Interhemispheric neural summation in the split brain with symmetrical and asymmetrical displays

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Abstract
The present study, investigates interhemispheric integration in the split brain. Four split-brained, two acallosal and 14 normal subjects carried out a simple reaction time task in which they responded to stimuli presented either singly in the left visual field, singly in the right visual field, or simultaneously in both visual fields. Stimuli were white against a black background and bilateral stimuli were either symmetrical or asymmetrical around the central vertical meridian. For unilateral stimuli, the difference in response time (RT) between crossed and uncrossed hand-field combinations (crossed–uncrossed difference, or CUD) measured interhemispheric transfer time. RTs to bilateral and unilateral stimulus displays were compared to provide a measure of redundancy gain (RG). Normal subjects exhibited small CUDs and RGs. Split-brained and acallosal subjects were found to have much longer CUDs, and to show enhanced RGs which could not be explained by a probability (race) model, implying subcortical neural summation. This summation did not depend on the preservation of symmetry, suggesting that it may not occur at the retinotopically organized superior colliculus, but at another site such as the pons or reticular formation. © 2002 Elsevier Science Ltd. All rights reserved.

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1. Introduction
A well established finding in studies of simple reaction time is that response times (RTs) to stimuli such as luminance onsets are faster if there is more than one stimulus presented. This is known as the redundancy gain (RG) and can often be explained as the result of probability summation [25,35]. If the two stimuli trigger independent response preparation processes and a response is made as soon as the first process is completed, then on average, responses will be made faster than when there is only one process in operation, as long as the two RT distributions overlap. As RTs to redundant signals are determined by the first process to be completed, this is known as the “race model”. If however there is an enhancement of RT greater than that which can be explained by probability summation, then coactivation or neural summation is implied [7,36]. Coactivation models involve activation from redundant processes combining towards reaching a single criterion for response initiation [25,30]. Thus, activity on otherwise independent channels is summed together to produce a speeded response [36].

The race model seems sufficient to explain RG data in most normal subjects [29,36], although there are exceptions [28]. However split-brained and acallosal subjects often show RGs which exceed the predictions of the race model by a far greater extent than the small violations occasionally observed in normal subjects [7,18,33,36]. This is despite the fact that redundant stimuli, displayed one in each visual field, are presented separately to hemispheres which have been disconnected at the cortical level by commissurotomy or callosotomy, or by congenital failure of callosal fibers to develop, as in callosal agenesis. Indeed bilateral presentation seems to be a necessary condition for race-model violations in at least one split-brained subject, since there was no violation when redundant targets were presented to a single hemisphere and the split-brained subject showed similar mean RT effects from redundant targets as normal subjects [36]. In addition to this, crossed responses, or those made by the hand contralateral to the visual field in which the stimulus was displayed, are usually slowed relative to uncrossed responses. In normals this crossed–uncrossed difference (CUD) is typically in the range of 2–6 ms [2,32] and has been used as an index of callosal transmission times. In split-brained subjects it is much longer, commonly around 70 ms, although there is considerable variation [7,14,36]. The CUD is also prolonged in acallosal subjects for whom CUDs of around...
30 ms are often observed. This delay may either reflect slow sub-cortical transmission [5,26], or the use of a relatively inefficient ipsilateral motor pathway in making responses [36]. This pairing of long CUDs due to hemispheric disconnection in these patients with large RGs from stimuli presented separately to each hemisphere is paradoxical.

If race-model violations in the split-brain depend upon subcortical convergence of information from separated hemispheres, and the prolonged CUDs in split-brained subjects reflect slow sub-cortical transmission, this slowing of crossed responses relative to uncrossed responses should make the convergence of processing from separate channels less likely. In turn this should serve to reduce the possibility of neural summation occurring, whereas in fact summation is enhanced in these subjects. Reuter-Lorenz et al. [36] found no strong effect of asynchrony of presentation on the RG, and hence concluded that the delay that causes the CUD happens at a later stage of processing than the convergence which produces a RG in the split brain. This delay was attributed, not to interhemispheric transmission, but to the use of the relatively inefficient ipsilateral pathway in making crossed responses.

Reuter-Lorenz et al.’s [36] account of the enhanced RG posited a hypothetical “and” gate which removed tonic response inhibition only when response preparation signals were received from both hemispheres. In the split brain this scenario would only occur upon presentation of bilateral target stimuli, while callosal transfer in the intact brain would ensure it occurred when unilateral targets were presented. This explanation relies on the summation, not of sensory information from both hemispheres, but rather of response preparation signals, possibly in the cerebellum.

