Improbable Areas in Color Vision

From: Visual Neurosciences (L. Chalupa and J. Werner, eds.)

Final version submitted: February 2002

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Introduction

Color vision is an exciting subject, that lends itself easily to theorizing. It is especially interesting to do so in relation to the human cerebral cortex, given the huge advances made in the past decade in imaging human brain activity in health and disease. It is also interesting to do so because understanding how the brain constructs colors promises to give significant insights into understanding the cerebral processes underlying aesthetics in the old Greek sense, that is to say the acquisition of knowledge through the senses. It is not surprising to find therefore that color has traditionally attracted the attention of all those who have been concerned with perception and with knowledge, including philosophers and physicists. But theorizing, to have any value, must be based on reliable facts and interpretations. How reliable are the facts derived from imaging experiments relating to color vision? And how compelling are the interpretations based on these results?

This paper deals with the quality of evidence that has been used to erect three cortical areas in the human brain, “VP”, “V4v” and “KO”, all of them related directly or indirectly to color vision. How solid is the evidence for the existence of these areas and how convincing is the interpretation of their functions? Let me emphasize that I am discussing the human brain, not the primate brain in general, though I shall refer of course to the evidence from monkeys from which some of the human areas are etymologically derived. Any conclusions reached about human visual areas must be derived from, and consistent with, evidence obtained from the human brain, without recourse to evidence from the monkey brain or from any other species. It is of course always good if the human evidence is supported by monkey evidence, but it should not have to rely on it. Too often in the recent past there has been an attempt to hide behind a monkey in pleading for an interpretation, because that interpretation is not sustainable through human evidence alone. This paper amounts, therefore, to an enquiry into the quality of evidence that we
have come to accept in human imaging studies, a quality that I believe falls far short of what the instrumentation that we rely on is capable of delivering.

**The location of the human cerebral color center and the visual field representation within it**

The notion that colour may be the function of a specialized cortical area was hinted at several times before Louis Verrey (1888) published his paper entitled *Hémiachromatopsie droite absolue* (absolute right hemiachromatopsia). Verrey’s advantage over his predecessors was that he had been able to examine the lesion that had led to the syndrome of acquired achromatopsia (cerebral colour blindness). Apart from some involvement of the body of the corpus callosum, the lesion was confined to the fusiform and lingual gyri, and was thus located in the inferior part of the occipital lobe (Figure 1). Every word of Verrey’s title, together with the figure representing

![Figure 1: The sites of the lesions in the brain examined by Louis Verrey (reproduced from Verrey, 1888).](image)

the lesion in the brain of his achromatopsic patient, is worth studying. Together they tell a great deal about the organization of the visual brain even today, though much of this is not mentioned by Verrey and is read by me into his evidence with hindsight. Foremost among the lessons to be learned is something about the topographic organization of the human visual brain. Given that the
lesion producing cerebral hemi-achromatopsia was in the lingual and fusiform gyri, the title implies that a center located in the lower part of the occipital lobe controls color vision in both the upper and lower parts of the entire contralateral visual hemi-field.

The discovery of a visual area lying outside the striate cortex was an embarrassment at the time (see Zeki 1990; 1993 for reviews). Henschen and after him Holmes had concluded, correctly, that the lower part of the calcarine cortex (V1) represents upper visual field and the upper part the lower visual field. They had concluded, also correctly, that the calcarine cortex, which Henschen referred to as the ‘cortical retina’ and Holmes as the ‘visuo-sensory’ cortex, and which we now call the primary visual cortex or area V1, was co-extensive with the striate cortex. This led both Henschen and Holmes to conclude, incorrectly, that V1 was the only visual center in the brain, and therefore had to receive all visual ‘impressions’, including color impressions (see Zeki 1993 for a review).

