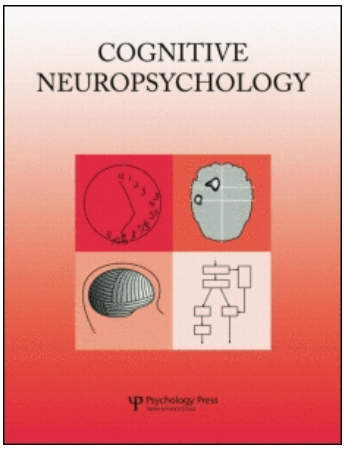

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An Annotated Summary and Translation of "On the Self-awareness of Focal Brain Diseases by the Patient in Cortical Blindness and Cortical Deafness" by Gabriel Anton (1899)

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This report will concentrate on the case described by Anton of lack of awareness of cortical blindness which has come to be known as Anton's Syndrome. This was part of a long paper (see above); it has not hitherto been available in translation.

INTRODUCTION

This paper was not the first description of lack of awareness of disease following brain damage—anosognosia, as it was later to be named by Babinski (1914). The phenomenon had been described in patients with aphasia, paresis and even blindness (von Monakow, 1885).[1] Nor did Anton provide a definitive theoretical interpretation, although his anatomically based views are of relevance. We focus on this case study since it clearly demonstrates the specificity of the deficit, given the patient's relatively preserved linguistic and intellectual functioning, and also its domain specificity. Anton also emphasised the focal nature of the underlying brain damage. It is these observations which give the case a contemporary relevance.

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[1]Von Monakow described two cases. One was a 70-year-old man with left hemiplegia and slight aphasic disturbance. He was unaware of blindness and stated that he thought he was in a dark hall. He had widespread and bilateral brain lesions affecting particularly the left occipital lobe and superior temporal gyri. The second was a 50-year-old epileptic man with cortical blindness and naming difficulties. Though disorientated, spatial orientation and object recognition were intact. He was said to be totally unaware of his blindness. He too had bilateral lesions including the lingual gyrus and third frontal gyrus.

A comment on the historical development and recent interest in anosognosia is given by Prigatano and Schacter (1991) in the introduction to their book, the first to deal comprehensively with the topic since Weinstein and Kahn (1955). They speculate that one factor is the decline in behaviourism, with the ascent of cognitive psychology and interest in consciousness as a concept. A second is the awareness on the part of clinicians of a range of striking disorders of consciousness seen following natural and surgical brain lesions. Such disorders, in addition to anosognosia and neglect, include amnesic syndromes, jargon aphasia, blindsight, and the split-brain syndrome, as well as neuropsychiatric conditions such as anti-social personality changes and reduplicative paramnesia. These disorders provide a framework for studying the otherwise diffuse notions of consciousness and awareness in normal psychology and as such exemplify the paradigm of cognitive neuropsychology. The final reason they give concerns the practical impact lack of awareness of disease has on neuropsychological rehabilitation.

Gabriel Anton was born in July 1858 in Saatz, Bohemia. He obtained his doctorate in 1882 from the German University in Prague. He worked in general medicine and psychiatry before entering Meynert's department of neuropsychiatry in Vienna in 1887. In 1899 he became honorary professor and director of the Neuropsychiatric Hospital, Innsbruck, and from 1894 he was professor of neuropsychiatry in Graz. He took over the chair in Halle following Wernicke's death in 1905 until 1926. Anton died in 1933 (Förstl, Owen, & David, 1993).

Although Anton published widely on topics ranging from agenesis of the corpus callosum (1896) to dementia associated with involuntary movements and liver disease (1908) (probably the first description of Wilson's Disease, four years before Kinnear Wilson), his observations on patients who showed impaired perception of sensory deficits are best remembered (see Owen, David, & Förstl, 1993). These were first published in 1893, a collection of four cases with proprioceptive loss. The case of cortical blindness was first reported at the Society of Physicians of Styria and published in the Society's Communications (Vol. 3, Book No. 8544; 1896). A detailed clinico-pathological account is given, along with two cases of cortical deafness, in the 1899 paper.