An alternative explanation of race-model violations involves the summation of sensory information, possibly at the superior colliculi. The colliculi form part of the retino-tectal visual pathway in mammals. Animal research suggests each superior colliculus contains a representation of the contralateral visual field [42] which is organized retinotopically [12,23,24,37]. The overall picture to emerge from cat and monkey studies is that the central visual field is represented more rostrally in the superior colliculi, while the peripheral visual field has a more caudal representation [43,44]. Commisural fibers connect rostral areas in each of the colliculi, with some fibers connecting corresponding points [11].

Collicular neurons are closely connected to the cortex. They receive retinal input from Po and Py ganglion cells [31] and both send fibers to and receive projections from the cortex. Afferent fibers ascend from the superior colliculi via the pulvinar nucleus to visual association cortex [14,28], in particular to parietal (dorsal stream) locations, where collicular input seems important [27]. In turn the colliculi receive cortical input from the primary visual cortex [22,24,45] and parietal association cortex, including the lateral suprasylvian and anterior ectosylvian areas [21,48]. There is evidence that retinotopically organized locations in the visual cortex send fibers to locations in the superior colliculi, which receive corresponding retinal input [22,45], although this may be an oversimplification [39]. This rich connectivity makes the superior colliculus a good candidate for the site of neural convergence.

An explanation of race-model violations invoking summation of sensory information is supported by data showing an effect of visual manipulations on the RG. Although Reuter-Lorenz et al. [36] found no strong effect of asynchrony of processing, the RG did reduce when stimulus luminance was reduced. Corballis [7] found that when redundant targets were presented as hue changes, equiluminant with the display background, the large RGs shown by three surgical split-brained cases were greatly reduced. As equiluminance should restrict visual processing to the retina-geniculate parvocellular system, this result suggested that the involvement of the superior colliculi may have been a factor in producing a RG sufficiently large to violate race-model predictions. RGs have also been shown in hemispherectomized patients in whom the collicular visual system has been spared [46], and race-model violations were observed in subjects with left visual field extinction as a result of right hemisphere damage [20]. Thus intactness of the superior colliculi may be important for the summation of sensory information, while cortical intactness is not.

Previous studies showing an enhanced RG in responses to luminance onsets have used displays with bilateral stimuli placed equidistant from the central fixation point [7,36]. Thus stimuli in these experiments are symmetrical around the fixation point, and evidence suggests that bilateral stimuli have to be presented across the visual meridian for neural summation to result [36]. As Corballis [7] pointed out, it remains to be seen whether neural summation occurs when bilateral stimuli are presented to locations that are not mirror images across the midline. A number of findings suggest that relations between mirror-symmetric locations across visual fields may be important, especially where subcortical commissures are implicated.

Poppel and Richards [34] found that residual visual function in the hemianopic fields of two patients was only found in locations that were mirror-symmetric with respect to small scotomas in their otherwise intact fields, suggesting that points in the two fields are related at the collicular level. These islands of residual function were small and resembled the scotomas in shape, suggesting that collicular interactions can occur between locations that are quite precise mirror images of one another. In normal subjects, threshold elevation for repeated light flashes could be reset by presenting light flashes at the mirror-symmetric location in the opposite field [41]. In addition the split-brained subject L.B. was found to be better at judging whether the number of dots in the two visual fields were the same or different when they were mirrored across the midline, compared to when they were arranged in the same pattern in each field [10]. Symmetry is one property identified early by the Gestaltists as important for perceptual salience and Julesz [19] has
suggested that this may be due to symmetrical organization of the central nervous system.

In the present experiment, we examine whether mirror-symmetric presentations are necessary for the enhanced RG to occur. Normal, split-brained and acallosal subjects were tested with bilateral stimuli that were either symmetrically or asymmetrically located across the midline. If the enhanced RG depends on symmetry, this might be taken as evidence for the superior colliculus as the site of neural summation. If the enhanced RG occurs regardless of stimulus location, this would support summation at a subcortical site where interhemispheric mapping is nontopographic, perhaps in the pons or cerebellum, rather than the superior colliculus.

2. Method

2.1. Subjects

We studied a total of 20 subjects, four of whom had undergone surgical section of the corpus callosum. Two subjects had agenesis of the corpus callosum and 14 were assumed to be neurologically normal. The split-brained subjects are known as J.W., V.P., N.G. and A.A., while the acallosal subjects are known as M.M. and J.P. Consent to test was obtained from all subjects.

