FROM BLINDSIGHT TO BLINDSMELL: A MINI REVIEW

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Received 11 October 2014 accepted 15 October 2014

1. Introduction

In this mini review our aim is to briefly document the history and case for blindsight, and then examine whether there is evidence for a parallel phenomenon in the olfactory system.

2. Blindsight: historical background and early evidences

According to the Oxford Concise English Dictionary [1], blindsight is “a condition in which the sufferer responds to visual stimuli without consciously perceiving them”. Thus, it seems that some blind people may process visual stimuli that they cannot see. Indeed, the term blindsight represents one of the most interesting and fascinating oxymora of modern visual neurosciences [2]. Maybe, this is one of the reasons why blindsight has received special attention from many scientists seeking to unveil the secrets of visual awareness. First we must examine what is cortical blindness.

Neurological diseases (e.g., strokes, traumatic brain injuries, tumors, and so on) can cause lesions to the primary visual area within the occipital striate cortex (Brodmann area 17 or V1). Complete lesions to both V1 areas result in cortical blindness for both hemifields. Complete lesions affecting either the right or the left V1 cause left or right homonymous hemianopia (i.e., blindness for one hemifield), respectively.

Finally, incomplete lesions of either left or right V1 cause “islands” of restricted blindness (i.e., scotomata). Consequently, patients are unaware of visual stimuli presented in the lesioned portions of their V1 area(s).

The leading role of V1 in visual awareness in humans has been underlined since the early 20th century. Indeed, the famous ophthalmologist Holmes concluded that visual field defects are absolute when the striate cortex was damaged [3]. Ferrier [4], however, was the first to report that apparently blind monkeys could show impressive residual abilities such as reaching out to grasp objects and avoiding obstacles. The results of Ferrier’s original observations were further supported by the findings of Humphrey [5], who showed that a monkey could still find her way among obstacles, even when V1 was completely removed. Thus, the literature on humans with respect to that on non-human animals is characterized by conflicting evidence on the role of V1 in accomplishing complex visual tasks. An initial explanation was that the abovementioned, conflicting findings could reflect the presence of a different neuroanatomical organization and functional role of V1 in human and non-human primates.

Pöppel, Held, and Frost [6] were the first to report experimental evidence for blindsight in humans (see also Bard [7]) by examining four war-veterans affected by visual field defects. These patients were presented with visual stimuli, which were briefly flashed within the patients’ scotomata. Although the patients declared that they have not seen any of the visual stimuli, they were able to perform eye movements towards the spatial positions occupied by the “unseen” stimuli. This was the first experimental demonstration of blindsight in humans reported. Nonetheless, the term blindsight was first coined the next year in 1974 after a talk held by Larry Weiskrantz at Oxford Neurology Department; the talk was entitled “Blindsight and hindsight” [2].

Weiskrantz and co-workers (for review, see Weiskrantz [8]) have extensively studied patient DB, who was affected by left hemianopia because of a benign tumor removal from his right occipital cortex. In a series of experiments that spanned the last four decades, DB’s residual visual abilities have been extensively investigated. Like other patients with blindsight, DB is unable to consciously report the presence of visual stimuli. Nevertheless, DB can perform above chance on a series of tasks including orientation, position and movement of visual stimuli. In addition, DB is accurate in reaching out and locating visual stimuli, in a way similar to that of monkeys without V1.

Following the seminal studies by Humphrey and by Weiskrantz and co-workers, a series of new cases of blindsight have been studied. For instance, GY is one of the most studied patients affected by blindsight [9]. GY suffered damage to the visual cortex, as a consequence of a
road accident when he was a child. It is worth noting that GY was the first patient to show also affective blindsight, that is, a residual ability to judge the emotional value of unseen visual stimuli [10].

