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Interhemispheric transmission times in the presence and absence of the forebrain commissures: effects of luminance and equiluminance

BETTINA FORSTER* and MICHAEL C. CORBALLIS

Department of Psychology, University of Auckland, Private Bag 92019, Auckland, New Zealand

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Abstract—One subject (L.B.) with full forebrain commissurotomy, one (R.B.) with callosal agenesis and 20 normal controls were tested for simple reaction time (RT) with each hand, to visual stimuli in one or the other visual field. RTs for uncrossed conditions (hand ipsilateral to the visual field) were subtracted from RT to crossed conditions (hand contralateral to the visual field) to yield the crossed–uncrossed difference (CUD), taken to be a measure of interhemispheric transfer time. CUDs increased from an average of 4.9 ms among the control subjects, to 23.3 ms for R.B., to 53.1 ms for L.B. Although overall RTs in all subjects increased with decreasing luminance of the stimuli, the CUD was not systematically affected and remained largely unaffected even under equiluminance. The results support previous evidence that interhemispheric transfer, even in the split brain, depends on visually insensitive pathways. © 1998 Elsevier Science Ltd. All rights reserved.

Key Words: split-brain; callosal agenesis; callosotomy, interhemispheric transfer; luminance; equiluminance.

Introduction

In 1912, Poffenberger [38] devised a simple technique for estimating the time for information to be transferred from one cerebral hemisphere to the other, using simple reaction time (RT) to laterally presented visual stimuli. He noted that crossed conditions, in which the responding hand is contralateral to the visual hemifield of the stimulus, must require interhemispheric transfer, while uncrossed conditions, in which the responding hand is ipsilateral to the visual hemifield, involve processing that is contained entirely within a hemisphere. He therefore proposed that subtracting the RT for uncrossed conditions from the RT for crossed conditions should yield an estimate of interhemispheric transfer time, on the assumption that transmission of simple sensory information and the initiation of uncomplicated movements were conducted over fixed neuroanatomical pathways. The difference between these conditions is known as the crossed–uncrossed difference (CUD).

In normals, the CUDs are typically in the range 2–6 ms [2, 7, 9, 28]. Although there is some variability, with negative values occasionally being reported, the short CUD in normals probably reflects transfer through fast callosal channels. However there may be different channels within the corpus callosum itself [17] and extracallosal channels such as the anterior commissure or midbrain commissures [7, 23] may also play a role. Extracallosal pathways can be examined by testing people who lack the corpus callosum and in some cases, the other forebrain commissures as well. Further, by varying the visual characteristics of the stimuli, something of the nature of the neural interhemispheric message can be inferred.

Interhemispheric transfer of simple visual stimuli is at least possible in subjects who have undergone surgical section of the forebrain commissures [10], since they can respond to simple signals under crossed conditions, but their RTs are abnormally long. The transfer is presumably accomplished via subcortical structures. Studies have shown, for example, that midbrain structures and commissures are implicated in visual perception and discrimination in split-brain monkeys [42] and split-brain cats [39] and similar pathways may account for inter-

* To whom all correspondence should be addressed: Bettina Forster or Michael C. Corballis, Research Centre for Cognitive Neuroscience, Department of Psychology, University of Auckland, Private Bag 92019, Auckland, New Zealand. Tel.: +64 9 373 7599; Fax: +64 9 373 7450; E-mail: b.forster@auckland.ac.nz or m.corballis@auckland.ac.nz

hemispheric integration in the human split brain [4, 20, 29, 44].

People with callosal agenesis do not exhibit the obvious signs of disconnection that are apparent in split-brained patients [3, 19, 27]. Even so, their CUDs are longer than those in normals [18], although they tend to be shorter than in those with surgical section of the corpus callosum [1, 27]. Recent research nevertheless suggests that transfer in acallosals is probably accomplished via subcortical pathways [3, 19, 31] rather than via the intact anterior commissure.

It has been argued that if changes in visual parameters such as stimulus intensity or eccentricity affect the CUD, then interhemispheric transfer must be occurring along visually sensitive pathways [4]. Studies with normal subjects have generally failed to reveal any effects of luminance [34] or eccentricity [4, 7, 9, 43] on CUD and studies of visually evoked potentials further suggest that the CUD represents interhemispheric transfer of motor or sensorimotor information, rather than purely sensory information [25].

The evidence from acallosals and split-brain patients is less consistent. Three such studies of acallosals have shown changes in CUD with variations in both stimulus luminance [29, 33] and eccentricity [24], suggesting that extracallosal transfer takes place through a visual channel, perhaps the anterior commissure, or more likely the collicular commissure since the superior colliculus of monkeys has been shown to be sensitive to stimulus light flux, brightness and pattern and is retinotopically organised [36, 42]. One exception, however, is the acallosal M.M., whose CUD remained invariant with changes in either the luminance or the eccentricity of the stimulus [9]. In that case, the CUD might have reflected either non-visual interhemispheric transfer or response initiation via ipsilateral motor pathways [22].