2.2. Split-brained subjects

J.W. is a 45-year-old right-handed male who underwent a staged calllosotomy at the age of 26 for the control of intractable epilepsy. V.P. is a 47-year-old right-handed woman who also had staged callosal surgery. The details of these two subjects’ neurological profiles can be found in Gazzaniga et al. [15]. Sagital MRI demonstrated complete resection of the corpus callosus in J.W., with sparing of the anterior commissure. In case V.P., sagittal MRI revealed resid-ual callosal fibers in the area of the splenium and rostrum and an intact anterior commissure. Subsequent MRI examination has not revealed any splenial residual fibers (Paul M. Corballis, personal communication, March 2001). N.G. is a 66-year-old right-handed woman who underwent complete section of the corpus callosum, massa intermedia and both anterior and hippocampal commissures at age 30. A.A. is a 49-year-old right-handed man who also underwent a complete cerebral commissurotomy where the corpus callosum, massa intermedia and both anterior and hippocampal commissures were severed. The completeness of the calllosotomy in these two patients was later confirmed by magnetic resonance [3]. The anterior commissure was not observed in this study, but for technical reasons it was not possible to tell whether it was completely severed.

2.3. Acallosal subjects

M.M. is a 40-year-old right-handed woman. She was slow to reach all developmental milestones and following the birth of her first child at age 17 she suffered epileptic seizures. MRI scans taken at the age of 34 revealed complete absence of the corpus callosum and an anterior commissure which fell within normal size limits. No other abnormalities were reported [9]. J.P. is a 33-year-old right-handed woman. MRI scans showed the corpus callosum to be entirely absent and prominent antero-posteriorly oriented bands of white matter (bundles of Probst) were observed. J.P.’s anterior commissure is approximately nine times normal area in cross-section and the massa intermedia is slightly more prominent than usual.

2.4. Normal subjects

The 14 control subjects included the experimenter and 13 paid student volunteers. There were four females and 10 males with ages ranging from 20 to approximately 40 years. All but one of the subjects were under 30 in age. Three were left-handed (all male) and the remainder were right-handed. All had normal or corrected-to-normal visual acuity.

2.5. Stimuli and procedure

Stimuli were filled white circles, subtending approximately 1.0° in visual angle, flashed to the left and right visual fields, on each side of a central fixation cross. There were two stimulus locations in each visual field (near and far from fixation), both horizontally aligned with the fixation cross. The inner edges of the more centrally presented (near) stimuli were 2.1° from fixation while the inner edges of the more peripherally presented (far) stimuli were located 7.2° from fixation. These locations ensured that the stimuli were displayed well outside any visual field overlap and were thus presented only to the hemisphere contralateral to the visual field in which they appeared [13].

These stimulus locations should also be appropriate for investigation of the effect of asymmetry on neural summation. In the superficial layers of the mammalian superior colliculus, where neurons which are exclusively visually responsive reside, receptive fields are small relative to the larger fields of deeper multi-modal neurons [24]. Evidence exists for high resolution in the mapping of receptive fields in the primate colliculus. For instance, Goldberg and Wurtz [17] found field sizes as small as a few tenths of a degree in diameter in superficial layer neurons. Receptive fields of superficial layer neurons are smallest within 10° of fixation, and fields in this region are typically smaller than 10 degrees² in area [47].

Both near and far stimuli in the present experiment were presented within 10° from fixation. In order for a receptive field to be impinged upon by both near and far stimuli in one visual field, a receptive field would have to extend from the outer edge of the near stimulus to the inner edge of the far stimulus. Assuming the receptive field was circular, this would require an area of 13.2 degrees², a field larger than those typically found in this region. Thus asymmetric bilateral stimuli...
should stimulate discrete and asymmetric locations in the superficial layers of each colliculus. It follows that if homotopic regions of each colliculus are more strongly connected than nonhomotopic areas, and neural summation occurs at the colliculi, evidence for neural summation should be maximised when mirror-symmetric stimuli are presented.

The stimuli were presented, on an SVGA monitor using the software package Micro Experimental Laboratory [40]. Due to testing constraints at different sites, experiments were run with J.W. and V.P. using different monitors. A third monitor was used for N.G. and A.A., a fourth was used to run M.M. and a fifth was used to run J.P. and the 14 control subjects. J.W. was seated with his eyes at 57 cm from a 15 inch screen. All other subjects were seated at 63 cm from a 17 inch screen. Care was taken to ensure that visual angles were strictly preserved between experimental set-ups and that screen luminance and ambient room illumination was roughly equivalent for all testing situations.

