FACIAL RECOGNITION 1990[1,2]

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It is appropriate at the outset of this Symposium to pay tribute to the two clinicians who, in the 1860's, first described and emphasized the significance of the loss of the capacity for facial recognition that we now call "prosopagnosia". Both were opthalmologists: Antonio Quaglino, who was a professor at the University of Pavia, and Giambattista Borelli, who was a practicing ophtalmologist in Turin. In 1867, they published a paper describing and discussing what they considered to be a remarkably instructive patient who had come under their care (Quaglino and Borelli, 1867).

Their patient was a 54-year-old man who had sustained a right hemisphere stroke that initially produced a left hemiplegia and an apparently complete loss of vision. The hemiplegia disappeared within a month and his vision gradually improved so that a year after the stroke he had a left visual field defect but excellent central vision. As the patient himself said, he "could shoot a bird out of a tree".

When he was seen a year after his stroke, his main complaints were:

(1) A loss of color vision. All objects and faces looked pale and colorless to him. Examination disclosed that he was indeed color-blind. In short, he showed a bilateral central achromatopsia.

(2) Some impairment in spatial orientation.

(3) Impairment in facial recognition; he could no longer recognize the faces of familiar persons and would try to identify them by their voices. In addition, apparently he could no longer recognize familiar houses.

Quaglino and Borelli used their case report to argue for the concept of localization of cerebral function. They diagnosed their case as one of cerebral hemorrhage involving the right hemisphere primarily but probably extending to the left hemisphere. They clearly recognized that their patient's facial agnosia and achromatopsia were the result of brain disease and not of some ocular condition. The first reference to their paper was made about 15 year later by a Ger-

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[2] Dedicated to Professor Carlo Loeb on the occasion of his retirement.

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man ophtalmologist, Mauthner (1881). Limitations of time make it necessary to restrict the scope of this presentation. It is focussed almost entirely on the implications of recent studies of prosopagnosia and impaired discrimination of unfamiliar faces in patients with brain disease. The many laboratory studies of normal subjects, which have produced such a wealth of intriguing findings, will not be dealt with. These findings are so numerous and varied that it is simply not possible to summarize them in any meaningful way in the course of a brief review. In this sense the title of the presentation is a misnomer. A truly comprehensive review would include a detailed consideration of both clinical and experimental observations as well as an attempt to integrate them into a coherent unity.

Ten years ago I assessed our understanding of facial discrimination and identification, as well as the disturbances in these capacities that one encounters in patients with brain disease (Benton, 1980). Five years later, I attempted another integrative summary (Benton, 1985). Now we find that, in the past few years, interest in the subject certainly has not abated. In fact, it has intensified with 30 to 40 papers on diverse aspects of facial recognition appearing each year. One reason for this surge of interest in a topic that not very long ago was of concern to only a few neurologists and neuropsychologists is that, in addition to its clinical relevance, analysis of the processes involved in facial recognition promises to offer important clues about how the brain integrates incoming sensory information and past experience to achieve specific perceptions and their specific meanings. A second reason is, of course, that people's memory for faces is a quite extraordinary human capacity. That we should be able to recognize a person whom we met on only one occasion 20 years ago (and this in spite of the changes in his facial features that have occurred with advancing age) is truly remarkable. Hence, facial recognition and, particularly, long-term memory for faces, is a phenomenon of great psychological interest in its own right. (Because of their preoccupation with brain-behavior relationships, neuropsychologists sometimes forget that not all behavioral science is neurobehavioral science.) Still another reason for our continuing interest is that questions about the anatomopathologic basis of prosopagnosia and about the nature of the cognitive disabilities underlying this curious failure in recognition still persist and, therefore, clinical investigators have continued to address them.

In 1980, I had occasion to note that confusion in the field was engendered by the failure to distinguish between impairment in the identification of familiar faces (prosopagnosia) and impairment in the discrimination of unfamiliar faces despite the fact that the two defects had been demonstrated to be separable and independent (cf., Warrington and James, 1967; Benton and Van Allen, 1972). Although most researchers now respect the distinction, this is not always the case and some degree of confusion remains. This is not to say that the two types of impairment may not be related to each other in some cases of prosopagnosia. However, to treat them as if they were the same disability is surely a mistake.