In people with surgical section of the forebrain commissures, the picture is similarly inconsistent. Clarke and Zaidel [9] tested three subjects with full forebrain commissurotomy, namely, N.G., A.A. and L.B. In N.G. and A.A., the CUD was sensitive to stimulus eccentricity but not to intensity variations, but in L.B. neither intensity nor eccentricity significantly affected the CUD. Clarke and Zaidel suggested that, in L.B., responses in the crossed condition were accomplished via ipsilateral motor pathways and it was this rather than interhemispheric transfer that caused the delay. Sergent and Myers [41] also found no effect of stimulus intensity on the CUD in either L.B. or N.G. One difficulty in interpreting these findings is that the CUD can vary quite markedly, and for unknown reasons, between blocks of trials within the same individuals [16].

This study examines the CUD in a group of 20 neurologically intact subjects, one man (L.B.) with section of the forebrain commissures and another man (R.B.) with callosal agenesis. The study is in two parts. In Part 1, the luminance of the stimuli was varied. It was expected that this would result in longer RT the lower the luminance and the question was whether luminance would also affect the CUD.

In Part 2 the stimuli were equiluminant with the background, providing a more critical test of the role of subcortical visual pathways. To achieve equiluminance, the stimuli were flashed in a low-level blue light against a bright yellow background [8]. In this display, the stimulus information is carried principally by the response of the blue/yellow chromatic pathway and isolates the short-wavelength-sensitive (SWS) cones. Vision with equiluminant-colour contrast is thought to register only in the parvocellular subdivision of the geniculocortical visual pathway, while the phylogenetically older magnocellular visual subsystem remains inactive [26, 35]. Furthermore, there is little evidence that true colour or wavelength sensitivity is present in the neurons of midbrain structure like the superior colliculus. Perry and Cowey [37] have shown that B-cone cells do not project to the superior colliculus in macaque monkeys and Kadoya *et al.* [21] reported that units recording in the colliculus of the squirrel monkey mainly responded to monochromatic stimulation in the red range. However, they also reported a few responses to green and blue light (3 of 33 units), suggesting that some colour discrimination may be possible at the collicular level, although it has also been suggested that these may be generated by cortical projections [32].

In Part 2, therefore, we expected equiluminant stimuli to activate only the parvocellular subdivision of the geniculocortical pathway, bypassing subcortical visual pathways. If equiluminance does not affect the CUD in the split brain, this would imply that subcortical transfer occurs after the stimulus has been encoded cortically.

General Method

Subjects

One commissurotomized patient (L.B.), one acallosal man (R.B.) and 20 control subjects were tested.

L.B. underwent section of the forebrain commissures, including the corpus callosum, anterior commissure and hippocampal commissure for the relief of intractable epilepsy in 1963. Magnetic resonance images (MRI) had confirmed complete section of the corpus callosum [5]. Further information on L.B.'s case history is provided by Bogen and Vogel [6]. At the time of testing L.B. was 45 years old. He is right-handed. His speech was slightly slurred and he showed an uncharacteristic listlessness on the days of testing (June and August, 1996) which might indicate some left-hemispheric dysfunction. We have no reason to suspect that this impaired interhemispheric transfer.

R.B. is a man with agenesis of the corpus callosum. Figures 1 and 2 show magnetic resonance images of his brain, taken in 1988 when he was 12 years old. The absence of the corpus callosum is apparent in both figures, with the horizontal section in Fig. 1 also showing greatly enlarged occipital horns of the lateral ventricles, typical for callosal agenesis. Fig. 2 shows that the anterior commissure is present, but the resolution of the image is too poor to indicate the presence or otherwise of Probst bundles. Standard neurological examinations have revealed no