3. New insights

Initially blindsight was considered a rare neurological disorder. Recent evidence, however, has suggested that 70% of hemianopic patients have blindsight [11]. Nevertheless, blindsight is not a unitary disorder. Indeed, at least two subtypes have been reported [8]. Type-I blindsight is characterized by complete absence of visual awareness: patients perform well on a series of visuospatial tasks but they are completely unaware of the presence of visual stimuli. In contrast, patients affected by Type-II blindsight show some residual awareness of “feeling” or “knowing” of the displayed visual stimuli. Nevertheless, exactly like Type-I blindsight patients, Type-II blindsight patients deny having any conscious visual experience of the stimuli that they, nonetheless, process implicitly.

Blindsight is a crucial neurological disorder for revealing the properties of our visual awareness and its neuroanatomical underpinnings. The explanation of blindsight can be found in the neuroanatomical organization of our visual system that is composed of two relatively independent pathways [2]. From a phylogenetic viewpoint, the most recent pathway is the retina-geniculo-striate one. This pathway originates from the eye and its projections reach V1, through the lateral geniculate nucleus of the thalamus. This pathway is responsible for conscious vision. If this is the case, then a different pathway must be activated when blindsight-related phenomena arise. Indeed, the most probable candidate explaining blindsight is the retina-tecto-pulvinar-extrastriate pathway, a phylogenetically older visual stream. In this pathway, visual information reaches the extrastriate cortices indirectly by means of projections that originate from the retina. These projections are directed to the superior colliculi, and then to the pulvini. Finally, from the pulvinar projections reach the extrastriate visual cortices. When visual input to this pathway is prevented, blindsight disappears [12].

In conclusion, blindsight is a fascinating and intriguing neurological disorder, which has helped us enormously to improve and deepen our understanding of the different neural systems subserving visual awareness. However, there has as yet been relatively little interest in whether similar a phenomenon can be observed in the olfactory system. In the following sections we examine for analogous findings in olfaction, first drawing upon some of the unusual aspects of routine olfactory perception that are reminiscent of blindsight and then examining the limited neuropsychological evidence for “blindsight.”

4. Indirect evidence for blindsight in routine olfactory perception

While the world of commerce has long realised that odours can surreptitiously affect behaviour (e.g., [13]), academic acceptance of this idea has been slower. The basic claim here is that odours can affect various psychological/physiological processes without someone knowing that this is occurring. This could happen either because the odour is not consciously perceived (i.e., detection without awareness) or because the impact of the odour (consciously perceived or not) is not recognised (i.e., impact without awareness). Effects of both kinds have been documented (see [14-17]).

4.1. Detection without awareness

The fact that odours may not be consciously perceived and still have detectable impacts on brain and/or behaviour has been documented in several types of study. The most convincing have either used some form of biological measure to indicate that the odourant has exerted an effect in the absence of any report of olfactory experience or have combined biological measures with some more objective test of awareness. An important early finding was made by Lorig et al. [18], who reported that odourants, which participants could not reportedly detect, could still significantly affect EEG patterns. A further example is Hummel, Mojet and Kobal’s [19] demonstration of significant electrical activity on the olfactory receptor surface in the absence of any reported odour experience when stimulated with low concentrations odourant. In both of these examples the odourant was seemingly detected by the brain but without conscious awareness.

Biological measures were combined with a more objective assessment of awareness in a study by Sobel et al. [20]. In this case, participants were examined by MRI while engaging in an odour detection task. Participants performed at chance level when attempting to detect low concentrations of the odourant (is it present/absent?), while being above chance for the higher concentration of the odourant. Importantly, significant brain activation was induced by both the low and high concentration of the odourant, whereas the objective measure of awareness was only significant for the high dose. An examination of the differences in brain activations associated with the low and high concentrations, revealed probable processing differences in the thalamus and inferior frontal gyrus. The former has been associated with olfactory attention (see [21]) and the latter with higher-level olfactory processing (e.g., [22]). In sum, the import of these types of studies is that odourants can be detected by the brain in the apparent absence of olfactory awareness.