The debate continues about whether or not a unilateral right hemisphere occipitotemporal lesion is sufficient to produce prosopagnosia. The elements of this debate are familiar. Clinical studies almost invariably indicate that the deficit is accompanied by signs of right hemisphere disease such as left visual field-

defect and visuospatial impairment. Yet, autopsy results are consistent in their indications of bilateral damage (cf., Lhermitte et al., 1972; Meadows, 1974; Benton, 1980; Damasio, Damasio and Van Hoesen, 1982; Nardelli et al., 1982). In addition, before 1985, most CT studies generated findings supportive of a bilateral basis for prosopagnosia, the few exceptional cases being flawed in one or another respect. Hence at that time, I was inclined to "accept the conclusion that bilateral disease is the necessary context for the occurrence of a persisting prosopagnosia" (Benton, 1985, p. 225).

However, since 1985 a substantial number of CT-scanned cases of prosopagnosia with lesions apparently confined to the right hemisphere have been described (cf., Torii and Tami, 1985; De Renzi, 1986a, 1986b; Landis et al., 1986; Michel et al., 1986). There is also the autopsied case of Landis et al. (1988) of a prosopagnosic patient who died 10 days after her stroke and in whom the post-mortem findings were a large infarct in the occipitotemporal region of the right hemisphere, an older infarct in the right frontal lobe and a older very small infarct in the left occipitoparietal region. The patient showed clinical signs of right hemisphere disease with no indications of left hemisphere involvement. Although the authors considered that their case represents one of prosopagnosia produced by a unilateral lesion, the evidence provided by it must still be regarded as somewhat equivocal, since one cannot be certain that the patient would have suffered from a persisting prosopagnosia if she had survived longer and, strictly speaking, she did have a bilateral lesion. But some of the autopsied cases that are cited in favor of a necessary bilateral involvement are even more equivocal. Finally, there is the recent case report of Sergent and Villemure (1989) describing a patient who had undergone a right hemispherectomy in early life and who was prosopagnosic.

Thus, the weight of evidence has shifted in favor of regarding prosopagnosia as a disability that *can* be produced by a right hemisphere lesion alone. In any case, the integrity of the right inferior occipitotemporal area, where conceivably visual information is transmitted to the mesial and inferior temporal region on the way to being linked up with a memory store, appears to be a crucial juncture, injury to which leads to impairment in the recognition of familiar faces. This is not to deny that the neural mechanisms underlying the identification of familiar faces may be bilateral in nature; in all probability, they are. But the crucial lesion that produces disability, like the lesion of aphasia in righthanded persons, is unilateral.

This is as far as one (or rather, as far as I) can go at this time. The formulation is not very satisfactory. There are too many unswered questions. Why is prosopagnosia an uncommon disability? Although it is not as rare as was once thought, it is sufficiently uncommon that almost every case that is encountered is written up for publication. And the Sargent-Villemure hemispherectomized prosopagnosic case must be balanced against earlier reports of patients who were *not* prosopagnosic after right hemispherectomy (cf., Smith, 1969; Damasio, Lima and Damasio, 1975).

From the evidence at hand, it appears that a right inferior occipitotemporal lesion is a crucial but not a sufficient determinant of prosopagnosia. One possibility is that such a lesion disrupts the neural network underlying facial identification only in combination with some atypical condition of the left hemi-

sphere such as might result from acquired disease, congenital weakness or perhaps age-related decline in functional efficiency. A second possibility, which has been advanced by De Renzi (1981b), is that the right hemisphere lesion must attain a certain size if it is to be effective. One might add that perhaps not only must the lesion attain a certain critical size but must also not exceed a certain critical size because a massive lesion of the right hemisphere might have such devastating effects on visual perception and directed attention as to destroy the specificity of a patient's prosopagnosia. Such a dual limiting condition with respect to size of lesion might account for the infrequent occurrence of the complaint of a specific prosopagnosia (cf. Damasio, Damasio and Van Hoesen, 1982). The concept of individual differences in hemispheric specialization has been invoked as a possibility. However, this concept remains entirely speculative. Thus, although we accept the proposition that a unilteral right hemisphere lesion can produce prosopagnosia, we do not know the overall neurological context within which this occurs.