The experiment consisted of 768 trials, made up of equal numbers (80) of eight different stimulus combinations, plus 126 catch trials in which no stimuli appeared. Single stimuli were presented in each of the four stimulus positions (left or right visual field and near or far from fixation). Bilateral stimuli were either symmetrical across the vertical meridian (Near-Left and Near-Right, or Far-Left and Far-Right) or asymmetrical (Near-Left and Far-Right, or Far-Left and Near-Right). This experimental design resulted in equal numbers of unilateral and bilateral trials. Each subject completed eight blocks of 48 trials with each hand, yielding a total of 384 trials with each hand. The hand used for responses alternated with each block, with all subjects beginning with the left hand. A practice block of 10 trials with the left hand was completed prior to beginning the experiment.

Blocks were initiated by the subject pressing the space-bar. The fixation cross appeared and remained on the screen for the duration of each trial. Subjects were instructed to keep their gaze at this location. On each trial a 250 Hz tone of 200 ms duration was sounded to warn the subject of the imminent stimulus arrival. On trials where stimuli were presented there was a variable interval of 300, 400, 500, 600 or 700 ms before the stimuli appeared. This variable interval was used to increase the subjects’ uncertainty about when stimuli would arrive, forcing them to wait for the onset of the stimuli and thereby reducing the number of anticipatory responses made. In every block each stimulus condition was paired once with each of the variable delays. This yielded 40 stimulus trials per block, which were randomly ordered.

Stimuli were displayed for 133 ms and responses were made by pressing the N key as fast as possible. The short display time ensured that subjects had insufficient time in which to direct a saccade towards the stimulus location. Following the offset of the stimulus, the subjects had 1000 ms in which to make a response. Responses faster than 133 ms (that is, those made while the stimuli were still on the screen) were treated as anticipations and not recorded. If a response was not made to a stimulus, the trial was re-run before the end of the block. This was to ensure that each stimulus condition yielded an equal number of RTs at the end of the experiment. On catch trials, subjects were required to withhold responses for 1.7 s following the warning tone. If a response was made to a catch trial, the trial was re-run during the block. Following either a response or the end of the response period, the fixation cross disappeared briefly and then returned to the screen. An interstimulus interval of 1000 ms then began before the 250 Hz warning tone sounded for the next trial.

3. Results

3.1. Reaction times

3.1.1. Subject Identifiers

Initials will be used in the results section to identify all subjects other than the control group. N.G. and A.A. will be referred to as commissurotomized, J.W. and V.P. as callosotomized, and M.M. and J.P. as acallosal.

3.1.2. Errors

A very small number of catch-trial errors were made by the control group. Only four such errors were recorded for each hand from the entire data pool. Most subjects made no responses to catch trials. Slightly more misses were made, 21 with the left hand and 26 with the right hand, which amount to negligible proportions of the total data pool (4480 stimuli presentations).

The split-brained and acallosal subjects also made very few errors, although callosotomized subject J.W. had some difficulty detecting unilateral stimuli in the Far-Right location when responding with his left hand. This stimulus-response combination resulted in 11 missed-stimulus presentations. No other condition resulted in more than five missed-stimulus errors for any of the split-brained or acallosal subjects, and none of these subjects made more than one catch-trial error.

3.1.3. Reaction times

Medians were used as the simplest measure of central tendency which was free of contamination by outliers. Median RT to each stimulus display condition was calculated for each individual subject. Average median RT across the 14 control subjects was also calculated for each condition. Averages across all conditions were then calculated for each subject and the control group, and are shown below in Table 1. The CUD was calculated by subtracting each subject’s RT to uncrossed stimuli, or those appearing in the visual field ipsilateral to response hand, from the RT to crossed stimuli. RG was calculated by subtracting each subject’s average median RT to bilateral trials from their average median RT to unilateral trials.

3.1.4. The crossed–uncrossed difference (CUD)

CUDs were calculated separately for each hand and for each individual subject. An analysis of variance, including
The factors of response hand and stimulus combination were carried out on the CUDs for the 14 control subjects. Three orthogonal contrasts were included to investigate the following specific questions. The first contrast compared CUD for Near presentation versus CUD for Far presentation. This contrast is referred to as Eccentricity. The second compared CUD for Near-Left and Far-Right presentation versus CUD for Far-Left and Near-Right presentation. This contrast is referred to as Direction of Asymmetry. The third compared CUD for symmetrical displays versus CUD for asymmetrical displays and is referred to as Symmetry.

The control subjects produced a CUD that was significantly greater than zero \( (F(1, 13) = 20.67, P = 0.001) \). There was no significant effect of stimulus eccentricity \( (F(1, 13) = 0.449, P = 0.514) \), nor of direction of asymmetry \( (F(1, 13) = 0.045, P = 0.835) \) on the CUD. The effect of symmetry approached significance \( (F(1, 13) = 3.717, P = 0.076) \). Control subjects had, on average, slightly larger CUDs for asymmetrical displays (7.6 ms) than symmetrical displays (4.5 ms).