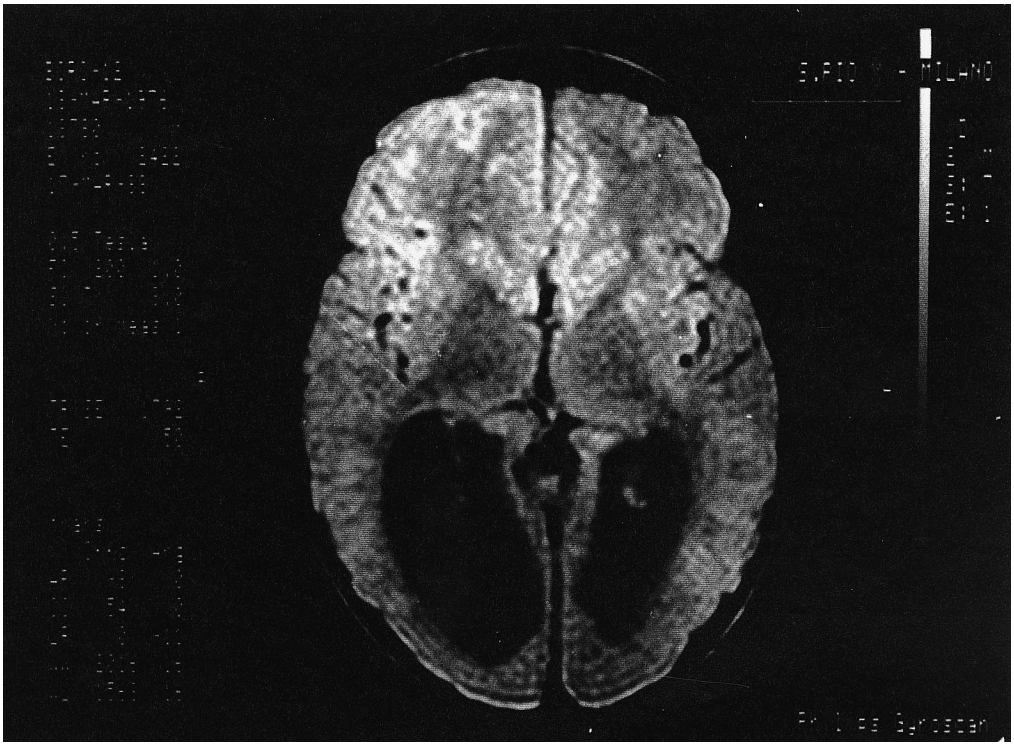

Fig. 1. Magnetic resonance image of R.B.'s brain in horizontal plane.

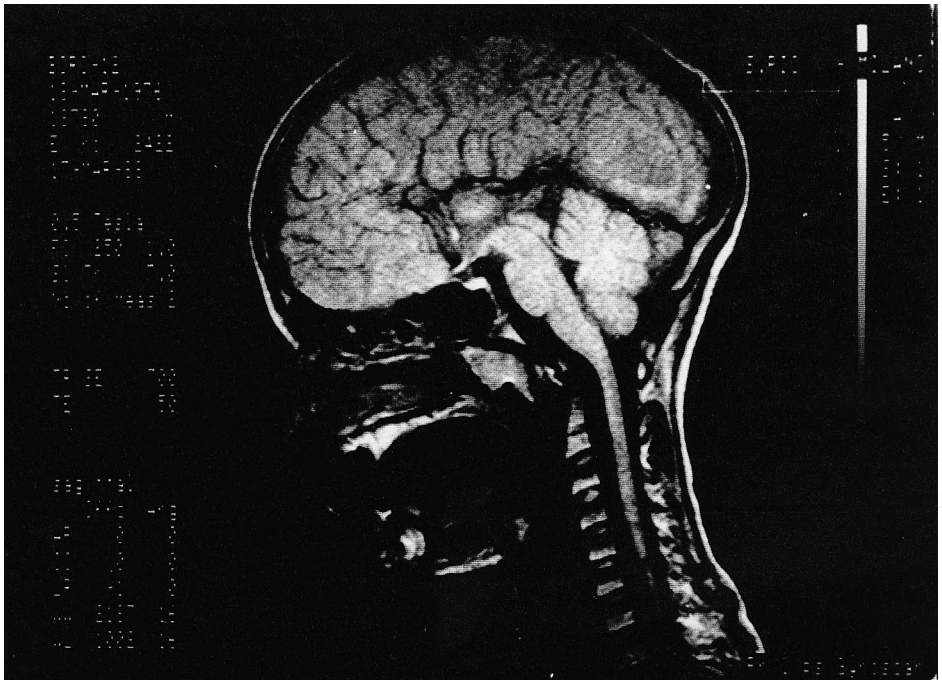

Fig. 2. Magnetic resonance image of R.B.'s brain in midsagittal plane.

clear manifestations of interhemispheric disconnection [1] except for a lengthened CUD and some subtle signs of defective visual communication between the hemispheres [11]. R.B. is left-handed.

The 20 control subjects comprised 10 men and 10 women and all participated voluntarily. All were right-handed and their age ranged from 21–35 years, with a mean of 24 years.

Apparatus and stimuli

The experiments were run on an IBM-compatible computer with fast-fade videographics adapter (VGA) screen. The software package Micro Experimental Laboratory (MEL) [40] was used to program the experiments.

The stimuli comprised filled circular disks of diameter 0.86°

in visual angle, presented singly for 133 ms centred either 2.5° or 5.5° to the left or right of a central fixation cross.

Procedure

Each subject was tested in a lit room. A chin rest was used to minimise head movements and to keep the subject's eyes at a distance of 0.57 m from the screen, so that 1 cm on the screen corresponded to 1° of visual angle. Subjects were instructed to press the N key as fast as possible whenever a light stimulus was flashed on the screen, using the forefinger of the designated hand. The control subjects initiated the trials by pressing the space bar with the non-responding hand, while the experimenter initiated the trials for the other two subjects by pressing either the 1 key or the = key. Between the initiation of the experiment and the appearance of the stimulus and between subsequent stimuli, there was a variable delay of 800 ms, 1100 ms, 1400 ms or 1700 ms. These delays were presented equally as often, but in randomised order, for stimuli in each visual field. There were eight practice trials with each hand in turn prior to each experimental block. The number of trials per block varied under different conditions, as specified below. The distance of the stimuli from fixation was constant within a block, but the stimuli appeared equally often in each visual field, in random order.

Subjects were told before each block which response hand they were to use. The order of the response hand used was counterbalanced across the control subjects and across sets of four blocks was either right, left, left, right or left, right, right, left. For both L.B. and R.B. (who is left-handed) the order was left, right, right, left.