From his single case of achromatopsia, Verrey had concluded, correctly, that there is a color center in the brain but had also supposed, incorrectly, that this is part of the ‘visuo-sensory’ cortex. The implication was obvious: that the ‘visuo-sensory cortex’, or the ‘cortical retina’ of the brain was larger than that supposed by Henschen and by Holmes, i.e. was not confined to the striate cortex. Verrey implied that the primary visual receptive cortex had a specialized subdivision dealing with color. Verrey’s conclusions on the cortical site for color processing [le centre du sense chromatique], together with his observation that both upper and lower contralateral quadrants were compromised in his unilaterally lesioned patient, implied that both quadrants are mapped in the lower part of the occipital lobe. Such an implicit supposition (which Verrey himself did not explicitly make) obviously cast a doubt on the way that Henschen and Holmes had supposed the visual field is mapped in the occipital lobe. Both Henschen and Holmes dealt with this in the same way, by brushing aside Verrey’s evidence or ignoring it altogether, until it vanished from the literature (for reviews see Zeki 1990, 1993).
There was a price to be paid for ignoring the significance of the finding that an area located in lower occipital cortex controls color vision in both contralateral quadrants. That peril was with us until well into the 1990s. Actually, it is probably with us even today. This is surprising because Damasio et al. (1980) alluded to it explicitly. They wrote: “...one single area in each hemisphere controls color processing for the entire hemifield. This is so regardless of the fact that such an area is eccentrically located, in the lower visual association cortex, classically related to upper quadrant processing only...The classic concept of a concentrically organized visual association cortex no longer appears tenable”. No one took much notice of what they said then, and I don’t think that anyone takes much notice of it today either.

There is probably a good explanation for this intellectual scotoma. With time, and the discovery that ‘areas 18 and 19’ (Brodmann 1905; von Bonin and Bailey 1951), or areas V2 and V3 (Cragg 1969; Zeki 1969) form concentric rings around V1 (Figure 2), it became customary to consider that the upper part of the visual field is represented in lower occipital lobe and vice versa. A new habit developed, of tagging on the “v” or the “d” to areas, to indicate that they are located in lower occipital lobe (“v”) and therefore represent upper fields or that they are located in upper occipital lobe (“d”) and therefore represent lower visual fields, respectively. This terminology begged for confusion and trouble, which was not long in coming. It was based on an implicit assumption that had already been discounted, namely that the upper contralateral quadrant is represented in lower occipital lobe, and vice versa. Verrey (1888) had shown that an area located in lower occipital cortex must represent both contralateral quadrants (otherwise how would one account for the complete contralateral hemi-achromatopsia produced from a unilateral lesion in lower occipital lobe?), physiological evidence had shown that that an area located in upper occipital lobe can represent both contralateral quadrants, as does area V3A (Van Essen and Zeki, 1978) or that it may be located somewhere between the two halves and still do the same, as does area MT in the owl monkey (Allman and Kaas 1971). Indeed, Allman and Kaas had
Figure 2: Brodmann’s cytoarchitectonic map of the brain. Brodmann numbered the areas according to the sequence in which he studied them. Note how areas 18 and 19 form rings around area 17 (from Brodmann, 1909).

Introduced the more neutral designations of + and – to indicate upper and lower field representation, respectively, without making either explicit or implicit assumptions about whether upper contralateral quadrants are only represented in lower occipital lobe (see Baker et al. 1981). It is unfortunate that their more sensible designations have not been more widely used. If they had, we may have avoided some of the present difficulties, though even that is not certain. At any rate, with implicit assumptions imposing themselves, if an area was found in lower occipital cortex, the “v” was hastily tagged on to it, irrespective of whether a dorsal counterpart could be found for it; if located in upper occipital lobe, the “d” was tagged on to it, again irrespective of whether a ventral counterpart could be found for it. It was but one step from this to describing
what Jon Kaas (1993) has called “improbable” areas, ones that represent only one quadrant, with no representation for the other corresponding quadrant. But what kind of visual information is restricted to only one quadrant that it alone should have a cortical representation, without a companion area to represent the same information for the ‘unrepresented’ quadrant? Assuming that the proponents of this bizarre terminology thought about it at all, they were not telling. A good example related to color vision is provided by area “VP”.