A procedural note is perhaps in order here. The combination of prosopagnosia and achromatopsia, which Quaglino and Borelli observed, is known to be a very frequent occurrence. This is not unexpected since the focal lesions producing the two disabilities are located in the same territory, namely, the inferior occipital region. Of course, the association is not obligatory; there are prosopagnostic patients who are not color-blind and achromatopsic patients who are not prosopagnosic. However, the presence of complete achromatopsia does indicate bilateral inferior occipital disease (providing, of course, that the patient is not congenitally color-blind or has some ocular condition that impairs color vision). Hence, systematic examination of a patient with agnosia for faces should always include assessment of his color vision. To be sure, neither a positive nor a negative finding in this respect would be absolutely decisive for concluding whether or not the patient's prosopagnosia was caused by a unilateral or a bilateral lesion. But it would shift the weight of evidence in one or the other direction. Thus the presence of a complete achromatopsia would not exclude the possibility that a patient's facial agnosia was produced by a right hemisphere lesion but it would make the possibility less likely. Conversely, intact color vision or a left hemiachromatopsia would favor a unilateral lesional basis for the impairment in facial recognition.

Turning to the psychological aspects of facial recognition, De Renzi (1986a, 1986b) has described a number of personally studied cases that support the validity of distinguishing between perceptual and amnesic forms of prosopagnosia. Stated in other words, it is useful to consider a patient's failure to recognize familiar faces as an outcome, as a failure in performance, that may result from one or another type of cognitive disabilty. The perceptual prosopagnosic will show prominent defects on visuoanalytic and visuospatial tasks while the amnesic prosopagnosic patient will show only minor defects in this respect.

This distinction, which goes back to the pioneer study of visual object agnosia by Lissauer (1890), applies to many types of agnosia; for example, to astereognosis and sound agnosia, as well as to facial and visual object agnosia. In this connection, it is worth recalling that Lissauer, who is credited with first making this distinction (or blamed for it, depending upon one's point of view),

actually did not postulate two *types* of mindblindness, apperceptive and associational. Instead, he emphasized that the achievement of recognition involves the interaction of perceptual and associational processes. Perception engenders relevant associations and these associations focus and sharpen perception. Thus, each process contributes to recognition at some point (or points) in its course. Thus, in Lissauer's view, one could speak of visual agnosia that was largely perceptual in nature or largely associational in nature but *not* of discrete types of agnosia. He thought that his own patient had primarily an associative visual agnosia because this somewhat demented old man could copy designs accurately and also correctly discriminated between similar visual patterns. Lissauer's position, which fits in well with modern concepts, has meaning for us today. If we accept it, we will abandon the concept of types of prosopagnosia but instead think in terms of the stage in the course of parallel and interactive information processing at which disruption may have occurred. In this regard, we should keep in mind that the autonomic and covert recognition studies of Bauer (1984; Bauer and Verfaellie, 1988), Tranel and Damasio (1985, 1988) and De Haan, Young and Newcombe (1987) have described still another dimension in facial identification, in addition to perception and association.

De Renzi's incisive review also considered the question of specificity. The form in which the question was usually posed 15 years ago was: How distinctive a deficit is prosopagnosia; is it an autonomous deficit or is it only a particular expression of a more pervasive disability in perception and/or memory? However, subsequent clinical studies of prosopagnosic patients involving assessment of their performances on diverse cognitive tests have indicated that their disability often is highly specific. While such a patient may show mild or moderate impairment in performance on these other tasks, it does not seem reasonable to infer that the observed impairment could account for his gross failure in facial identification. In any case, many patients who show even more severe impairment on these other tasks prove not to be prosopagnosic. Thus Bodamer (1947), who coined the term "prosopagnosia" and who espoused an extreme view about the special character of facial recognition, has been vindicated to some degree. At least we are nearer to his position than we were 15 years ago when most of us refused to accept his rather romantic description of the unique status of facial recognition as a cognitive process.