### 3.1.5. Split-brained and acallosal subjects

Subsequent separate analyses, including the same contrasts as mentioned above, compared each individual neurological subject with the controls, so that each neurological subject was treated as a separate group. Differences between the controls and each subject would thus be revealed by interactions with the factor of group. In each group comparison the error variance in the data was defined solely by the performance of the control group, while single CUD values were entered for each condition for one neurological subject only. Each group comparison thus tests whether the data from an individual split-brained or acallosal subject fell within the bounds of normal performance.

All but one of the split-brained and acallosal subjects produced greater CUDs than the control group, as shown in Table 1. These differences were significant for N.G. \( (F(1, 13) = 20.67, P = 0.001) \), A.A. \( (F(1, 13) = 80.267, P = 0.001) \), J.W. \( (F(1, 13) = 158.165, P = 0.001) \), V.P. \( (F(1, 13) = 65.558, P < 0.001) \), and J.P. \( (F(1, 13) = 13.965, P = 0.002) \). The difference was not significant for acallosal subject M.M. \( (F(1, 13) = 0.009, P = 0.925) \). Three subjects showed a further interaction between hand and group, N.G. \( (F(1, 13) = 288.081, P < 0.001) \), A.A. \( (F(1, 13) = 5.497, P = 0.036) \), and V.P. \( (F(1, 13) = 8.081, P = 0.014) \). Table 1 shows that N.G. and A.A.

### Table 1

<table>
<thead>
<tr>
<th>Subject</th>
<th>Median RT</th>
<th>CUD</th>
<th>Redundancy gain</th>
<th>Median RT</th>
<th>CUD</th>
<th>Redundancy gain</th>
</tr>
</thead>
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<td>2</td>
<td>14</td>
<td>263</td>
<td>7</td>
<td>14</td>
</tr>
<tr>
<td>N.G.</td>
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<td>10</td>
<td>85</td>
<td>451</td>
<td>218</td>
<td>152</td>
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<tr>
<td>A.A.</td>
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<td>37</td>
<td>61</td>
<td>434</td>
<td>66</td>
<td>75</td>
</tr>
<tr>
<td>J.W.</td>
<td>363</td>
<td>71</td>
<td>61</td>
<td>358</td>
<td>33</td>
<td>47</td>
</tr>
<tr>
<td>V.P.</td>
<td>317</td>
<td>32</td>
<td>63</td>
<td>323</td>
<td>10</td>
<td>29</td>
</tr>
<tr>
<td>M.M.</td>
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<td>32</td>
<td>323</td>
<td>10</td>
<td>29</td>
</tr>
<tr>
<td>J.P.</td>
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<td>24</td>
<td>18</td>
<td>282</td>
<td>27</td>
<td>24</td>
</tr>
</tbody>
</table>

The results of the second contrast, comparing the direction of midline asymmetry, were more consistent across the six neurological subjects. Four subjects produced three-way interactions between direction of asymmetry, group and hand. Subjects A.A. \( (F(1, 13) = 21.995, P < 0.001) \), V.P. \( (F(1, 13) = 9.400, P = 0.009) \) and M.M. \( (F(1, 13) = 12.243, P = 0.004) \) showed larger CUDs with the right hand for Far-Left and Near-Right stimulus presentations than Near-Left and Far-Right presentations. With the left hand these subjects showed greater CUDs for Near-Left and Far-Right presentations than for Far-Left and Near-Right presentations. Commisurotomy subject N.G. \( (F(1, 13) = 17.307, P = 0.001) \) produced the exact opposite pattern of hand and direction of asymmetry effects to that shown by the three subjects mentioned above. Callosotomy subject J.W. produced a two-way interaction between direction of
asymmetry and group which approached significance $(F(1, 13) = 3.566, P = 0.081)$. J.W.'s CUDs to Near-Left and Far-Right presentations exceeded his CUDs to Far-Left and Near-Right presentations with both hands, but the difference was slightly greater in the left hand (29 ms) than the right hand (14 ms). The acallosal subject J.P. showed no significant interaction between direction of asymmetry and group.

The third contrast investigated the effect of symmetry on the CUD. None of the individual subjects were significantly different from the control group, suggesting that symmetry had little effect on the CUD for these subjects.