**An improbable area, “VP”**.

Area V2 of the primate brain surrounds area V1 and is in turn surrounded by area V3. The latter has a dorsal and a ventral subdivision, just like V2 (Figure 3). Throughout its dorsal and ventral extent, the representation of the horizontal meridian forms a common boundary between it and V2, and the representation of the vertical meridian is at its anterior border (Cragg 1969; Zeki 1969). In 1986 a number of papers from Van Essen’s group, summarised in Burkhalter et al. (1986), confirmed the manner in which the visual field is mapped in V3 (see also Shipp et al., 1995). They nevertheless reported that the lower part of V3 does not receive a direct input from V1, unlike the upper part of V3. They also reported that the lower part of V3 contains a high concentration of color cells, again unlike upper V3. This led Van Essen to propose that the lower part of V3 is not part of V3 at all, but a distinct area that they called ‘VP’. This made of ‘VP’ one of Kaas’ “improbable areas”, for it implied that something happening in upper quadrants (in this case color, *inter alia*) is processed there, without a machinery for processing that same attribute when it occurs in lower quadrants. Also unaccounted for was why, among the areas that process color, ‘VP’ alone should process this attribute in upper visual fields only. Such an asymmetric representation is not characteristic of V1, or V2, or V4 – all of them involved in color processing. Burkhalter et al (1986) gave a half-hearted explanation. They wrote unconvincingly: “any asymmetries relating to V3 and VP might well be cancelled by compensatory asymmetries in
In the absence of any direct evidence in favor of a dichotomy between V3 and VP in the human, and evidence against it instead, there seems little doubt that the habit of naming the ventral part of human V3, “VP” is inherited from the macaque monkey. But how reliable is the macaque evidence? The negative anatomical evidence regarding the absence of a direct input from V1 to lower V3 in the macaque is not only doubtful but probably wrong. Such a projection has been found in Cebus (Rosa et al., 2000) and Lyon and Kaas have recently found the same not only in the marmoset monkey but also in the macaque monkey (Lyon and Kaas 2001; 2002). It is
instructive to compare the approach of Lyon and Kaas with the approach adopted by some in the imaging community. In their 2001 paper, Lyon and Kaas postulated that a V3 with upper and lower subdivisions would be characteristic of all primate visual brains. But they also tested the postulate by studying the macaque monkey (Lyon and Kaas 2002). In the brisk world of human imaging experiments, this expensive and time devouring luxury is seemingly not an option for everyone. Instead, what we have witnessed is the identification of a visual area in the human brain based on questionable evidence from the macaque. This is unfortunate. Given the real differences between species, the identification of human visual areas should be based on human measurements.

The anatomical evidence in favor of the absence of a direct input from lower V1 to lower V3 in the monkey is therefore far from convincing and indeed all the evidence speaks against it. The physiological evidence that, unlike V3, there is a high concentration of color cells in ‘VP’ is even less convincing, indeed all human imaging studies speak against it too. These studies have succeeded in showing that there is an area lying anterior to V2, with the map characteristic of V3, but not a single one of them, even those that use the term 'VP' for lower V3, has succeeded in showing any specialization in ‘VP’ for color, or any specific activation of ‘VP’ with color stimuli (see for example Zeki et al., 1991; Sereno et al., 1995; De Yoe et al., 1996 among others). Where the question has been specifically addressed, it has been found that lower V3 is not specifically activated by color (Wade et al. 2002). On the other hand studies have found that ‘VP’ is activated in the same way as V3 (see for example Smith et al., 1998). In spite of this, many persist in this folie à plusieurs, of calling the lower part of V3 ‘VP’, oblivious to the fact that to have an area representing one quadrant without having a representation for the other quadrant, makes of it an improbable one. Improbable, but also possible. But if the latter, it needs a convincing explanation, which no one has yet provided. Instead, the precedence has been used to describe other improbable areas, as we shall see. Thus the evidence from the human brain does not support a separation into VP and V3, but rather tells of an area V3 with upper and lower subdivisions,
representing lower and upper visual fields, the two subdivisions being activated in the same way. It is of course right to record that not all have accepted this division into V3 and ‘VP’ unquestioningly (see for example Wandell 1999).

Another improbable area, “V4v”

Our early imaging experiments showed that the color center in the human brain is located in the fusiform gyrus (Lueck et al., 1989; Zeki et al., 1991). It was called V4, without attaching a “v” or a “d” to it. At the time, this was not unreasonable. We had used full field stimulation and had no means of distinguishing upper from lower visual field representation within it. Unless one wanted to study its topography, which was not our aim, there was little need for us to do otherwise. We had read Verrey’s paper, and remembered its title, which indicated that both upper and lower quadrants are mapped in the ‘color’ center, located for him in the ventral part of the occipital lobe. This, we therefore supposed, was an area in which both quadrants are mapped, and there was little reason to tag letters to its upper and lower parts. After all, neither we nor anyone else has bothered to tag such letters to area V3A, located in the dorsal part of the occipital lobe, and in which upper and lower quadrants are separately mapped (Van Essen and Zeki 1978; Zeki 1978). Moreover, there was good reason to suppose that both upper and lower visual fields are separately mapped within the color center, because clinical evidence has shown more than once that the achromatopsia resulting from lesions there can be restricted to a quadrant.

