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Knowledge Without Awareness: An Autonomic Index of Facial Recognition by Prosopagnosics

Abstract. *Prosopagnosia, the inability to recognize visually the faces of familiar persons who continue to be normally recognized through other sensory channels, is caused by bilateral cerebral lesions involving the visual system. Two patients with prosopagnosia generated frequent and large electrodermal skin conductance responses to faces of persons they had previously known but were now unable to recognize. They did not generate such responses to unfamiliar faces. The results suggest that an early step of the physiological process of recognition is still taking place in these patients, without their awareness but with an autonomic index.*

Patients with prosopagnosia are unable to recognize visually the faces of persons they previously knew or ought to have learned without difficulty. They fail to experience any familiarity with those faces, and, even after they recognize the faces through other cues, such as voices, their physiognomies remain meaningless. Prosopagnosia is due to a complete failure to evoke memories pertinent to specific faces or to a defective evocation that fails to reach awareness. The condition is caused by bilateral damage to mesial occipitotemporal cortices or their connections.

Investigators of prosopagnosia have generally relied on the verbal report of the patient's experience as the sole index of recognition, an approach that does not address potential covert processes of which there may be no subjective awareness. In this study we used the electrodermal skin conductance response (SCR) as a dependent measure and found that two prosopagnosic patients generated significantly larger SCR's and responded more frequently to familiar faces than to unfamiliar ones (*1*). These results indicate that, despite their inability to experience familiarity with the visual stimulus and to provide verbal evidence of recognition, prosopagnosics still carry out some steps of the recognition process for which there is an autonomic index.

The subjects were two female patients with stable prosopagnosia caused by bilateral occipitotemporal damage, as determined from computerized tomography (CT) and nuclear magnetic resonance (NMR) imaging (*2*). We conducted several experiments. In each the patient was shown 50 black-and-white photographs of faces, depicting a full frontal pose on a white background (*3*). Forty-two of the faces were of persons entirely unfamiliar to the patient ("nontarget" faces) and eight were of persons with whom the patient was well acquainted ("target" faces). Both subjects were shown two sets of target faces selected from a period preceding the prosopagnosia (these target faces were randomly interspersed among the nontargets). In one of the sets, "family" faces, the target faces included those of the patient herself, family members, and close friends; in the other set, "famous" faces, the targets were famous politicians and actors. Subject 2 was exposed to a third set of target stimuli, "anterograde" faces, in which the targets were persons with whom the patient had had extensive contact since the onset of her illness but not before (physicians, psychologists, and so forth).

The subjects were given two presentations of each of the two sets of stimuli (or three sets, in the case of subject 2). During the first presentation skin conductance was recorded with Ag-AgCl electrodes from the thenar and hypothenar eminences of the nonpreferred hand on a Beckman type RM Dynograph recorder. Slides were presented for 2 seconds at intervals of 20 to 25 seconds. During the first viewing, no response was required of the subject; during the second, she was asked to verbally rate the familiarity of each face (*4*). Skin conductance was not recorded during the second presentation.

The results are presented in Table 1. As expected on the basis of her pervasive syndrome, subject 1 showed a complete failure to recognize any of the targets in the family and famous faces sets. Yet not only did she produce more frequent and consistent SCR's to the target stimuli, she also generated larger SCR's to the target faces than to the nontargets. The amplitude data were compared by the Mann-Whitney *U* test, a nonparametric test that avoids statistical assumptions not fulfilled by the data sets generated in this study. The average SCR amplitude for the target faces was significantly larger than that observed for the nontargets for both family faces ($U = 241$, $z = 4.01$, $P < 0.001$) and famous faces ($U = 265.5$, $z = 1.80$, $P < 0.05$) (*5*).

Subject 2 also evidenced more frequent and significantly larger SCR's to the target stimuli in the family faces ($U = 362$, $z = 4.63$, $P < 0.001$) and famous faces ($U = 204$, $z = 3.19$, $P < 0.001$) sets (Table 1), but, consistent with her lack of retrograde prosopagnosia, she also recognized accurately the familiar faces in these two sets. In the anterograde faces set, however, in which she was not able to recognize the target faces, she again produced more consistent and significantly larger SCR's to the target faces ($U = 283$, $z = 3.95$, $P < 0.001$). Thus this subject also showed a highly accurate autonomic index of recognition of familiar faces, despite a complete inability to experience familiarity with these faces and to recognize them formally.

The dissociation between the absence of an experience of recognition and the positive electrodermal identification may mean that in these subjects an early step of the physiological process of recognition is still taking place, but that the results of its operation are not made available to consciousness. Dissociations between overt recognition and unconscious discrimination of stimuli have been reported (*6*). Healthy subjects can show accurate autonomic discrimination of certain target stimuli, even when they are presented in a degraded or camou-

Table 1. Skin conductance response and verbal rating data for the two prosopagnosic subjects. For each category of faces (family, famous, and anterograde), two presentations of 8 target and 42 nontarget faces were made. The SCR data are based on the first presentation, while the verbal rating data are based on the second. Values in parentheses are standard deviations.