4.2. Impact without awareness

The literature regarding impact without awareness is considerably larger than for detection without awareness. Odourants have been documented to impact mood and become implicitly associated with other on-going events. Further, there can be significant changes in olfactory perception that do not seem to be accompanied by awareness of these changes (e.g., [23-26]). Here, we will examine some representative examples.

Several studies now suggest that placing participants in environments that are odourised where the odourant concentration is clearly liminal, affects participants’ mood states making people feel happier with a pleasant smell or sadder with an unpleasant smell. What is particularly revealing is that
these types of effects can occur irrespective of whether a participant’s attention is drawn to the odour or not [23]. Although the claim that certain odours can exert more specific effects on behaviour, such as for example the claims made by aromatherapy, are doubtful, there is general agreement that odours can exert effects on mood, and that these can occur without participants attributing this effect to the odourant.

A second type of effect concerns associating a particular odour with a particular event. Many examples of this phenomenon have been documented. Zucco et al. [25] showed that a low intensity odorant paired with a stressful task influenced people’s mood without their awareness when they later re-experienced the odorant separately. Li et al. [24] demonstrated that olfactory stimuli presented under threshold levels can drive implicit preferences and attitudes for human faces. While human conditioning effects are easily demonstrated in the laboratory, what is unusual about odour-related conditioning is the claim that it can occur with minimal conscious awareness (e.g., [27]). This type of claim is highly contentious, because most examples of learning without awareness have been found on closer study to actually involve some knowledge (however fragmentary) of the relationship between the to-be-learned events (e.g., [28]). For olfaction, this debate has played out most extensively in relation to flavour-evaluative conditioning, where one odour dissolved in water and experienced as a flavour with a sweet taste comes to be liked more at the end of the experiment than an odour experienced just in plain water as a flavour (e.g., [29]). Attempts to nail down whether this form of odour learning involves awareness have not reached any clear conclusion (contrast [30] with [31]). However, it is clearly the case that while human participants can learn relationships between odours and other events, they find it particularly difficult to explain what they may have learnt.

A third example comes from a completely different investigation, but reflects largely the same issue. While most experimental psychologists can quickly and easily describe several examples of visual illusions (and probably auditory and somatosensory ones too), they cannot do so for olfaction [32]. One reason for this seems to be that we are particularly poor at recognising changes in the olfactory percept (e.g., [33]). In other words most olfactory illusions may go unnoticed. Stevenson and Mahmut [26] examined this empirically using binaral rivalry, in which participants smell an odour mixture (e.g., mint-banana) and their perception fluctuates back and forth between percepts dominated more by mint, to percepts dominated more by banana. These effects are clearly demonstrable when participants are asked to rate how “banana-like” and how “mint-like” each sniff is. But when participants are asked to judge whether two mixtures smell different (e.g., sniffing a mint-banana mixture on one trial followed by a mint-banana mixture on the next) they never spot the change. Yet again, this suggests that we may be particularly poor at noticing the consequences of olfaction on psychological processes, in this case a change in perception.

5. Discussion

We have illustrated two types of phenomena. First, that odours can be detected by the brain, without accompanying evidence of conscious awareness. Second, that odours can affect psychological processes – mood, learning, perception – without conscious awareness that these processes have been affected. Both of these types of phenomena suggest that humans may frequently experience olfactory events that in the visual domain would seem reminiscent of certain aspects of blindsight – detection and impact without conscious awareness. In the final section of this review we examine some far more direct analogues of visual blindsight.

5.1. Blindsight

Three papers have reported evidence seemingly documenting an olfactory analogue of blindsight. The first reports the case patient S [34], who sustained a traumatic brain injury. Neuroimaging revealed relatively selective damage to the right orbitofrontal cortex, but with no apparent damage to other brain structures known to be involved in olfactory perception. On standardised tests of olfactory functioning patient S was completely anosmic, confirming his self-report that he could no longer smell since having his brain injury. Endoscopic examination of the inside of his nasal cavity indicated no damage to the facial or nasal bones that might obstruct access of odourants to his nose.