Then came the method of visual field mapping in human prestriate cortex, using phase encoding (Engel et al. 1994). The early data were interpreted to reveal an area called ‘V4v’ (Sereno et al., 1995; De Yoe et al., 1996), but at the time no one seemed much bothered by the fact that it did not reveal its dorsal counterpart ‘V4d’ or if they did, they did not communicate their worries. This was strange. How could a sophisticated mapping method that stimulates the entire visual field activate the ventral subdivision of an area but not its dorsal counterpart? The
studies of De Yoe et al. (1996) suggested that “V4v” overlapped, at least in part, with our V4. They wrote: “The location of V4v corresponds to some of the locations identified in positron emission tomography studies as having color selective responses. However there is sufficient variability to make it difficult to be certain that such responses could not have come from VP”. The study of Sereno et al., however, did not mention any overlap with the color center that we had defined. The tagging of a small ‘v’ to V4 and calling the area thus defined as ‘V4v’ nevertheless implied that this was the ventral part of V4, in which the upper contralateral quadrant alone is mapped. No one seemed to worry much about such an asymmetric representation, at least not in print. Indeed, why should anyone have thought about it at all? There was, after all, the precedence of “VP” to go by.

This was something of a puzzle for us. Could it be true that only upper visual fields are mapped in the part of the color center located in the fusiform gyrus, with lower visual fields mapped elsewhere, perhaps on the lateral surface of the occipital lobe? Though this seemed improbable, we decided to undertake another study in which we mapped the representation of the visual field using colored and achromatic Mondrians presented separately in the upper and lower hemi-fields. Compared to the sophistication of the phase encoding method, the approach that we used was hoary with age. But the results (McKeefry and Zeki 1997) showed what we had suspected, and what the clinical evidence from Verrey onwards had implied, to us at least – that both upper and lower quadrants are separately mapped, side by side, within the color center and that therefore a center located in the lower occipital lobe is indeed responsible for the elaboration of color in both the upper and lower visual fields. This accounted for why lesions in the color center of one hemisphere can lead to a hemi-achromatopsia, or even to an achromatopsia restricted to one quadrant (see Meadows 1974 and Zeki 1990 for reviews).
Figure 4: A glass brain projection showing three areas discussed in the text. The areas were located by using the Talairach co-ordinates of the three areas given in the paper by Hadjikhani et al. (1998). O corresponds to area V4 defined in Lueck et al. (1989; Zeki et al., 1991; McKeefry and Zeki, 1997); X corresponds to the "new" area "V8" of Hadjikhani et al. (1998) and the + to the area V4v defined by Sereno et al. (1995).

Old wine in new bottles

The repetition of our experiment, using phase encoding, and the confirmation of our results by Hadjikhani et al. (1998) represents perhaps one of the most surprising events in the history of mapping, not for the results obtained, which were in fact identical to ours, but for the way in which these resulted were presented and for what has been read into them. They claimed to have found "a new retinotopic area that we call 'V8', which includes a distinct representation of fovea and both upper and lower visual fields". This “previously undifferentiated cortical area” "was consistently located just beyond the most anterior retinotopic area defined previously, area V4v" (the emphasis of the “v” is mine). These claims gained added weight from an accompanying article by Heywood and Cowey (1998) which declared uncritically: "it is area V8, not the favorite candidate V4" that, when lesioned, produces cortical color blindness. Heywood and Cowey claimed that these results show that "the human color center is distinct from area V4. The newlydefined color area contains a complete retinotopic map of the contralateral visual half field, responds more robustly to color and, unlike V4, is activated by induction of color after
effects” (my italics), which would seem to leave out of account the earlier discoveries of Sakai et al. (1995) on activation of the color center through color after effects.

The many uses of “v”