Part 1: Variations in Luminance

Method

Stimuli. In Part 1, the stimuli varied in luminance over each block of trials. There were two series of blocks, one in which the eccentricity of the stimuli was 2.5° and one in which it was 5.5°. The luminance of the stimuli was 64.9 cd/m^2, 23.2 cd/m^2, 7.8 cd/m^2, or 3.1 cd/m^2, against a 0.5 cd/m^2 background (measured using a Pritchard Photometer, model PR 1980A). These were presented in random order within a block.

Procedure. Each control subject received four blocks of 128 trials, including all four luminances, at the 2.5° eccentricity and four blocks of 96 trials, including only the three highest luminances, at the 5.5° eccentricity. Half of the subjects received the four blocks at the 2.5° eccentricity followed by the four blocks at the 5.5° luminance; this order was reversed for the other half.

L.B. was given four blocks of 96 trials with the three brightest stimuli (64.9 cd/m^2, 23.2 cd/m^2 and 7.8 cd/m^2), at each eccentricity. R.B. received four blocks at the 2.5° eccentricity only. Each was then given one additional session of four blocks of 64 trials with the two dimmest stimuli (7.8 cd/m^2 and 3.1 cd/m^2) at the 2.5° eccentricity.

Results: control subjects

Trials on which RTs were less than 133 ms or greater than 833 ms were discarded,* resulting in a loss of 1.08% of trials at the 2.5° eccentricity and 0.7% at the 5.5° eccentricity. The remaining trials were averaged for each condition of visual field, hand, luminance, delay and eccentricity for each subject and the mean RTs were then subjected to separate analysis of variance for each eccentricity, with hand, visual field, luminance and delay as within-subject factors and gender a between-subjects factor.

There were no significant main effects of hand or visual field at either eccentricity, but the interaction between the two was significant in both; at the 2.5° eccentricity ($F(1,18) = 21.91$, $P < 0.0001$) and at the 5.5° eccentricity ($F(1,18) = 21.07$, $P < 0.0001$). The mean CUDs computed from these interactions by subtracting mean RT from the uncrossed conditions from that for the crossed conditions were 5.2 ms at the 2.5° eccentricity and 5.7 ms for the 5.5° eccentricity.

The effect of luminance was significant at both eccentricities (at 2.5°, $F(3,36) = 71.37$, $P < 0.0001$ and at 5.5° $F(2,36) = 29.77$, $P < 0.0001$). However the triple interaction between luminance, hand and visual field was not significant (at 2.5° $F(3,54) = 2.48$, n.s. and at 5.5° $F(2,36) = 0.46$, n.s.), suggesting that variations in luminance did not reliably affect the CUD, even though they affected overall RT.

Considering the three brightest luminances, the subjects responded on average 8.3 ms faster at the 2.5° eccentricity than at the 5.5° eccentricity, but the difference did not approach significance ($F(1,37) = 0.11$, n.s.). The three-way interaction between hand, visual field and eccentricity was insignificant, suggesting that the CUD was impervious to variations in eccentricity. Fig. 3 shows the mean RTs for crossed and uncrossed conditions at each luminance and eccentricity. Although the CUD is slightly negative at the brightest luminance at the 2.5° eccentricity, this is unlikely to represent a systematic trend, since the effect of luminance on the CUD did not reach significance and since the same pattern was not repeated at the 5.5° eccentricity.

Results: L.B.

L.B.'s RTs were subjected to a within-subject analysis of variance. In contrast to the analysis carried out for control subjects, this analysis permits conclusions to be generalised only to L.B. himself and not to a population of split-brained people. In the first two sessions, only the three brightest stimuli were presented. 1.30% of the trials at the 2.5° eccentricity and 1.84% at the 5.5° eccentricity

* Milner [30] compared different methods of treating the same RT data to eliminate "noise" produced by long RTs. He found that different ways of calculating means (arithmetic or harmonic) in combination with high cut-off or high and low cut-off procedures lead to similar results which lie well within the experimental error.

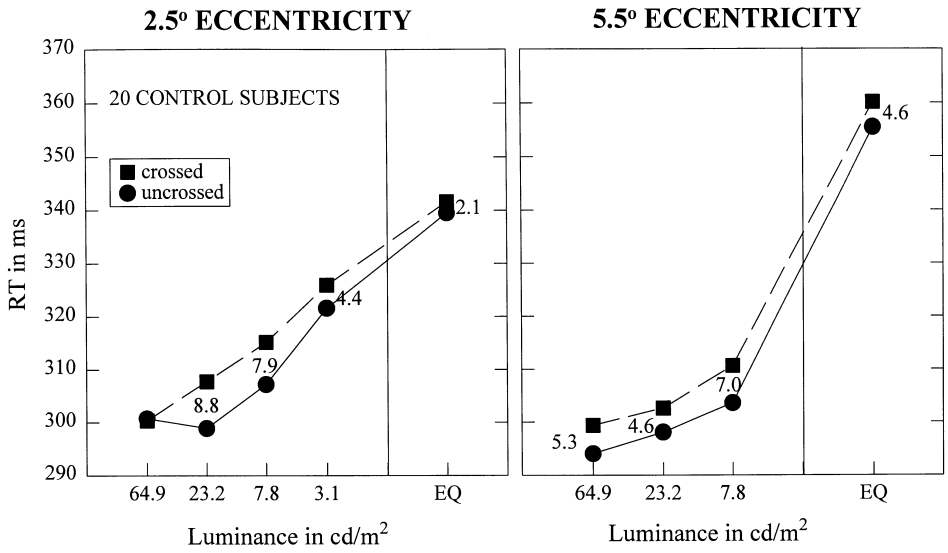

Fig. 3. Mean crossed and uncrossed RTs and corresponding CUD of the control subjects are shown. Mean results for each luminance of Part 1 and for Part 2 are presented according to eccentricity.

