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The construction of anosognosia: History and implications

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ABSTRACT

The construction of anosognosia as a clinical ‘disorder’ resulted from the convergence (in the work of various writers and culminating in Babinski) of a name, a concept, and a clinical phenomenon. During the early stages of this convergence, unawareness of neurological dysfunction was not considered as an independent clinical phenomenon. Started in the work of Anton, the process of separating it as a differentiable clinical state is completed by Babinski who reaffirmed the semiological independence of ‘unawareness’. The history of the construction of ‘anosognosia’ parallels the late 19th century debate on the nature and brain inscription of the concept of ‘consciousness’.

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1. Matters historiographical

The celebration of the centenary of ‘anosognosia’ (Babinski, 1914) provides an opportunity to revisit the history of its construction.¹ This is no easy task for from its inception anosognosia² has: 1) shown permeable boundaries allowing for the accumulation of all manner of clinical descriptions and explanatory hypotheses; 2) been definitionally reliant upon concepts such as awareness, consciousness, and knowledge whose definition, structure and content are known to have

changed during the last century; 3) included clinical phenomena³ with vague ontology⁴ (i.e., are they best defined as events or as natural kinds?) and; 4) made use of explanatory mechanisms ranging from agency to passivity (is anosognosia the result of ‘denial’, self-deception,⁵ disconnection or disruption of brain pathways?).

To deal with the historiography of such a nebulous clinical notion the historian needs to resort to categories borrowed from the sociology of knowledge⁶ and make use of a ‘convergence’ model. According to the latter, clinical categories result from the conjunction, at a given historical time,

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of a behaviour, a term, and a concept (Berrios, 2011).⁷ This approach is adopted here and examples of illustrative clinical cases⁸ are summarised under each of these headings. Convergences are historical events and hence a historical context will be briefly provided.

2. The historical background

Anton, Babinski and the other writers who were involved in the construction of ‘anosognosia’ thought and wrote during a specific historical period. Their work can only be understood if something is known about their conceptual milieu. Twenty-first century views on awareness and consciousness are not helpful in this regard.⁹

Scholars agree on the view that the concept of ‘consciousness’ (in the sense in which this notion is understood nowadays) is absent in Plato and Aristotle (Cancrini, 1970; Ellrodt, 1983). This claim can be variously interpreted. If it is assumed that ‘consciousness’ is a thing or state necessarily generated by the brain (and on the assumption that brains have not changed since Classical times) then, it could be asked why was it not possible for Plato or Aristotle to ‘discover’ it or ‘describe’ it. If, on the other hand, it is posited that consciousness is a construct then it has to be asked what mind concepts and other cultural factors were present in Classical Greek thinking that made the concept of consciousness either redundant or impossible.

The history of the construction, or ‘invention’ (as it has been aptly called by Balibar, 2013) of the concept of consciousness in Europe since the 17th C is long and tortuous (Brain, 1958; Clarke & Jacyna, 1987; Williams, 1958). In Descartes and Locke the term ‘consciousness’ refers to that act whereby man ‘gets to know’ (becomes aware of) the world around and himself. Hence, it is used in a predominantly epistemological sense. It was not meant to refer to a mental faculty or power, to a circumscribed ‘thing’ which in the fullness of time might be considered as the specific ‘function’ of a brain structure or network.

The concept of consciousness was fully ‘psychologized’ only during the 19th century. In practice, this meant that in addition to being a metaphysical or epistemological notion (Shepherd, 1915) consciousness started to be considered as a central component of the mind and hence of the brain. This made possible the development of germane notions such as self-reflexion (Janzen, 2008) and of research methodologies such as introspection (Danziger, 1980). This process reached its culmination in the work of Laycock (1845; 1869) who expressed the view that consciousness was sited in the cranial ganglia, was a mere accompaniment of perception and hence had no causal role.

In conceptual terms, the 19th century entertained a reworded version of the old Aristotelian dilemma (Caston, 2002) about the nature of consciousness. Was it: a) an independent mental function (Consciousness Type₁) and hence needed to be inscribed in a dedicated region of the brain or b) just a secondary feature, an epiphenomenon that appears associated with each mental function or sensory organ (Consciousness Type₂)¹⁰ (Hamilton, 1877). The fact that C₁ could lead to an infinite regress,¹¹ convinced some to opt for C₂. Both

options, however, generated different models of anosognosia. In normal functioning, according to C₁, the brain region where consciousness was sited required to be ‘connected’ with each other sensory region (this connectionism is typical of Wernicke’s model). Hence anosognosia was to be explained as a disconnection syndrome.¹² In the case of C₂, anosognosia required a different explanation and soon enough various were marshalled such as partial lesion, sensory disorder, memory disorder; and after the advent of Freud, concepts such as denial and suppression (illustrations for these explanatory models are provided below).

3. Anosognosia: the behaviours

Descriptions of patients showing ‘unawareness’ of neurological dysfunction (e.g., blindness, Redlich & Dorsey, 1945) can be found before Babinski coined ‘anosognosia’ in relation to hemiplegia. Amongst others, unawareness was reported in relation to blindness (Anton, 1899; Dejerine & Viallet, 1893; von Monakow, 1885; Redlich & Bonvicini, 1911), deafness (Anton, 1899) and alexia (Bonhoeffer, 1903).

In the context of searching for correlations between clinical findings and brain lesions von Monakow (1885) described 4 patients with cortical blindness.

Case 1.

70 year old man who had been well until 4 years prior to admission when he developed severe nose bleeds followed by a left sided facial paralysis, left hemiplegia and mild dysphasia. He had visual hallucinations and some visual impairment. He recovered but was left with mild gait unsteadiness, mild visual impairment and subtle intellectual impairment. The next year, following an epileptic seizure he developed mild left sided facial and limb pareses and his visual impairment became more obvious. Again he improved and remained relatively well for 2 years. He then began complaining that his eyesight was worsening and he was experiencing difficulty with writing. At that time, he complained of a thick fog in front of his eyes – blaming this on bad weather. Ophthalmological examination was normal. The next month following another ‘apoplectic attack’ he became completely blind as well as suffering visual hallucinations, mobility problems and speech problems (he could not understand the spoken word). His mobility improved quickly. However, his blindness remained and from this point on he was not aware of his blindness. Initially he thought that he was in a dark pit or cellar and shouted for light and fire. Later, he appeared to have got accustomed to the visual hallucinations and so the notion that he could not actually see anything did not reach his awareness. He complained that he was old, stupid and weak – but he never articulated that he was blind. This, together with the speech problem, gave the impression that he was totally confused and disorientated and robbed him of the possibility of making sense of his environment. He often believed that he was outside the home and demanded to go home. Although remaining well kempt, empathic, and at times able to speak coherently,

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