3.1.6. The redundancy gain

RGs were calculated separately for each subject for each of the four bilateral conditions. This was accomplished by subtracting the RT to the bilateral display from the mean of the RTs to the two corresponding unilateral displays. An analysis of variance, including the factor of bilateral condition, was carried out on the RGs for the 14 control subjects. As with the CUD analysis, three contrasts were included to investigate differences between conditions. The first of these compared RGs across symmetrical-near versus symmetrical-far bilateral displays and is referred to as Eccentricity. The second compared RGs for Near-Left and Far-Right presentation versus RGs for Far-Left and Near-Right presentation. This contrast is referred to as Direction of Asymmetry. The third compared RGs for symmetrical displays versus RGs for asymmetrical displays and is referred to as Symmetry.

The control subjects showed a significant RG $(F(1, 13) = 48.482, P < 0.001)$. There was a significant effect of eccentricity $(F(1, 13) = 5.054, P = 0.043)$, with symmetrical-far $(15.6 ms)$ displays producing larger RGs than symmetrical-near displays $(11.6 ms)$. There was no effect of direction of asymmetry $(F(1, 13) = 0.883, P = 0.365)$, nor of symmetry $(F(1, 13) = 0.008, P = 0.931)$ on the RG.

3.1.7. Split-brained and acallosal subjects

As with the CUD analysis, subsequent separate analyses compared each individual neurological subject with the controls across the factor of group. Again the error variance was defined by the control group.

Five of the six subjects showed significantly larger RGs than the control group. This was true for N.G. $(F(1, 13) = 193.672, P < 0.001)$, A.A. $(F(1, 13) = 50.838, P = 0.001)$, J.W. $(F(1, 13) = 34.942, P < 0.001)$, V.P. $(F(1, 13) = 31.155, P < 0.001)$ and M.M. $(F(1, 13) = 5.082, P = 0.042)$. The acallosal subject J.P. did not show a significantly greater RG than controls $(F(1, 13) = 0.940, P = 0.350)$.

None of the neurological subjects showed a significant interaction between group and the effect of eccentricity on RG. This suggests that these six subjects showed an effect of eccentricity similar to that shown by the control group, namely larger RGs to more eccentric stimuli. This was true for all subjects except for J.P. The degree to which each subject’s RG for symmetrical-far stimuli exceeded that for symmetrical-near stimuli were as follows: N.G. = 8 ms; A.A. = 16 ms; J.W. = 7 ms; V.P. = 3 ms; M.M. = 3 ms. J.P. showed a RG that was 4 ms larger for symmetrical-near stimuli.

The second contrast, concerning the direction of midline asymmetry, produced significant interactions between condition and group for commissurotomy subjects N.G. $(F(1, 13) = 6.930, P = 0.021)$ and A.A. $(F(1, 13) = 51.273, P < 0.001)$. Both of these subjects produced larger gains to Near-Left and Far-Right redundant stimuli than Far-Left and Near-Right displays, N.G. by 19 ms and A.A. by 55 ms. None of the remaining subjects showed significant interactions between the direction of asymmetry and group.

Only the commissurotomy subject N.G. produced a significant interaction between group and symmetry as defined by the third contrast. N.G. showed a bigger RG for symmetrical $(124 ms)$ than asymmetrical $(115 ms)$ displays $(F(1, 13) = 9.426, P = 0.009)$. All other subjects resembled the control group in showing no difference between RGs to symmetrical and asymmetrical displays, except for M.M. whose interaction between group and symmetry approached significance $(F(1, 13) = 4.252, P = 0.060)$. M.M. showed a slightly bigger RG for asymmetrical displays $(34 ms)$ than for symmetrical displays $(28 ms)$.

3.1.8. Alternative analysis

In the above analysis, the RG was calculated as the difference in RT between responses to bilateral stimuli and the average median RT to the two corresponding unilateral stimuli. This measure best reflects the race model, the analysis which has previously shown performance differences between split-brained, acallosal and normal subjects [7,36], as the race model compares responses to bilateral stimuli with responses to constituent unilateral stimuli. However, differences in the size of the RG between normal subjects and split-brained subjects may be due to differences in the size of the CUD. As the RT to unilateral stimuli is determined by averaging the RTs to crossed and uncrossed stimuli, a long CUD, such as those shown by split-brained subjects, will increase this value. This will have the effect of inflating the calculated RG for these subjects.