were excluded from the analysis, since they lay outside the range 133–833 ms. To adjust for the resulting unequal cell frequencies the RTs were analysed by the method of unweighted means [45].

At both eccentricities, L.B. responded significantly more slowly with his right than with his left hand: At 2.5°, the difference was 92 ms ($F(1,331) = 80.04$, $P < 0.0001$), and at 5.5° it was 75 ms ($F(1,329) = 78.89$, $P < 0.0001$). This slowness with the right hand is uncharacteristic of L.B. [9] and may be related to the same left-hemisphere dysfunction, of recent origin, that also resulted in some slurring of speech and general listlessness. There was also a significant interaction between hand and hemifield at each eccentricity (at 2.5° $F(1,331) = 15.19$, $P < 0.0001$ and at 5.5° $F(1,329) = 85.06$, $P < 0.0001$). The corresponding CUDs were 39 ms and 73 ms, respectively. This increased CUD at the 5.5° eccentricity was made up of both a decrease in RT in the uncrossed conditions and a similar increase in RT in the crossed conditions.

There was no main effect of luminance at the 2.5° eccentricity ($F(2,331) = 2.36$, n.s.), or at the 5.5° eccentricity ($F(2,329) = 0.16$, n.s.). Further, the triple interaction between hand, visual field and luminance was not significant in either case ($F(2,331) = 0.04$, n.s. and $F(2,329) = 0.30$, n.s., respectively), suggesting that variations in luminance did not affect the CUDs. Fig. 4 shows the mean RTs for crossed and uncrossed conditions at each luminance and eccentricity.

L.B. responded on average only 0.5 ms faster at the 5.5° than at the 2.5° eccentricity and this did not approach significance. However, a significant three-way interaction between hand, visual field and eccentricity ($F(1,732) = 9.31$, $P < 0.0001$), indicated that the CUD was significantly longer (by 41 ms) at 5.5° than at 2.5°.

In the further session with the two dimmest stimuli at the 2.5° eccentricity, L.B. again responded significantly more quickly (by 63.8 ms) with his left than with his right hand ($F(1,224) = 39.42$, $P < 0.0001$). The interaction between hand and visual field was significant ($F(1,224) = 18.55$, $P < 0.0001$), but although there was a significant main effect of luminance ($F(1,224) = 4.18$, $P < 0.05$) the interaction between hand, visual field and luminance did not approach significance ($F(1,224) = 0.02$, n.s.), suggesting that luminance had no effect on the CUD. L.B.'s mean RTs for crossed and uncrossed conditions are included in Fig. 4.

L.B.'s RT was significantly longer (by 24 ms) on this session than in the earlier session at the 2.5° eccentricity ($F(1,615) = 9.81$, $P < 0.0001$), probably because stimulus luminance was lower, but this difference did not interact with hand and visual field, ($F(1,615) = 0.31$ n.s.). This again suggests that CUD was not influenced by variations in luminance.

Results: R.B.

R.B. was tested only at the 2.5° eccentricity. Only 0.26% of trials were excluded as being outside the range of 133–833 ms and RTs were again analysed by the method of unweighted means to adjust for unequal cell frequencies. The main effects of hand and visual field were not significant, but the interaction between them was highly significant ($F(1,335) = 15.05$, $P < 0.0001$). The CUD was 26 ms. The luminance of the stimuli had no significant effect on the overall RT ($F(2,335) = 1.92$, n.s.) or on the CUD ($F(2,335) = 0.62$, n.s.).

Like L.B., R.B. received a second session at the 2.5° eccentricity with the two lowest luminances. There were