The two cases of cortical deafness set the scene for the main case. The first was a 64-year-old man who suffered a head injury some 10 years before. He was said to experience "vivid auditory hallucinations." In addition he did not notice anything amiss when watching other people speaking despite being unable to hear them. When asked to read the statement "Mr. F is deaf" he protested that his hearing was fine. Anton mentions that the patient was fully orientated but named objects incorrectly, used incorrect syntax and sometimes used meaningless words (no examples are given).

The second case was a 69-year-old woman who was followed up to post-mortem. She presented with severe confusion and was found not to

react at all to loud noises or speech. She never complained of her deficit and, indeed, was said to be "completely indifferent" to it. Assessment must have been complicated by noticeable aphasia. The patient's speech was described as "remarkably disturbed" with incorrect words, grammar, and neologisms. She was also a letter-by-letter reader. Nevertheless, she could recognise people, could name objects, localise touch verbally, and generally behaved appropriately. Post-mortem examination showed extensive bilateral temporal lobe softening.

There now follows a full translation of the clinical and relevant pathological findings on the main case described in Anton's classic paper.

ON THE SELF-AWARENESS OF FOCAL BRAIN DISEASES BY THE PATIENT IN CORTICAL BLINDNESS AND CORTICAL DEAFNESS: A TRANSLATION

Ursula Mercz, a 56-year-old needlewoman, was treated in Graz Hospital for Nervous Diseases from 30th November 1894 until 29th May 1895.

All we could find out regarding the previous history was that she has been frequently suffering from vertigo and headache, that even four years ago she had appeared changed to the people who were close to her, and that she had been completely unable to work for two years.[2] The patient survived three spontaneous abortions.

Some of the facts from the medical findings shall be emphasised. The patient's movements, particularly walking, were severely handicapped by pain; almost all of her nerve-trunks were extremely sensitive to pressure, particularly alongside the spinal column.[3] The bones showed striking mobility and flexibility. The face is symmetric, the pupils react promptly. The fundi are completely normal apart from increased tortuosity of the veins. All skin and tendon reflexes are lively.

During conversation, it was soon realised that the patient could not remember various words, particularly names of objects, and that she preferred to use indirect descriptions (*Umschreibungen*). Whenever she used the wrong words, she soon became aware of it; this perception and the lack of ability to find expressions that she wanted to use, and that she was looking for, caused vivid and angry reactions.[4] She found the expressions

[2]The description is too vague to allow a diagnosis but the history suggests perhaps a multi-infarct disorder.

[3]This suggests a "neuritis," the cause of which is entirely unexplained.

[4]This is a crucial observation. The patient had retained an acute awareness for her apparently modest word-finding difficulties (Marshall & Tompkins, 1982). This dissociation clearly challenges any proposal that the possible early dementia or language problem per se (see also McGlynn & Schacter, 1989) is sufficient to account for loss of awareness of blindness. The dissociation between sensory or motor impairment and anosognosia has been clearly demonstrated (Bisiach, Vallar, Perani, Papagno, & Berti, 1986). Similarly, Brain (1941)

for her own spontaneous line of thought rather well.[5] She had great difficulties with naming items from memory[6] when prompted by the physician. She often asked to be put on the right track and not to be bothered.

Sometimes the patient showed a minimally preserved visual field, but this finding was followed by complete blindness on the very same day.[7] At the beginning of January [1895], she could sometimes see and recognised dim outlines of objects, e.g. a circle drawn on a board, a triangle, scissors, a red ribbon. This state was replaced by *complete blindness* although pupils and fundi remained normal.[8] The patient could not distinguish light and

noted that neglect of extra-personal space could coexist with intact awareness of representational space, and more recently Schacter (1990) described dissociations between types of amnesia. However, it is the presence of the propensity to complain of bodily malfunction in one area or domain while remaining indifferent to often more serious malfunction in another that is striking and supports the domain-specificity of anosognosia. Stengel and Steele (1946) described a patient with a frontal meningioma who denied his dense blindness, describing his vision as "pretty good, pretty decent" (also memory impairment—he said his memory was "very good") yet complained excessively of a varicose ulcer around his left foot saying ". . . the only thing that is the matter is the left foot."

