailed forms are emphasized. This is in part because of an increased appreciation of both language and language processing. As a part of this refinement, a simple distinction which placed some aphasias as ‘asymbolic’ appears to have more or less vanished from the clinical literature.⁹

9 Chapter 7 of Shallice (1988) contains extensive discussion of these taxonomic debates. Note that, so far as we can tell, ‘asymbolia’ as a term is not mentioned anywhere in Shallice’s seminal work.

Similarly, other breakdowns have received more specific terminology. Hence ‘pain asymbolia’ ends up persisting as something of a lexical orphan.

Third and finally, we have the rise of depersonalisation as a clinical entity in its own right. This work tended to absorb the discussions of syndromes in which typically motivational states lacked motivational force. However, it appears to be a feature of this work—particularly in its popularization by Janet—that it tended to emphasize the extent to which patients appear normal from the third-person perspective but complain about loss of affect (as appears, for example, in d’Allonnes’ patient above). As Billon (2017, p. 206ff) expertly discusses, the fact that many such patients appear to have accurate interoceptive judgments is taken as a mark against cenesthetic theories.

That is interesting, however, because there are clearly asymbolia-like reports even in early discussions of depersonalisation. Janet, for example, writes of a patient who reports that:

It was painful and my arm felt like withdrawing, but it was not a genuine pain, it was a pain that did not reach the soul.... It is a pain, if you want, but the surface of my skin is miles away from my brain, and I do not know whether I am suffering (Janet 1928, quoted in Billon 2017).

6. Returning to Schilder

We want to finish by considering one interesting historical fact about Schilder, who (to repeat) published with Stengel the first official report of pain asymbolia in the scientific literature in 1928. In a retrospective discussion of his early work on pain asymbolia published in 1950, it is quite clear that he still thinks of it as a failure to connect meaning to pain sensations. Writing of pain asymbolia, he describes it as a case “in which the perception of pain is seemingly preserved, although the individual does not take into consideration the pain he actually feels” (1950, p. 101). He attributes this to an “incomplete utilisation of the pain perception” (p. 101).

But, interestingly, he then goes on to suggest that this incomplete utilisation arises in turn because the pain becomes dissociated from the body image and therefore the “personality” of the patient (p. 102). Notably, this occurs in the midst of a long discussion of the development of the body image, and in a broader book concerned with the human body image and various deficits that might arise therein. On this view, the ‘meaning’ of pain that asymbolics lack is thus clearly connected to its links to the body image.¹⁰ Schilder further notes that

10 Note, however, that this is not necessarily uniform even among earlier writers; Pötzel and Schilder (1936) at least appear to distinguish pain asymbolia from a broader “Danger Asymbolia” (*Gefahrsaymbolie*). This distinction does not appear to have caught on, however.

Patients [with pain asymbolia], as I have mentioned, are also insensitive to threatening gestures and to dangerous situations generally. Danger is, after all, a danger for the body. One could even talk of an asymbolia for danger. The patients also do not react to loud noises. But the lack or the incompleteness of the reaction to pain is still the outstanding feature in cases of this type. (p. 103).

Thus, despite his connection to the German tradition, Schilder seemed to move in his 1950 book toward a more syndrome-based approach of the type espoused by the French tradition and developed by Klein. He even seems to provide some inductive support for the main prediction of Klein's explanation—the prediction that pain asymbolia brings with it more general deficiencies in reacting to immediate threats to one's body.

In the same passage, Schilder also notes that the patient from the original 1928 report subsequently died and presents an autopsy report. The picture he gives is broadly consistent with the later postulation of insular damage as a key cause of asymbolia (Berthier et al 1988).¹¹

Now, suppose that Schilder was indeed in the process of making such a move to a more *syndrome*-based approach typical of the French tradition. That does seem to raise a puzzle, however. To our knowledge, Schilder never drew an explicit link between pain asymbolia and depersonalisation, and that is despite the fact that he wrote extensively on depersonalisation in his career. So, if Schilder was beginning to entertain in 1950 the kind of view developed by Klein where pain asymbolia is understood as part of a larger issue related to bodily care, then why didn't he draw the corollary that there is a tight link between pain asymbolia and depersonalisation?

The story we have told in the last few sections suggests a plausible two-part answer to that question. On the one hand, asymbolia is introduced in the German tradition in the manner of a specific deficit, parallel to aphasia, associated with specific kinds of lesions. This contrasts it with depersonalisation, which is a syndrome with a less certain etiology. Indeed, Schilder himself was also aware of the complexities of this tradition, for he

11 Schilder reports that:

The autopsy showed a small lesion in the frontal lobe and a rather extended lesion, which reached from Heschl's circonvolution and the upper part of the gyrus temporalis primus to the supramarginal gyrus, which was destroyed in its lower part, and extended to the angular gyrus, in which the lesion was much less pronounced than in the supramarginal gyrus, which was decidedly in the centre of the lesion. (Schilder 1950, p. 102).

These areas overlie the insular cortex, and it would not be a stretch to suppose that a lesion of this size would either involve the insular cortex or the white matter connecting to it. Schilder himself is convinced that the supramarginal gyrus is the key to asymbolia, because he thinks it has something to do with the buildup of the body image – see subsequent discussion at p. 102 and also p. 327. Pötzel and Stengel (1937) also have a brief discussion of a case of aphasia with asymbolia along with an extensive autopsy, and similarly emphasize the role of insular lesions (p. 188).

explicitly distinguishes asymbolia and the cenesthesias (1950 p. 139)! He gives very little by way of reasoning or justification, but if the picture above is correct, that is because he has already conceptualized asymbolia as belonging to a quite different class of phenomena. On the other hand, the emphasis in the depersonalisation literature comes to be on preserved reactions despite feelings of distance and emptiness, which (we suggest) might downplay more severe asymbolia-like cases. This may have been encouraged in particular by Janet's own theory of pain, which makes a strong distinction between physical pain and suffering, and Janet's claim that depersonalisation affects only suffering.¹² Schilder's taxonomic presuppositions thus prevented him from seeing pain asymbolia as a kind of depersonalisation and from operating a complete move toward the more syndrome-based approach that originated with the French tradition.

7. Conclusion: Taxonomy and History

Psychiatry is a realm where taxonomic questions have always loomed large. Indeed, in a report to the World Health Organisation in 1959, Stengel bemoans the fact that "The lack of a common classification of mental disorders has defeated attempts at comparing psychiatric observations..." (p. 601). The last 41 pages of his report collect up several dozen taxonomies—many still familiar today.

The question of how broadly asymbolia-like phenomena can be found is ultimately an empirical question. Our goal has not been to settle that question, but rather to show how the taxonomic assumptions with which a concept was introduced can continue to shape thinking about that concept long after the assumptions have vanished, much as the layout of an ancient city ends up affecting the contemporary street plan.

A better sense of the history of a concept—the tradition it was postulated in and how that shaped choices about what to include and what to exclude—can reveal how that history continues to echo in modern debates.

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12 See e.g. Janet (1928 p. 239):

In a very elementary way, pain is simply a reflex act of distancing and we must not speak of a disagreeable emotional tone. In patients who have a high degree of the feeling of emptiness, we observe bizarre expressions: "Yes, you hurt me by pinching me, it's pain, if you like, but it's not real pain, that is not painful to me, I do not suffer.' Suffering is, in fact, something more than pain: it is constituted by recoil and anguish in addition to distancing."

The distinction between pain and suffering is made on similar grounds to that of Klein (2015a): the latter also accompanies states like fatigue (see e.g. Janet 1928, p. 65–66).

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