In order to test whether the larger RGs shown by the split-brained and acallosal subjects was entirely due to a prolonged CUD, the RG was also calculated as the difference between RTs to bilateral stimuli and RTs to the fastest unilateral stimulus (usually the uncrossed stimulus). This yielded a measure independent of the CUD, which was subjected to analysis of variance and group comparisons as before. The control subjects produced a significant RG $(9 ms)$ $(F(1, 13) = 21.003, P = 0.001)$. Three subjects showed RGs which were significantly greater than that shown by the control subjects. They were commissurotomy subjects N.G. $(62 ms)$ $(F(1, 13) = 45.251, P < 0.001)$ and A.A. $(40 ms)$ $(F(1, 13) = 15.688, P = 0.002)$, and callosotomy...
subject V.P. (23 ms) \(F(1, 13) = 5.307, P = 0.038\). Two subjects produced RGs which were bigger than that shown by the control group, but not significantly so. They were ca- losotomy subject J.W. (23 ms) and acallosal subject M.M. (24 ms). Acallosal subject J.P. produced a RG of 8 ms, 1 ms less than the control group.

Thus, when the possible inflationary effect of the CUD is controlled for there is still evidence that five of six surgically split-brained and acallosal subjects produced greater RGs than normal subjects, although the differences between these subjects and normal subjects are less apparent than when the CUD is included in the calculated RG. The relationship between the CUD and the RG has been explored in more detail elsewhere [6].

3.2. Cumulative probabilities

3.2.1. Control subjects

In order to gauge whether the RG evident in the RTs to bilateral stimuli exceeded that which could be explained by the race model as simple probability summation, cumulative probability functions were calculated for each hand and combination of stimuli. The race model was defined as follows. If responses to bilateral (S\(_{ic}\)) stimuli were calculated for each hand by the race model as simple probability summation, cumulative probabilities were calculated for each unilateral stimulus condition (for example, Far-Left only) against the corresponding bilateral stimulus condition (here, Far-Left and Far-Right). To calculate the values of \(P_{ic}, P_{i}, \) and \(P_{c}\) for each combination of unilateral and bilateral stimuli, probabilities were cumulated in bins of 10 RTs in which the first bin contained the 10 fastest RTs, the second bin the next 10 fastest, and so on. This was done separately for each subject and for each response hand. As each subject responded to 40 trials of each stimulus condition with each hand and probabilities were cumulated across two ipsilateral and one corresponding bilateral condition, there were 120 RTs in total which were sorted into 12 bins of 10. After accumulation the number of RTs in each bin were converted into probabilities for each stimulus condition, allowing the predictions of the race model to be tested.

Fig. 1 plots the mean of the expression \(P_{ic} – (P_{i} + P_{c})\) with standard error bars for all 14 subjects. The panel shows a separate plot for each combination of \(S_{L}, S_{R}, S_{c}\) and each response hand. The symmetrical stimulus displays are those labeled Mirror. Six of the plots have mean violation values which are greater than zero, four with the left hand and two with the right. All but one of these violations are confined to the fastest bin, although error bars for some slower bins do exceed zero on some plots. The mean violation values for the slower bins are well below zero, indicating that, for these bins, RG was less than that predicted by the race model. Of the plots which violate the race-model predictions, three represent data from symmetrical stimulus displays and three plot data from asymmetrical displays.

The two right-handed plots which violate the race model (Far-Left/Far-Right presentation and Far-Left/Near-Right presentation) exceeded zero by greater than two standard errors in the fastest bin, although to a very small degree (0.009 and 0.002, respectively).

3.2.2. Split-brained subjects

Cumulative probabilities were calculated for the split-brained subjects in the same manner as for the control subjects. Fig. 2 plots the expression \(P_{ic} – (P_{i} + P_{c} – P_{e}P_{c})\) for all four split-brained subjects. All subjects violated the race model to a far greater degree than the small violations observed in the control subjects. These violations were evident in every combination of display symmetry, eccentricity and response hand.

3.2.3. Acallosal subjects

Fig. 3 plots race-model violations for the two acallosal subjects. Both subjects show violations which exceed race-model predictions but these are less marked than those shown by the split-brained subjects. M.M. exhibited slightly greater violations than J.P., who showed no violations under some conditions (FL/FR: left hand and FL/NR: right hand). For M.M. there does not seem to be an obvious and systematic effect of display symmetry on the degree of violation. On five of the eight plots M.M.’s violations reach 0.1. Two of these plots are for symmetrical displays and three for asymmetrical displays. No systematic effect of display symmetry is apparent in violations show by J.P., which ranged from 0 to 0.2.
Fig. 1. Mean violation of the race model for 14 control subjects with standard-error bars. (FL/NR = Far-Left plus Near Right stimulus presentation).
Fig. 2. Race-model violations for all commissurotomized and callosotomized subjects.
Fig. 3. Race-model violations for both acallosal subjects.
4. Discussion

The experiment reported here replicates and extends the results of previous research. As expected, the split-brained and acallosal subjects showed CUDs that were prolonged relative to the mean for the normal subjects, although in one of the two acallosal subjects (M.M.) the difference was not significant. The CUD was unaffected by the eccentricity of the stimuli in the normal subjects, the acallosal subjects, and three of the four split-brained subjects. Only in commissurotomized subject A.A. was the CUD significantly different for far and near stimuli, and this relationship was not consistent across hands. The results therefore suggest that the CUD reflects the transfer of response information rather than stimulus information (cf. [5,14]). Alternatively, the prolonged CUDs in the split-brained and acallosal subjects may reflect initiation of crossed responses by the hemisphere ipsilateral to the responding hand (cf. [36]).