Anyone reading these articles casually may be forgiven for supposing that a new color area, distinct from what we had called human V4, had been found. But how "new" was this area? A slightly more careful reading of the results in Hadjikhani et al. (1998) shows that this "new" area is nothing more than a rediscovery of what we had defined, dressed up in a new name. Hadjikhani et al. (1998) wrote: "Based on the anatomical location and functional comparison used here, this collateral [sulcus] color selective patch appears equivalent to the previously reported [Lueck et al., 1998; Zeki et al. 1991] area involved in achromatopsia". In fact, a comparison of their Talairach co-ordinates with those given by us in previous publications, shows that the 'new' color selective area, ‘V8’, is identical in position to the location of our human V4 (Figure 4). Moreover, Hadjikhani et al. (1998) found that "When we averaged the Talairach coordinates of the color-selective area 'V4' described in previous studies...we found that it was about twice as close to the location of our retinotopically defined V8, compared to our retinotopically defined V4v" (emphasis on the small ‘v’ is mine). But here comes the catch. Hadjikhani et al. write, in the very next sentence, "This supports all the other evidence that the color selective activity is located in area V8, rather than in 'V4’” which, presumably, is why they say that they had discovered a "previously undifferentiated" area. To any moderately careful reader, the first part of this sentence says that V8 is almost identical to V4, while the second part says that V8 is distinct, and lies anterior, to it. How is this feat achieved? Very simply, by dropping the small v at the end of the sentence! This is no eristic quibble. The statement would have been unexceptionable if they had written “color selective activity is located in area V8, rather than in V4v” but, then, the claim of a ‘new’, ‘previously undifferentiated’ visual area
would have been difficult to sustain. The omission of the small ‘v’, together with claims of a
“new”, “previously undifferentiated” cortical area being discovered, have apparently convinced
some of the innocent who work, or comment, on color vision that something new has indeed been
discovered. Tootell and Hadjikhani (2001) have since written: "The Talairach coordinates of the
original ventral color-selective region (‘V8’ or ‘V4’ or ‘VO’) were never in dispute, although this
has been a matter of apparent confusion". The source of the confusion is not hard to trace. It lies
in the claim that a “new”, “previously undifferentiated” color area located anterior to V4 has been
discovered (Hadjikhani et al. 1998). The confused include even the experts, as witness the article
of Heywood and Cowey about the “newlydefined” color area and about the color center being
"area V8, not the favorite candidate V4" (Heywood and Cowey 1998).

Some of the comments made about the discovery of a “new” “previously undifferentiated”
color center are worth noting, for they speak volumes about the care with which papers are read,
at least in the color vision and imaging communities. I restrict myself to one here, which says:
"There is currently a heated debate about the location of the color center in the visual cortex"
(Gegenfurtner 2001). Actually, there isn't and there never was. The debate is about whether a new
area has been discovered, and the admission of Tootell and Hadjikhani (2001) that it has not
settles the issue. There is of course an additional debate not alluded to in the above quote, which
is about whether there is an area V4v, distinct from V4 (see below).

Cross-species comparisons

In trying to make claims for ‘V8’ as being color selective as against V4 (although they are
the same area), Tootell and his colleagues have taken refuge in a long debate about whether
macaque V4 has any color selectivity. The argument is along two lines: The first one runs like
this: “Zeki believed that monkey V4 is color selective. This is not so, and therefore the color
selective region in human prestriate cortex is not V4 but V8”. That debate, to which I shall return
elsewhere, is irrelevant to the issue of whether a “new” color center has been discovered in the human brain, distinct from what was previously described by us. But it also helps to deflect attention from another problem, namely the status of human “V4v”. The line of argument here is: “Macaque V4, as defined by Zeki, is the fourth visual map after V1. But in human there is a color unselective “V4v” lying anterior to V3 and posterior to the color center called V4 by Zeki and his colleagues and recently re-discovered by us. It is therefore inappropriate to call this re-discovered area V4”. This argument is also irrelevant to whether a “new” color center has been discovered. But it raises an interesting question. Does human “V4v” exist, as an entirely separate and improbable area, which represents upper visual field only? There is no compelling evidence in its favor, which is not to say that there may not be some day. Tried though we have, we have found no evidence for an area “V4v” that is separate from area V4 as we have defined it, or from the same area rediscovered by Hadjikhani et al. (1998).

The most extensive and detailed retinotopic mapping experiments in the human visual brain to date have been done recently by Wandell and his colleagues at Stanford (Press et al. 2001; Wade et al. 2002). Their results show convincingly that there is no quarter field map corresponding to a putative ‘V4v’ in ventral occipital cortex. There is thus no area “V4v” which, given its improbability, is just as well. Their results also show that V4 is the fourth visual map, and that it abuts lower V3. Thus, through this work, one of the arguments outlined in the above paragraph is emasculated and loses its force. In sum, there is no current compelling evidence for the existence of area “V4v” and the onus is on its proponents to demonstrate it convincingly and unequivocally. If they cannot do so, they should withdraw it. What is surprising, given the flimsy evidence for the existence of “V4v”, is the numerous papers that, in addressing other aspects of human visual brain organization, refer to it, thus leaving one with doubts about the quality of evidence that is deemed acceptable in the imaging community. It is hard to imagine that the more hard-nosed physiologists would ever have accepted the existence of a ‘V4v’ based on the kind of
evidence that is currently available.