A particularly striking example of the specificity of anosognosia has been provided by Young, De Haan, & Newcombe (1990) who report a woman with marked prosopagnosia, recognising only 3 to 4 out of 20 famous faces. Prior to a cerebrovascular accident, she was a gifted amateur portrait painter. She reacted with mild surprise or disbelief when confronted with her deficit, insisting that the photograph was a poor likeness, and that she could recognise faces, "as well as before." She could not recognise her own paintings without a laborious deductive method yet did not admit to anything unusual about this. Such lack of insight was contrasted with appropriate concern for her impaired memory, hemiplegia, and hemianopsia.

Finally of interest is the relationship between psychiatric disturbance and Anton's syndrome. Psychiatrists frequently comment on a psychotic patient's "lack of insight," referring to a failure to recognise that they are in some general way suffering from a (mental) illness as well as not recognising certain abnormal mental events, such as hallucinations and delusions, as being unfounded (see David, 1990). Many descriptions of anosognosia have a "quasi-psychotic quality" (Bisiach, 1988). Brockman and von Hagen (1946) described two patients, the first of whom had paranoid delusions prior to the onset of cortical blindness with denial, and a second, who experienced auditory and visual hallucinations as well as delusions of persecution while suffering temporarily from blindness due to hypertensive cerebro-vascular disease. Dissociations between "psychotic" unawareness and "neurological" unawareness have yet to be reported.

[5]This is a fair description of anomic aphasia (for historical survey of this symptom see Benton & Joynt, 1960). Unfortunately, no examples are given so that no assessment can be made of, e.g., semantic errors or modality specificity. There does seem to be a discrepancy between spontaneous speech and conversation, with the latter producing more word-finding difficulties.

[6]This refers to the view, current at the time, that saw naming difficulties in terms of a memory defect, hence "amnestic aphasia" (Pitres, 1898). We take this to mean simply "anomia." Although authors have frequently noted similar aphasic disturbances in these patients (e.g. F. Redlich & Dorsey, 1945) they are not invariably associated. E. Redlich had earlier reported three cases, two of whom had no aphasia (Redlich & Bonvicini, 1908).

[7]This fluctuation was noted in a subsequent case of loss of awareness of blindness (Redlich & Dorsey, 1945, case 4).

darkness, she could not perceive objects, either when close to or distant from her, even a sudden approach or quick intensive illumination did not elicit lid reactions.[9] Her gaze stared into empty space; fixation could not be confirmed.

It was very striking that the patient did not take any notice of extreme and, later on, complete loss of vision. The patient, who was otherwise complaining a lot, was almost unaffected by this loss. When objects were presented in front of her, she—probably according to habit acquired during recent years—immediately tried to touch them, but she did not make an effort to recognise something by looking at it. If the object was presented at some distance or if touching was prohibited, she gave it any name purely by guessing. It was obvious that she, like many blind people, had become an experienced guesser. Her language capabilities were certainly sufficient to permit this statement.[10] When asked directly about her vision, she answered in vague, general terms, "people see better when they are young, that's just how it is."[11] She confirmed, calmly and faithfully, that she could see the presented objects, whereas almost daily examination proved the opposite. She also claimed to see things that were not actually presented to her.

The patient was *not aware* of her loss of vision, and this defect did not cause her any further thought or conclusions, neither sorrow nor dislike.[12] And all this during a time when a missing name or remembering a word caused obvious pain.[4]

A further striking symptom was her *lack of spatial orientation*. The patient could not find her way in a room where she had been lying for several weeks. She does not find the glass which is always standing at the same place beside her. She does not know where her dresser, her chair,

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[9]The distinction being emphasised refers to the cortical nature of the blindness, which was bilateral. Redlich and Dorsey (1945), Stengel and Steele (1946), and Hemphill and Klein (1948) each described a case where the blindness was due to optic nerve pathology. The former two cases were due to pressure from frontal tumours causing optic atrophy. The third case is of interest since the patient's longstanding blindness, secondary to optic atrophy, was within full awareness until the onset of a confusional state. Incidentally, Stengel and Steele described a second patient in whom gradually worsening paraplegia was suddenly denied following a presumed cerebro-vascular incident and manic psychosis.

[9]There is no residual vision and the patient is unlikely to be "faking" the blindness.