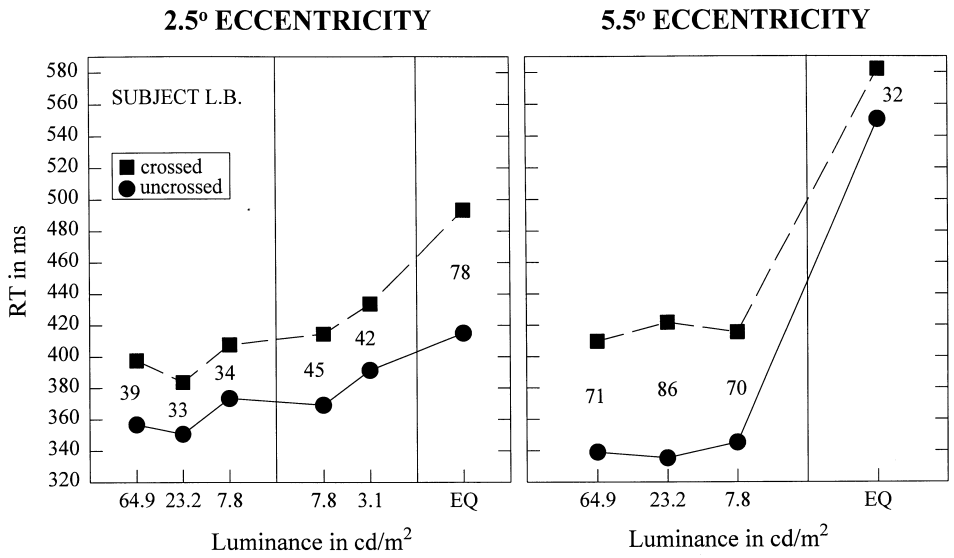

Fig. 4. Mean crossed and uncrossed RTs and corresponding CUD of L.B. are shown. Mean results for each luminance and session of Part 1 and for Part 2 are presented according to eccentricity.

no significant main effects of hand or visual field, but again the interaction was significant ($F(1,246) = 8.13$, $P < 0.0001$). The main effect of luminance was not significant ($F(1,246) = 0.14$, n.s.), nor was the interaction between hand, visual field and luminance ($F(1,246) = 0.77$, n.s.). Fig. 5 plots the mean RTs for crossed and uncrossed conditions for each luminance in both sessions. Although the CUD fluctuates somewhat, in neither session did the effect of luminance on the CUD approach significance. R.B. responded on average 53 ms more slowly in the second session at the 2.5° eccentricity than in the first ($F(1,617) = 92.51$, $P < 0.0001$), presumably because the luminances were lower, although there was also a clear context effect in that R.B. responded much more slowly at the 7.8 cd/m^2 luminance in the first session than at the same luminance in the second session. The difference between first session and second session did not interact with hand and visual

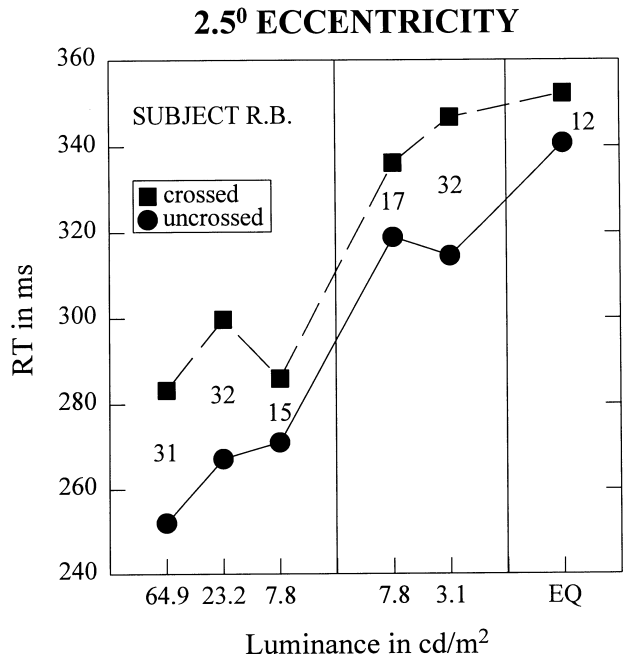

Fig. 5. Mean crossed and uncrossed RTs and corresponding CUD of R.B. are shown. Mean results for each luminance and session of Part 1 and for Part 2 are presented.

field ($F(1,617) = 0.46$, n.s.), suggesting that the lowered luminance in the second session did not influence the CUD.

Discussion

For the normal subjects, there was a clear effect of luminance on overall RT, but as in earlier studies neither luminance [3] nor eccentricity [4, 7, 9] had a significant effect on the CUD. That luminance affected overall RT but not the CUD implies that interhemispheric transfer takes place after encoding of the visual stimulus. The information transferred presumably has to do with response initiation rather than stimulus detection. There was no main effect of eccentricity, so the lack of any effect of eccentricity on the CUD is not decisive as to the locus of transfer.

The CUDs for both L.B. and R.B. were considerably longer than those for the normal subjects, supporting the idea that transfer in normals takes place via the corpus callosum. L.B. also differed from the normal control subjects in that his CUD was considerably longer when the eccentricity was 5.5° than when it was 2.5°. This is in line with the finding of Clarke and Zaidel [9] for the split-brained patient N.G. and A.A., but is at odds with their results for L.B. Although Clarke and Zaidel's results are consistent with ours, as well as those of Sergent and Myers [41], in that luminance did not affect the CUD in L.B., they found eccentricity to have no effect. The fact that eccentricity did influence the CUD nevertheless implies that transfer took place via a channel sensitive to retinal location. The most likely contender is the collicular commissure, since efferents from the visual cortex to the colliculus are topographically organised, at least in the monkey [36, 42].

R.B.'s results, like those of L.B. and the normal subjects, revealed an increase in RT with an increase in luminance, but his CUD was not significantly affected, which is contrary to reports that increasing luminance decreases the CUD in other acallosal subjects [29, 33]. R.B.'s CUD was also markedly shorter than L.B.'s. This might indicate that transfer occurs along different commissures in these two individuals, or that some compensatory effect is responsible for the shortened CUD of R.B.