There is a price to be paid for such uncritical acceptance of the evidence. An example is to be found in a paper by one of the co-authors of the Hadjikhani et al. (1998) paper. Cavanagh et al. (1998), in trying to account for why achromatopsic patients can see moving color stimuli, state, “In humans, the most recent fMRI studies [of Hadjikhani et al.] suggest a more anterior site, V8, for color analysis in a location consistent with the damage in the achromatopsic patients. The human homologue to V4 on the ventral surface (V4v) is probably also damaged in these patients but it includes only a representation of the upper visual fields”. But since V8 is nothing more than human V4 re-named, since Wade et al. (2002) have shown convincingly that there is no quarter field representation corresponding to the ‘V4v’ of Hadjikahni et al. (1998) and since, therefore, the existence of “V4v” is in considerable doubt, the speculation is worse than useless. Such is the price that we pay for having, in the human imaging community, standards of evidence and of proof that fall well short of what is expected, and delivered, in other branches of neurobiology.

The human color center

The color center turns out to be more complex than previously thought (Figure 4). In our work, we have frequently referred to the V4 complex, in both monkey and man, and in our most recent work, we have described this complex in the human as comprising at least two subdivisions, which we refer to as V4 and V4a (Bartels and Zeki 2000). V4 itself is topographically organized (McKeefry and Zeki, 1997; Hadjikahni et al., 1998; Wade et al., 2002), while V4 a is not obviously so, which is not to say that more sophisticated mapping techniques may not reveal some topographic mapping within it in the future (Bartels and Zeki 2000). It is important to realise that V4a lies anterior to V4 and therefore anterior also to the “newly discovered” color center of Hadjikahni et al. (1998). It is a part of the color center that they missed in their studies.
Figure 5: The segregation of the colour selective region in the fusiform gyrus (the V4-complex) into two areas, the posterior retinotopically organised area V4 and the anterior area V4α, as revealed by the re-analysis of the V4 mapping study (McKeefry and Zeki, 1997). (a) **Left:** Statistical parametric map (SPM) viewed in glass-brain projections of the comparison of all chromatic stimuli vs. their achromatic counterparts for both upper and lower visual field stimulation (group of four subjects; threshold: Z>4.81, p<0.05, corrected for multiple comparisons, equivalent to p<0.000001 uncorrected). **Right:** Slices taken through an SPM of a single subject, superimposed on its structural image (slices at x: -33 and z: -14 mm). (b) Projection of the comparison of either upper field (in red) or lower field (in green) stimulation with colour vs. their achromatic counterparts onto a ventral view of a human brain (overlapping regions are shown in yellow). For V4 (bottom), the SPM of the following comparison is projected onto the drawing: (superior coloured vs. [superior achromatic + inferior coloured + inferior achromatic]); (group of four subjects; threshold: Z=4.81, p<0.05 corrected). For V4α (top), SPMs of a comparison of colour vs. achromatic stimuli within the corresponding hemifield is projected onto the drawing (threshold: Z=3.09, p<0.001 uncorrected). (c) An independent component analysis (ICA) separates spatially independent maps of brain activity without *a priori* knowledge about the stimulus conditions. ICA isolated the complete V4-complex, including the posterior (V4, bottom) and the anterior (V4α) subdivisions in both hemispheres, shown here in the glass-brain view of a single subjects' brain. (From Bartels and Zeki, 2000)
The topographic organization of V4 accounts well for why the achromatopsia induced by lesions in the fusiform gyrus may involve the whole of the contralateral hemi-field (as in the patient of Verrey 1888) or may be limited to a quadrant. Less certainly, the subdivisions of the color center into V4 and V4a may eventually help to account for what is problematic with the syndrome of cerebral achromatopsia. In some patients, the achromatopsia is transient, whereas in others it is more long lasting and even permanent. Moreover, in some patients the achromatopsia is less severe than in others, and can be described as a dyschromatopsia. In such patients, the color loss is not complete, being greater for some colors (usually the blues and the greens) than for others (see Zeki 1990 for a review). Moreover, the color vision of some dyschromatopsic patients is very much wavelength based, in that they are not able to compensate for the predominance of one wavelength over another when the wavelength composition of the viewing conditions change (Kennard et al., 1995). It is possible, but yet to be shown conclusively, that these variations depend upon the extent to which the lesions in the fusiform gyrus involve both subdivisions of the human color center (for a review see Bartels and Zeki 2000).