[10]Such a confident statement could not be made regarding the other patients, with cortical deafness, described in Anton's paper, the second of whom had severe jargon aphasia (see Owen et al., 1993). Presumably the patient used auditory cues to realise an object had been placed in front of her, although Anton does not elaborate on this.

[11]It could be argued that such statements betray some awareness of a problem with vision. Most cases in the literature have included statements of this kind or that the lights had been turned off (e.g. Cusumano, Fletcher, & Patel, 1981) etc.

[12]This lack of affective response has been termed anosodiaphoria by Critchley (1957). For recent views on this, see Gainotti (1972) and Säring, Prosiegel, & von Cramon (1988).

etc., stand.[13] When asked where the door, the windows, the patients' dining table are she gives the wrong directions.[14] Even auditory stimuli, which have certainly been perceived, could not be localised. She heard the ticking of a watch from a distance of 35cms, but she locates it next to her ear.[15] Often she gives the wrong side from where she hears the sound and she cannot determine the side of the bed where the examiner, who is speaking in a loud voice, is standing. When she bumps violently against the frame of her bed, i.e. after tactile stimuli, she can find her way quite quickly. She often makes the wrong distinction between right and left in spite of lengthy consideration.[16] On several days, the patient gave two wrong answers after being asked five times.

It can be noted that the patient can localise touch and pain sensation on her body rather well. When asked to do so, she also touches her nose, her mouth, her ear, etc., quite skilfully.[17] She can determine the position of her extremities and she imitates the movements and positions that are given to the contralateral extremities without gross mistakes. When told to do so, she is also able to draw circles and other figures in the air or to cut these figures from a paper with scissors.

In her memory, the patient has retained her mental imagery (*Gesichtsvorstellungen*). She describes where she lived, her house, her animals, their sizes and colours, as far as her expressive capabilities allow.[18] While doing so, she cried—but this was probably a normal emotion. She

[13]Although this could be an example of topographical amnesia (De Renzi, Faglioni, & Villa, 1977) normally attributable to right hemisphere damage, the patient's visual loss should be taken into account. Nevertheless, localisation of sounds is also impaired.

[14]This statement is more in line with a representational deficit but right–left disorientation (usually ascribed to a left parietal lesion) and visual impairment may both be confounders (see later).

[15]This is a good account of a fairly specific deficit in auditory localisation, a phenomenon rarely studied (Kase, Troncosos, Court, Tapia, & Mohr, 1977).

[16]Right–left disorientation is an element in the so-called Gerstmann syndrome, due to lesions of the dominant parietal lobe (see Benton, 1977). It is not clear how the patient's deficit in right/left discrimination was elicited. We presume it was by naming.

[17]See Pick (1908) for the first description of impairment in the localisation and naming of body parts.

[18]This preservation of visual imagery (including dimensions and colour), plus her ability to draw, is of note. According to Farah (1984), this shows intact generative functions, despite bilateral occipital damage. It could be hypothesised that such systems are necessary in order to produce hallucinations in a "blind" visual field, and may even underlie the lack of awareness of the blindness. The link between hallucinations and Anton's syndrome has been revived by Swartz and Brust (1984), who report a 60-year-old man of normal intelelct, blind through trauma to both eyes, during alcoholic withdrawal. The patient experienced hallucinations and offered descriptions of his surroundings, believing his blindness had been cured. Interestingly, he distinguished between the typical alcoholic hallucinations of people and animals all around him, realising these to be false perceptions, and what he regarded as his true vision.

recognises the persons around her by hearing and touch. She does not make any attempt at all to form a visual image of these people.[19]

Her physical and mental condition deteriorated remarkably in May. The left side of her body became paretic. Only feeble pulsations could be felt over her right carotid. The patient died after a long coma (29th May 1895).

Postmortem (pp. 94)

. . . No severe atrophy . . . cystic necroses primarily affecting the white matter of the left and right occipital lobes . . .

* * *

[Anton discusses under the heading "Considerations" the nature of the problem. He refers to Dufour (1889) in stating that: ". . . patients with a unilateral visual loss also lose their feeling for the lack of this visual field (*Hemianopsie nulle*)." Friedrich Müller (1892) expressed similar thoughts. Anton goes on to remind the reader that hallucinations can occur in the hemianopic field. He concludes this section by stating that ". . . a central sensory impairment can only remain latent for the patient if further brain lesions are present."[20] In the discussion paper (pp. 121 onwards), Anton offers a largely anatomical account of the patient's unusual presentation. A comprehensive review of these and other theories in provided by McGlynn and Schacter (1989).]