Part 2: Equiluminant Stimuli

Method

Stimuli. Here, a tritanopic display was used: The stimuli were displayed in a low-level blue light against a bright yellow background. This method, suggested by Cavanagh *et al*. [8], generates a near-equiluminous colour display which inhibits the occurrence of artefacts at the image border.

For all subjects equiluminance settings were determined using flicker photometry in an RGB system allowing each of red, green and blue wavelengths to be varied on a 64-point scale. The background of the VGA screen was set to yellow by choosing maximum settings of 63 for each of red and green and the 0 setting for blue. A black circle with radius 1° then appeared in the center of the screen. After 700 ms, it was replaced by a flickering disk, also of radius 1°, for 1 s. The disk was generated by adding 30, 35, 40, or 45 units of blue to the yellow background and at the same time subtracting 0, 1, 2, or 3 units of red. The grey disc so generated alternated with the yellow background at 16 on-off cycles per s. There were five blocks of 60 trials, involving all combinations of added blue and subtracted red. The subjects were asked to indicate whether the disk seemed to flicker or not. The settings used in the equiluminance displays were the combinations that minimised the perception of flicker. For both L.B. and R.B., the disc was seen as continuous (not flickering) when the blue increment was 35 units and the red decrement 3 units. This was also the setting most often used for the control subjects.

Results: control subjects

RTs outside the range of 133–833 ms were again excluded, resulting in the loss of 0.78% of trials at the 2.5° eccentricity and 0.75% of trials at the 5.5° eccentricity. The mean RTs for each subject for each hand, visual field and delay were subjected to analysis of variance; the between-subject factor was gender and the within subject-factors hand, visual field and delay. Separate analyses were first carried out for each eccentricity.

The main effects of hand and visual field were not significant at either eccentricity. The interaction between them failed to reach significance even on a 1-tailed test at the 2.5° eccentricity ($F(1,18) = 2.77$, n.s.) where the CUD was 2.1 ms, but was highly significant at the 5.5° eccentricity ($F(1,18) = 11.39$, $P < 0.005$) where the CUD was 4.7 ms. Combining eccentricities revealed a significant overall interaction between hand and visual field ($F(1,36) = 13.48$, $P < 0.001$). Although overall RT was 17 ms shorter at 2.5° than at 5.5°, the main effect of eccentricity was not significant ($F(1,36) = 1.49$, n.s.), nor was the interaction between hand, visual field and eccentricity ($F(1,36) = 1.49$, n.s.). Figure 3 shows the mean RTs of the control subjects, along with those from Part 1.

The average CUDs in both Part 1 (5.4 ms) and Part 2 (3.4 ms) lie within the typically estimated transfer time in normal subjects [2, 7, 28]. Furthermore, in contrast to the RTs themselves, the CUDs of Part 2 were not prolonged; if anything, they were reduced.

Results: L.B.

RTs outside the range of 133–2233 ms were omitted, resulting in the loss of 1.25% of trials at the 2.5° eccen-

tricity and 1.56% at the 5.5° eccentricity. This higher cut-off criterion was adopted to compensate for the fact that L.B.'s right-hand responses were considerably slowed, especially at the larger eccentricity: At the 2.5° eccentricity the right-hand responses were 66 ms slower than left-hand responses ($F(1,300) = 17.64$, $P < 0.0001$) and at the 5.5° eccentricity the difference was 147.6 ms ($F(1,299) = 16.63$, $P < 0.0001$).

The interaction between hand and field was significant at the 2.5° eccentricity ($F(1,300) = 25.91$, $P < 0.0001$), but not at 5.5° ($F(1,299) = 0.74$, $P < 0.0001$). Despite this discrepancy, the interaction between eccentricity, hand and field was not significant ($F(1,699) = 1.49$, n.s.), even though the CUD at 2.5° was more than double that at 5.5°. These rather paradoxical findings might be attributed largely to the significantly higher variance of the RTs at the 5.5° eccentricity ($F(299,300) = 5.40$, $P < 0.0001$). The CUDs at the two eccentricities are shown in Fig. 4, which plots shows L.B.'s mean RTs for crossed and uncrossed conditions, along with those from Part 1. L.B. responded on average 111 ms faster for the 2.5° eccentricity than for the 5.5° eccentricity ($F(1,599) = 33.28$, $P < 0.0001$).

L.B. responded on average 132 ms faster in Part 1 than in Part 2. Despite this large difference in RT, his average CUDs in the two parts (56 ms and 48 ms, respectively) did not differ substantially.

Results: R.B.

R.B. was only tested at the 2.5° eccentricity, and only 0.94% RTs were omitted because they lay outside the range of 133–833 ms. His mean RT was significantly shorter to left- than to right-visual-field stimuli ($F(1,301) = 4.55$, $P < 0.05$) and there was a significant interaction between hand and field ($F(1,301) = 5.41$, $P < 0.025$), reflecting a CUD of 12 ms. There was a significant main effect of the delay between trial initiation and stimuli onset ($F(3,301) = 17.68$, $P < 0.0001$), but delay did not interact with any of the other factors.