Of course, there are those who believe that the whole notion of a cortical specialization for color vision is a fantasy and the search for it an outmoded folly. How convincing are their arguments? Here is one of the most forceful. Lennie (1999) writes: “The controls used in most functional imaging studies have involved comparing activity evoked by the presentation of an array of colored surfaces with the activity evoked by the presentation of the same elements at the same luminance, but now set to be gray. Visibility and salience can be quite different. Overall, the evidence for a specialized color pathway is not strong” (Lennie 1999). Consider this statement carefully. It says essentially that the color center may be nothing more than a salience centre. But color does make the visual world more salient. And so there is a color center after all, even after this denial, except that in this instance we call it the “salience center”! If there are good arguments against a color centre in the human brain, why resort to such wholly unconvincing arguments?
**An improbable area in the making, "KO":**

We are currently witnessing the making of another improbable area. It is known as the “kinetic occipital” area or area “KO”, for which an alternative and, to me, more acceptable name is area V3B (Smith et al. 1998; Press et al., 2001). Area “KO” has been described as not only being “specialized in the processing of kinetic contours” but “genuinely specialized” in this task (Van Oostend et al., 1997). The acronym given it by Orban and his group reflects this belief (Orban et al. 1995; Dupont et al., 1997; Van Oostend et al., 1997). Its relation to color vision lies in the current attempts to homologize it with macaque V4 or, more accurately, with the dorsal part of macaque V4, dubbed “V4d” (Tootell and Hadjikhani 2001), to which I return below. This would naturally make of ‘KO’ another improbable area, since all the evidence shows that cells in the upper part of V4 (‘V4d’) have their receptive fields in the lower contralateral quadrant (Van Essen and Zeki 1978; Pinon et al., 1998). This in turn would imply that V4d processes kinetic contours in the contralateral lower field only, with no equivalent area for processing the same signals when they occur in the upper contralateral visual field, or at least no declared area.

Before going into such speculations, it is important to consider the evidence that “KO” is “genuinely… specialized in the processing of kinetic contours” (Van Oostend et al. 1997). The idea that there are brain areas specialized for the processing of forms derived from motion was actually first posited by Gulyas et al. (1995) but it was based on a faulty analysis of their imaging data which has attracted criticism (Frackowiak et al 1996), and is therefore questionable. How reliable is the evidence of Orban’s group?

In their work, Orban’s group (Orban et al., 1995; Dupont et al., 1997; Van Oostend et al., 1997) compared the activity produced in “KO” when humans viewed bars produced from kinetic contours, from luminance differences and from translational motion. They found that several areas are activated in these comparisons, including V3A, V5 and “KO”, though the latter was more active. This led them to conclude that the brain devotes a special area to the processing of
kinetic contours. But there is a critical omission in these comparisons. What if “KO” is specialized for the extraction of shapes, no matter how derived, rather than the extraction of shapes from kinetic contours specifically? A critical comparison to distinguish between the two possibilities would be to study the activity in “KO” when contours are extracted from other attributes, for example equiluminant colors. Color is generally agreed to be the most separate in its cortical representation from motion even if there is much anatomical opportunity for cross talk between the two systems and even if such cross talk does occur (Callaway, 1998). If “KO” is inactive when humans view visual stimuli in which simple shapes are extracted from equiluminant colors, then a fairly strong, though perhaps not definitive, case can be made for “KO”’s putative specialization. But if it is equally active when contours are extracted from equiluminant colors, as it is when the same contours are extracted from kinetic contours, then “KO” can be safely considered not to be specialized for the processing of kinetic contours. Our own studies show that area V3B, a better name for ‘KO’, is in fact engaged when shapes are extracted, regardless of their provenance. This is not the place to go into this evidence, for what I am emphasizing is that claims for the specialization of ‘KO’ have been made in the absence of critical comparisons which would establish whether it is specialized in the extraction of shapes from kinetic stimuli. At present, the evidence relating to the “genuine specialization” of ‘KO’ for processing kinetic contours is so incomplete that it should raise considerable doubts in the neuroimaging community. Such evidence may of course be forthcoming in the future, but it is not available today. On the other hand, there is considerable evidence for ‘cue-invariance’ in the visual brain, by which is meant that cells in different areas of the brain respond to their preferred stimuli no matter how these stimuli are derived, that is whether from luminance differences, kinetic boundaries or equiluminant colors (Albright 1992; Geesman and Andersen, 1996; Grill-Spectator et al., 1998; Sary et al., 1993). To overwrite this evidence, it is not sufficient to keep repeating that ‘KO’ is specialized for the processing of kinetic contours, as if every utterance
establishes its role more solidly. What is needed is a solid experiment to establish its specialty. Such evidence is not currently available.