But, if prosopagnosia is a more or less specific disability, how is it to be explained in psychological terms? A longstanding formulation, first advanced by Faust (1955) and more recently explicated by Damasio, Damasio and Van Hoesen (1982), is that it consists in a failure to identify individuality within a set of similar objects comprising a class such as men or women, automobiles, neckties, etc. This is more or less the situation of the prosopagnosic patient. He knows that the face he is looking at is a face; he knows that it is a face of a man or a woman; and that it is the face of an old person or a younger person. But he cannot tell *whose* face it is. Thus, on this view, prosopagnosia is a restricted but not absolutely specific disability.

In attempting to evaluate this reasonable hypothesis, let me cite a bit of history. In 1870, Finkelnburg advanced the idea that aphasia was not a mere instrumental disorder of language but instead a partial expression of a more ge-

neral impairment in symbolic thinking to which he gave the name "asymbolia". To support his idea, he cited some of his cases. One patient could not longer understand the value of different coins; another could no longer understand the meaning of insignia of rank (which was a distinction of considerable importance in 19th century Germany); still another patient could not identify environmental sounds such as the ringing of a bell or the barking of a dog; still another failed to grasp the meaning of pantomimed actions. What Finkelnburg failed to specify is whether the patient who had lost the capacity for understanding the monetary value of different coins also failed to understand pantomimed actions, insignia of rank and environmental sounds; or whether the patient who could not comprehend the meaning of pantomimed actions failed the other tasks as well. Perhaps he presented only one task to each patient but this seems unlikely. More probably, the patients succeeded on these tasks, but since their success was not relevant to his concept, he mentioned only the single instance of failure in each case.

As De Renzi (1986b) has pointed out, essentially the same state of affairs hold for the "identification of individuality" hypothesis in prosopagnosia. Faust's patient could not discriminate between ordinary chairs and armchairs; the patient of Damasio, Damasio and Van Hoesen could not identify her automobile until she read the license plate; still another patient could not discriminate between domestic and foreign coins (Blanc-Garin, 1984). But, so far as one can see, in most cases this was the only disability in the "identification of individuality" that each patient showed. Logically, the "identification of individuality" hypothesis demands that patients be tested systematically on a number of relevant tasks and that confirmation of the hypothesis should be based on the finding that they fail a considerable number of these tasks and not just one or two. In fact, De Renzi (1986b) has described such a systematic procedure that was applied to one of his prosopagnosic patients with, as it happens, negative results; the patient behaved quite normally on such tasks as identifying his own handwriting among a set of distractors, his own razor, and Italian coins from foreign coins. However, other prosopagnosic patients might well show this type of disability and thus conform to the hypothesis.

Finally, it is worth mentioning that a hitherto unrecognized form of prosopagnosia is the subject of ongoing studies by Tranel and Damasio (1989). It is developmental prosopagnosia, an inability to recognize the identity of familiar faces that is presumably present from birth or the early years of life. The disability is usually at least moderately severe and it can be the basis for disturbed and embarrassing social interactions. The affected individuals are aware of their disability and try in various ways to compensate for it. It is, in some ways, analogous to developmental dyslexia and it bears the same tenuous relationship to acquired prosopagnosia that development dyslexia bears to acquired alexia. The case material is characterized by a predominance of males and by an excessive number of lefthanders; these are associations that have not been observed in acquired prosopagnosia. The subjects are typically of above average intelligence; however, this may be an artifact of case selection. Initial findings are that these subjects often show defects in color perception and some difficulty in recognizing the identity of stimuli other than faces. Some subjects complain of

difficulty in reading although, at least to date, none have proven to be severely dyslexic. This is about all that is known about the condition at this time. The chances are that developmental prosopagnosia, like acquired prosopagnosia, is an uncommon, but not a rare, defect.

Turning to the area of the discrimination of unfamiliar faces, I should like to comment on the perennial topic of hemispheric asymmetry in facial discrimination. Clinical observation has continued to support the finding that it is primarily the non-aphasic patient with posterior right hemispheric disease who shows this disability. We infer from this that the right hemisphere is to some degree specialized with respect to the operations involved in the discriminaton. An extension of this inference has now come from animal experimentation. As we know, rhesus monkeys can easily learn to discriminate between the representations of the faces of monkey (cf. Rosenfeld and Van Hoesen, 1979; Hamilton and Vermeire, 1983). The recent studies of Hamilton and Vermeire (1988, 1990) have demonstrated that split-brain monkeys show a significant left visual field superiority on the task. Thus it is clear that right hemisphere superiority for facial discrimination is not a unique characteristic of the human brain.