Three procedures were administered to patient S at the same time during the test phase. He was asked to engage in an odour detection task while in a MRI scanner, with electrodes attached to his hand to determine any emotional reactivity to olfactory stimuli. Patient S was then exposed to a series of trials that involved the presentation of an odourant in left nostril/right nostril as follows: air/air, pleasant odour/air, air/pleasant odour, unpleasant odour/air, air/unpleasant odour. S’s task was to judge whether an odour was present or absent on each trial. While being below chance at odour detection on both the left and right sides, he was significantly better at detection on the left side than on the right. Indeed performance on the left only narrowly missed being above chance. As olfactory processing is largely ipsilateral, this suggests some preservation of processing on the uninjured side, albeit unaccompanied by any apparent awareness of smell.

Two further sources of evidence suggested registration of the odourants by the brain. First, there was left-sided activation of the orbitofrontal cortex consistent with the greater left-sided detection performance. Second, there was some indication of emotional reactivity to the smells. The import of these findings would seem to be that while patient S could not reportedly experience a conscious olfactory percept, various sources of evidence – behavioural, emotional reactivity and imaging – suggested that the brain had detected and processed this olfactory information. But these findings were not sufficient to enable a conscious percept. As we noted earlier in the Sobel et al., paper [20], it would appear that structures within the inferior frontal gyrus, and notably on the right side and in orbitofrontal cortex, seem to be important in mediating conscious awareness of smell.
The second investigation by Zucco et al. [35] was also a single case study. In this case patient MB had had surgery for a meningioma. This resulted in the loss of the left olfactory bulb and the partial loss of the right olfactory bulb, in addition to damage to the left and right gyrus rectus and the left orbitofrontal cortex. After recovering from his surgery MB reported total anosmia. A post-surgical neuropsychological work-up indicated no generalised cognitive deficits, suggesting that the major complication resulting from the surgery was his anosmia. Further testing indicated intact taste identification, suggesting that MB was readily able to detect and identify chemosensory stimuli, suggesting that his anosmia was unlikely to reflect a more generalised chemosensory, cognitive or task-related deficit.

The key component of our testing involved presenting MB with three classes of stimuli; low-irritant odours, high-irritant odours, and blank stimuli. MB was asked to sniff each of the stimuli and to describe all his experiences, even to guess if necessary whether or what he might be smelling. For the high-irritant odours, namely those that stimulated both the trigeminal and olfactory systems, MB was above chance in noting that something was present, although he was not able to identify what it was. For the blanks, he never reported any type of experience. For the low-irritant odours a rather different pattern emerged. For around half of the low-irritant odours MB reported he could smell nothing even though he correctly identified three of these odourants. For the remainder, MB reported that he could still smell nothing, but he then qualified this by claiming some form of sensory experience, which was rarely enough to support identification. We suggest that this indicates something akin to blindsight, in that there is minimal olfactory consciousness (i.e., no “smell”), but there is some residual capacity to utilize this information, which in MB’s case involves a vague report of something present.

Both patient S and MB had damage to the right inferior frontal gyrus. There is at least one additional case report of localised damage within this region producing a profile similar in some ways to MB. Caminiti et al. [36] reported that a patient who had sustained a traumatic brain injury involving damage to the right orbitofrontal cortex was able to detect odours above chance, but was unable to discriminate them or identify them. This patient also identified herself as being completely anosmic, yet managed to retain some residual functionality that was seemingly independent of conscious olfactory experience. The common theme across these three cases is damage to the right inferior frontal gyrus.

6. Conclusion

Studies of the intact olfactory system seem to generate many examples of phenomena that share similarities with blindsight. These studies indicate that the brain can detect odourants when this is not accompanied by subjective and objective measures of conscious perception. Other studies indicate that liminal odourants can affect various psychological processes, without the participant being aware that they are being affected. Finally, and perhaps more directly paralleling blindsight, three case study reports indicate that odours can affect psychological processes (e.g., detection tasks), without subjective awareness. In each of these cases, a common theme is right-sided damage to the inferior frontal gyrus.

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