**Precedence replaces evidence**

The doubtful status of “KO” as an area that is specialized in the processing of kinetic contours is rendered more emphatic by the current attempts to make of human “KO” the homologue of the dorsal part of macaque V4, or V4d (Tootell and Hadjikhani 2001; see also the two contradictory abstracts by Orban’s group - Fize et al. 2001 and Vanduffel et al. 2001), based on the argument that “KO” is the “topologue” of V4d. In replicating the findings of Orban’s group, Tootell and Hadjikhani (2001) believe that they have confirmed that “KO” is indeed specialized for kinetic contours. But they used the identical stimuli and paradigm as Orban’s group, and in essence replicated Orban’s study. So complete was this repetition that the critical omission, the one that casts doubt on the conclusions of Orban’s group, was not remedied at all. The critical comparison, of determining whether ‘KO’ is also activated as well by shapes derived from other attributes, and especially from equiluminant colors, was not done. The evidence of Tootell and Hadjikhani (2001) therefore adds nothing to the unconvincing evidence of Orban’s group.

The issue is further compounded, and actually compromises the status of “KO” further, by the suggestion that it is the homologue of V4d. If so, then “KO” must be registering events occurring in the contralateral inferior quadrant only, since cells in V4d have their receptive fields there (Van Essen and Zeki 1978; Pinon et al., 1998). This would make of “KO” an area that is specialized for the processing of kinetic contours when these occur in the lower contralateral quadrant only, leaving the same activity occurring in the upper contralateral quadrant unrepresented in the cortex, or represented in another area, which would make of that latter, yet to be discovered area, another improbable one. In fact, the evidence of Press et al. (2001)
suggests otherwise, in showing that both upper and lower contralateral quadrants are mapped in V3B, the better name for ‘KO’, a fact that appears to have been ignored in the headlong rush to homologize ‘KO’ with V4d, since the latter represents lower contralateral quadrants only. Thus, a circle of confusion, consisting of increasing numbers of improbable areas, follows from the initial assumption that there is an improbable area, VP. The best way of breaking that confusion is to get rid of the assumption, or at any rate to test it before accepting it. Where the assumption has been tested, it has not found to be true.

It is of course possible that this is exactly the way that the cortex is specialized and that, in thinking of areas that represent one quadrant only, Jon Kaas, and I after him, are being extremely unsophisticated in our approach to cortical mapping. Yet one cannot help feeling that the evidence in favor of these improbable areas is very weak at present. The danger is that, once an improbable area is accepted on the basis of such weak evidence, then one does not even have to produce strong evidence in favor of new improbable areas. Nor is this a hypothetical danger. In justifying their homology of “KO” with V4d, Tootell and Hadjikhani (2001) write: “Although such ‘separated’ quarter-field representations are conceptually unsatisfying, they are not unprecedented: the quarter-field representations in macaque ‘V3’ and ‘VP’ have long been considered separate areas by some investigators, based on empirical differences between V3 and VP (Burkhalter et al., 1986; Van Essen et al., 1986; Felleman and Van Essen, 1991; Felleman et al., 1997). Yes, by some but not by all (see above). And even if it is accepted by all, the present evidence in favor of areas with just a quarter field representation is still not compelling. To date, at least two (‘VP’ and ‘V4v’) have already been convincingly knocked out in both the human and the macaque brains (Wade et al. 2002; Lyon and Kaas 2001, 2002). This does not auger too well for those who want to use the precedent of dubious quarter field representations to hastily erect new ones.
Conclusion

I have already reviewed extensively the early literature on the cortical involvement in colour vision, and have tried to show to what extent it was dominated by preconceived notions (Zeki 1990; 1993). Looking at what has happened since then, it seems that the considerable technological sophistication that is at our disposal for studying the human brain has not been matched by an equal sophistication in thinking about the brain. Where something is improbable, the evidence in its favor should be impeccable. The evidence of the past few years shows, instead, that, in studies of cortical color vision, the improbable has imperceptibly become the acceptable and set the precedent for other improbables to become acceptable, without the intervention of questioning.

The story of color vision as it relates to the cortex is thus a very sad one. In neurobiology it is indeed, to quote the title of Ford Maddox Ford’s book, The Saddest Story.

There are of course also good things that have happened about our understanding of the role of the cortex in color vision. I will not relate these, but leave it to the commentators on discoveries in cortical color vision to do so.

The work of this laboratory is supported by the Wellcome Trust, London.
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