Subject	First presentation				Second presentation; average verbal rating (4)	
	Stimuli responded to (%)		Average SCR amplitude (μS)		Target	Nontarget
	Target	Nontarget	Target	Nontarget		
			Family faces			
1	71	12	0.934 (0.723)	0.048 (0.134)	6.0 (0.0)	6.0 (0.0)
2	100	36	1.660 (1.110)	0.146 (0.317)	1.0 (0.0)	5.1 (1.1)
			Famous faces			
1	63	12	0.731 (0.652)	0.012 (0.034)	6.0 (0.0)	6.0 (0.0)
2	63	19	1.080 (1.420)	0.022 (0.052)	2.6 (1.9)	5.0 (1.2)
			Anterograde faces			
2	75	17	0.345 (0.274)	0.022 (0.060)	4.4 (1.8)	4.6 (1.7)

flaged manner that precludes overt discrimination and identification (*7*). There is some parallel between such findings in healthy individuals and the observations described above, even if the mechanisms that lead to failure of recognition are different. Our results are also compatible with those of a recent study of prosopagnosia in a single patient, who showed discriminatory electrodermal responses to correct but not incorrect face-name matches (*8*).

We will attempt to interpret this "covert" recognition phenomenon in terms of a model of facial learning and recognition (*9*). The model includes step 1, perception; step 2, use of a template system, in which dynamic intramodal records of the elaboration of past visual perceptions of a given face can be aroused by the perception of that face (*10*); step 3, activation, in which multiple multimodal memories pertinent to the face are evoked; and step 4, a conscious readout of concomitant evocations that permits an experience of familiarity and either a verbal account of that experience or the performance of nonverbal matching tasks (*11*).

Prosopagnosia cannot be explained as being due to an impairment of the basic perceptual step (numerous indices of visual perception are normal, and patients are able to match unfamiliar faces and describe separate visual details of the faces). Nor can it be explained by an impairment of associated memories because they can be easily evoked through other channels. The defect may be explained, however, by an impairment of the activation step, which would either not take place or take place inefficiently. That, in turn, might be due to a dysfunction of the template system, which could be (i) intact but inaccessible to ongoing percepts, (ii) destroyed, or (iii) intact but prevented from activating multimodal memory stores. From the evidence above, it appears that facial templates are intact: the electrodermal "recognition" can be interpreted as being an index of successful matches between percepts, that is, correctly perceived target faces, and templates of those faces. Furthermore, the data on subject 2 suggest that, with respect to newly encountered faces, the process of template formation can proceed automatically in the absence of normal recognition processes.

The prosopagnosia of the two subjects can be viewed as a complete or partial blocking of the activation that normally would be triggered by template matching. From the anatomic specifications of the model (*11*), it appears that the blocking occurs either in white matter connections of the occipitotemporal region (linking both visual cortices to anterior temporal cortices, and the latter to multimodal sensory cortices) or in anterior temporal cortices.

According to the model, the findings presuppose the intactness, at least unilaterally, of the primary visual cortex and the inferior and mesial visual association cortex. Anatomic analyses of images of both patients obtained by CT and NMR verify these predictions (*2*). It is of great interest that the lesions that block activation of associated memories do not block the autonomic response. The anatomic substrates of the autonomic response remain to be elucidated.

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References and Notes

1. We previously showed, using the SCR as a dependent measure in neurologically intact subjects, that familiar faces have a notable "signal value" (D. Tranel, D. C. Fowles, A. R. Damasio, *Psychophysiology*, in press).
2. Subject 1 has both anterograde and retrograde prosopagnosia (she cannot recognize any faces that were familiar before the condition developed, including her own, nor has she learned any new faces during the 7 years of her prosopagnosia). She is a 62-year-old, right-handed woman who suffered bilateral strokes involving the occipitotemporal region. The lesion involves the white matter of both occipitotemporal regions but spares the most mesial and rostral regions of the inferior visual association cortex. As determined by a comprehensive neuropsychological assessment, her intellect, language, and visual perception are normal. Subject 2 has anterograde prosopagnosia only (she has not learned any new faces since the onset of her prosopagnosia 3 years ago). She is a 20-year-old, right-handed woman who had herpes simplex encephalitis leading to bilateral lesions of the occipitotemporal region. The lesions are located more anteriorly than in subject 1, so they also spare the mesial and rostral aspects of the inferior visual association cortex. Her language abilities are intact and her visual perception is compatible with normal recognition of faces learned before the onset of her illness.
3. The slides were constructed so that all the faces were of similar size. No slide contained features below the neckline, and no clothing was seen. The nontarget faces were selected so as to be similar to the targets in terms of age range and sex ratio. Brightness, contrast, and resolution were comparable in target and nontarget faces.
4. A six-point rating scale, ranging from "certain familiarity" (1) to "certain unfamiliarity" (6), was used. Ratings of 3 or less indicated some degree of familiarity with, or recognition of, the stimulus; conversely, ratings of 4 or greater indicated that the subject did not recognize the stimulus.
5. All *P* values are corrected for ties (data points with equal values), as recommended by S. Siegel [*Nonparametric Statistics for the Behavioral Sciences* (McGraw-Hill, New York, 1956), pp. 123–126].
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10. The template system, which involves a dynamic process sensitive to the acquisition of new sensory information, permits the categorization of normal percepts according to physical structure. These dynamic templates do not contain information about the identity of a particular face. The latter information is stored not in a single site but rather in various sensory association cortices, including the visual, in both nonverbal and verbal forms. The templates serve as an interface between the repeated perception of a face and the multiple stored traces of information pertinent to the face; that is, the perception of a previously known face matches the respective template system, which in turn activates the pertinent multimodal memory stores.
11. The anatomic substrate of step 1 comprises bilateral visual system structures up to and including the primary visual cortex; the anatomical basis of step 2 is focused on bilateral mesial and inferior visual association cortices; the anatomic basis of step 3 includes bilateral anterior temporal structures, both mesial and lateral, and bilateral association cortices of different sensory modalities (*9*). Step 4 depends on the same association cortices.
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