* * *

The anatomical main event is the interruption of pathways to other brain areas in smaller or greater distance; this is a breakdown of the functional relationships with other cortical areas. It is undoubtedly possible that the function of one part of the brain, which is not damaged itself, can be altered through other parts.[21]

* * *

Sometimes subcortical centres can represent the substrate of complex functions in the presence of cortical lesions . . . In case II (Ursula M.) we have

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[19]It is not clear whether what is meant here is that the patient did not "invent" a visual description of these people (unlike her tendency to "guess" when asked to name objects).

[20]This is the "two lesion" theory, frequently advanced to explain anosognosia (see Stuss & Benson, 1986). The second lesion is often assumed to be in the frontal lobes, so impeding self-awareness or monitoring. Interestingly, Anton's description and drawings of the pathological lesions found in this case do not indicate frontal damage. Modern neuro-imaging techniques such as CT scanning confirm that single lesions outside the frontal lobes may cause anosognosia (Bisiach et al., 1986).

[21]This is essentially a version of what later became known as the disconnectionist account (Geschwind, 1965).

to consider the possibility that cortical areas, which are cut off from the periphery, can be stimulated from other cortical areas . . . Microscopic examination has demonstrated a sufficient number of shorter and longer association pathways, which can, e.g., mediate stimuli from the temporal lobe to the fairly preserved visual centre.[22] The question whether the patient misinterpreted these stimuli as genuine sensory perceptions . . . cannot be answered on the basis of a single case. We can also only surmise that preserved subcortical seeing and hearing—a major factor in lower animals—can elicit dark sensations, which mask the loss of conscious cortical sensory perception.[18]

Summary of Post-mortem and Histological Findings

Apart from the lesions of the second and third occipital gyri of the posterior angular gyri, which could be recognised from the surface, there was a destruction of the largest part of the pathways leading from this level of the occipital brain to the other lobes. This affected the fibres of the fasciculus arcuatus and the connections with the frontal lobe (fasciculus fronto-occipitalis), which have not yet been examined sufficiently.

The pathways connecting the cortical areas of the occipital brain were affected similarly, particularly the cortical and transversal occipital bundles. The short cortical connections, the fibrae propriae, were preserved far better.

Furthermore, the radiations of the superior colliculus and of the lateral geniculate body were cut off in their largest superior parts. This was caused partly by the upper larger lesion and partly by a smaller lesion in the posterior temporal gyrus.

In the same area, the largest part of the lower longitudinal bundle was also destroyed by both lesions, leading to a progressive degeneration and atrophy. The central terminals of the visual pathways, i.e. the cuneus and the calcarine fissure, were essentially unaffected, but the visual pathway was cut off, causing a degeneration of the optic radiation in this area. Therefore this centre was cut off from the periphery.

Finally, the posterior part of the forceps corporis callosis was destroyed by the larger lesion and its fibres at the lateral ventricular wall degenerated. Therefore both occipital lobes were mutually disconnected.

[22]The theory advanced here (and reiterated by Cobb, 1943) is that subcortical or temporal lobe inputs to the occipital lobe take the place of and "reverberate with" inputs from the primary optic pathways and so produce a sensation of vision. This could be thought of as the mirror opposite of blindsight (Weiskrantz, 1986). Here, it is assumed that alternative visual pathways are functioning but the final area which subsumes "awareness" is damaged. The patient claims he is blind when, in truth, he is not. In Anton's syndrome, the patient has phenomenon awareness in the visual domain from nonsensory inputs, so maintains that he is not blind when, in fact, he is.

Considering the subcortical parts, the superior colliculus and the lateral geniculate body showed a considerable atrophy on both sides, but the microscopic examination revealed normal optic tracts. The medial parts of the occipital lobes were basically well-preserved, particularly the "tapestry" of the wall of the ventricle, of the medial ventricular wall, and the extension of the fasciculus cinguli.

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