Figure 5 presents R.B.'s mean crossed and uncrossed RTs and corresponding CUD for both Part 1 and Part 2. As with the control subjects, R.B.'s RTs were longer in Part 2 than in Part 1 at the 2.5° eccentricity; the mean difference was 70 ms. However, the CUD in Part 2 (12 ms) was shorter than that in Part 1 (26 ms), indicating that equiluminance did not lengthen the CUD and if anything, shortened it. R.B.'s mean RT was almost identical to the mean for the control subjects ($F(1,79) = 0.03$, n.s.) (Fig. 3), but the significant interaction between hand, visual field and the comparison between R.B. and the controls was highly significant, ($F(1,79) = 20.93$, $P < 0.0001$), indicating that R.B.'s CUD was reliably longer than normal.

Discussion

Part 2 was based on the supposition that equiluminant stimuli should largely, if not completely, restrict visual processing to the parvocellular division of the geniculostriate projection system. As shown in Figs 3–5, overall RT was increased by equiluminance, but the CUD was not; in fact it was reduced, if anything. This provides added evidence that transfer was not accomplished via a callosal channel sensitive to luminance.

It was also clear that interhemispheric transfer was not in any way impeded in either L.B. or R.B. by equiluminance. Since one would expect subcortical visual pathways to be insensitive to equiluminance, this result provides strong evidence that the information transferred was not visual. As in the normal subjects, it is likely that the information transferred had to do with response initiation rather than stimulus detection. Alternatively, the CUD of L.B. and R.B. might reflect ipsilateral motor control, which is less effective than contralateral control. This possibility has been suggested by Clarke and Zaidel [9] for both L.B. and an acallosal known as M.M.

General Discussion

When averaged over all conditions, the CUDs increased from 4.9 ms in the control subjects, to 23.3 ms in the acallosal subject R.B., to 53.1 ms in the commissurotomized subject L.B., confirming previous evidence that callosal agenetics lie between normal and commissurotomized people with respect to the speed of interhemispheric transfer. This pattern was present with both non-equiluminant (Part 1) and equiluminant (Part 2) stimuli.

Furthermore, all three groups showed a clear lengthening of overall RT with diminishing luminance-contrast. This trend is already present in Part 1 and there was a further increase with equiluminance in Part 2. This increase might also be due in part to a shift from the fast but colour-blind magnocellular system to the slower parvocellular pathway [26], since the equiluminant stimuli used in Part 2 should, in principle at least, have largely reduced, if not eliminated, both the magnocellular and subcortical visual responses. Surprisingly, this did not affect either callosal or extracallosal transfer, as CUDs were not prolonged. If anything, they were reduced, with one exception: In Part 1, L.B.'s CUD at the 2.5° eccentricity increased from an average of 39 ms for the non-equiluminant stimuli to 78 ms for the equiluminant stimuli.

Any attempt to explain the effects of eccentricity or luminance on the CUD must be weighed against the apparently random variation in CUD between blocks of trials [7, 16]. Figure 2 shows, for example, that L.B.'s CUD under equiluminance was approximately double that under the non-equiluminant conditions at the 2.5° eccentricity, while the reverse effect occurred at the 5.5° eccentricity, where the CUD under equiluminance was approximately half that under luminance contrast. When averaged over the two eccentricities, the CUD was 53 ms under luminance contrast and 55 ms under equi-

luminance, suggesting that equiluminance had no systematic effect on CUD. It may of more than passing interest, moreover, that the CUDs are approximately bimodal, clustering either around 40 ms or around 80 ms. These values may reflect alternative interhemispheric pathways and the choice of pathway may depend more on unknown random influences than on stimulus conditions.

A similar variation is evident in Fig. 3, which plots the results from R.B. His CUDs also appear to be approximately bimodal, clustering either around 15 ms or around 30 ms. It may be no more than coincidence that in the case of both L.B. and R.B., the ratio of the two modal values are approximately 1 : 2 and that L.B.'s modal values are approximately twice those of R.B.

Taken overall, the data suggest very little systematic variation in CUD with variations in luminance, including equiluminance. In the case of L.B., this is an especially striking result, since transfer can only have occurred via subcortical pathways. Hoffmann *et al.* [15] have shown that in monkeys transection of the corpus callosum leads to a loss of ipsilateral visual field information in the cortex and consequently in the corticopretectal pathway. If the afferent pathway from retina to superior colliculus can be ruled out under equiluminance [26, 32], it might still be argued that there was transfer via the collicular commissures after cortical processing. There are substantial subcortical efferents from the visual cortex (areas 18, 19 and 17) to the superior colliculus. These fibres are topographically organised [36, 42]. If the same pathways were also used with non-equiluminant displays in Part 1, this might explain the variation in CUD with equiluminance in Part 1, although it does not explain the reverse effect in Part 2—as noted above, these variations might simply reflect the apparently random fluctuations that plague this area of research and that may in turn reflect alternative transfer routes. Besides the superior colliculus, another possible transfer route is the cerebellum [13].

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