In 1979, Hamsher, Levin and Benton reported that, while among non-aphasic patients with unilateral disease, those with right hemispheric lesions account for most of the failures on a facial discrimination task, there is a subset of left-hemisphere damaged aphasic patients with significant impairment in oral comprehension who also show a high frequency of defective performance. If this finding is confirmed and if the failure of these aphasic patients was not due to lack of understanding of the task instructions (we believed that it was not), this observation has important implications for our understanding of the neural mechanism underlining facial discrimination. However, so far as I am aware, there have been no attempts to cross-validate this finding. Aphasic patients have been included in some recent studies but they have not been separated into subgroups with and without significant defects in oral comprehension.

Finally, I should like to mention a study dealing with an unusual application of a test of facial recognition, namely, in the field of diagnostic pathology. Perhaps it will come as a surprise to some people to learn that there is considerable variation in the ability of pathologists to detect subtle abnormalities in surgical specimens of tissue and to identify the nature of those abnormalities. Consequently, postgraduate training programs in pathology that take their mission seriously are concerned with this variability which does not seem to be correlated either with general intellectual level or with performance in medical scool. The capacity to inspect a small specimen of tissue and correctly interpret what one sees (or does not see) makes demand on perceptual acuity, the ability to isolate significant features and the ability to detect invariance within different contexts.

Berbaum and Platz (1988) reasoned that, since our test of facial recognition makes such perceptual demands, it might be useful both for selecting candidates for training in pathology and for identifying trainees who need special attention. They therefore undertook a study to determine the relationship of the test of facial recognition to the performances of residents in pathology. Ratings of the performances of the residents by members of the faculty who supervised

them served as the criterion measure. The relationship of two predictors of performance, as measured by these ratings, was investigated. One was a slide test employing surgical tissue specimens which was part of a national examination in pathology. The other consisted of the nine most difficult items (generating 27 responses) in the test of facial recognition.

The Berbaum-Platz findings were as follows: The correlation between scores on the slide test and the ratings was .79 while the correlation between scores on facial recognition test and the ratings was .70. The correlation coefficient between scores on the slide test and the facial recognition test was .49. It should be kept in mind that the slide test presupposes considerable knowledge of human pathology while the facial recognition test does not.

It is interesting that a procedure that was designed to detect a deficit which is related to focal brain abnormality could also be useful for detecting a special talent. In passing, it may be noted that this reversal in the purpose to which a procedure is put is not altoghether unique in neuropsychology. The anatomist, Oskar Vogt, who developed a highly differentiated architectonic map of the cellular structure of the cerebral cortex, once tried to correlate specific cognitive abilities with specific architectonic areas and to provide an anatomic basis for individual differences in talent (cf. Vogt, 1951).

It is clear that some significant advances in our knowledge of facial recognition and its derangements have been made during the past decade. In turn these advances point to the questions that now need to be addressed in order to achieve a deeper understanding. To paraphrase an observation once made by Winston Churchill — this is not the end or even the beginning of the end but perhaps it is the end of the beginning.

Abstract

A review of recent studies of prosopagnosia suggests that the weight of evidence has shifted in favor of regarding it as a disability that can be produced by a right hemisphere lesion alone even though bilateral disease remains the more frequent anatomical basis. It is possible that prosopagnosia resulting from a right hemisphere lesion occurs only within the context of some atypical condition of the left hemisphere. "Types" of prosopagnosia continue to be postulated and the "identification of individuality" hypothesis continues to be advanced. Autonomic and covert recognition studies of prosopagnosic patients have described a new dimension in facial identification. Right hemisphere dominance for the discrimination of unfamiliar faces in non-aphasic patients has been confirmed but the performances of left-hemisphere damaged aphasic patients has still not been fully investigated. New developments include the study of developmental prosopagnosia and novel applications of test of facial discrimination.

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