The results also replicate the enhanced RG in split-brained and acallosal subjects, clearly exceeding both the predictions of the race model and the RG in normals. This confirms previous evidence from J.W. [36], and from N.G. and A.A. [7], and extends the finding to callosotomy subject (V.P.) and two acallosal subjects (M.M. and J.P.) not previously tested on this task. Moreover, the extent of RG was related to the degree of disconnection; it was largest in the two subjects (N.G. and A.A.) with complete forebrain commissurotomy, smaller in the two subjects (J.W. and V.P.) with callosotomy, and smaller still in the two acallosal subjects. Moreover it was smaller (and nonsignificant) in the acallosal subject J.P., whose anterior commissure is considerably enlarged relative to that in normals, than in M.M., whose anterior commissure appears to be within normal limits. This pattern of results is similar to that seen by Iacoboni et al. [18]. Although these authors found no common anatomical denominator of neural summation, the degree of disconnection, as indexed by the CUD, was correlated with race-model violations. This relationship is consistent with the suggestion of Reuter-Lorenz et al. [36] that the RG in the split brain results from the removal of inhibition by bi-hemispheric processing of redundant targets, while callosal transmission in normal subjects ensures that inhibition is also removed for responses to unilateral stimuli, resulting in a smaller RG. Limited transfer in cases of callosal agenesis could account for the finding of a RG somewhat smaller than that seen in the surgically split-brain, but still larger than that seen in normals.

However it is unlikely that the effect is due to removal of response inhibition, as proposed by Reuter-Lorenz et al. [36], since it was largely abolished by equiluminance, at least in split-brained subjects [7], suggesting a sensory component. This result, together with the finding of Tomaiuolo et al. [46] for a RG following hemispherectomy, also implies that the effect is subcortical. The important finding of the present study is that the RG did not depend on the redundant stimuli being symmetrical about the midline. Only in one split-brained subject, N.G., were RGs significantly greater to symmetrical than to asymmetrical displays, but the race model was clearly violated in both cases. Similarly, although RGs were larger for more eccentric stimuli than for more centrally presented stimuli in all but acallosal subject J.P., the race model was still violated for both display types. On balance this means that the neural summation implied by the enhanced RG in the split-brained and acallosal subjects did not depend on topographic mapping. This in turn may be taken as evidence against the suggestion of Corballis [7] that the superior colliculus is the likely site of the summation effect in these subjects. If the superior colliculus is ruled out, possible alternative sites include the cerebellum [16], the pons, or the reticular activating system. For example, inputs may proceed via subcortical pathways from the retina to the pons, where they summate and activate the reticular formation, creating an arousal effect that speeds response. Alternatively, the activation summated may be response preparation signals from both hemispheres [36].

The question remains as to why violations of the race model are not evident in subjects with intact forebrain commissures, except perhaps with the shortest RTs under some conditions. One possibility is that the forebrain commissures somehow act to inhibit the summation. In normal subjects the processing of a stimulus by one hemisphere may suppress stimulus processing by the other hemisphere, via the corpus callosum. An inhibitory cortical interaction such as this may largely negate the facilitatory effect of subcortical neural summation, or could even reduce the RG to a level below that predicted by the race model [8]. An alternative explanation is not that the summation is inhibited by the forebrain commissures, but rather that cortical interhemispheric transfer allows for subcortical summation even when the input is unilateral. That is, a single unilateral stimulus is projected to the contralateral hemisphere, relayed via the forebrain commissures to the opposite hemisphere, which then projects to the subcortical site responsible for the summation effect. This summation would not occur in hemispherectomized subjects or in those with complete forebrain commissurotomy, but might be weakly present in those with callosotomy but with the anterior commissure still intact and in those with callosal agenesis. Since this cortical component depends on interhemispheric transfer prior to subcortical activation, the summation might be slightly delayed, which could explain why evidence for violations of the race model in the normal subjects was restricted to the shortest RTs only.

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