TABLE 2 Genetic correlations between stripe and behaviour scores for each population

Tenmile (29)	Distance	Reversal	Display	Speed
Stripe	−0.191	−0.751	0.150	0.280
	−0.173	−0.427**	0.106	0.219
Alsea (19)				
Stripe	0.384	−1.200	−0.180	0.635
	0.196	−0.499**	−0.060	0.291
McGribble (29)				
Stripe	0.491	−0.184	0.495	0.293
	0.187	−0.012	0.543*	0.303

The top row indicates covariance component estimate of the correlation based on full-sib data[2,22]. Litter mean correlations are presented on the second row for purposes of significance testing[4,25]. The number of litters for each population is shown in parentheses. Asterisks indicate the significance level of the litter mean correlation (** = $P < 0.01$; * = $P < 0.05$). See Table 1 for methods.

examined (Table 2). In two populations, significant negative genetic correlations were detected between the stripe index and the number of reversals during flight. In the third population, a significant correlation was observed between the stripe index and antipredator display, but this correlation was due almost entirely to a single family and it was disregarded.

Sudden terminations of flight, such as the reversals scored here, can cause a predator to lose track of its quarry, allowing the prey a second chance to use crypsis as a defence[16,17]. The correlations observed in the Tenmile and Alsea populations indicate that striped snakes flee directly whereas unstriped and spotted snakes exhibit a greater tendency for cryptic behaviour. This resembles the interspecific correlations between colour pattern and antipredator behaviour observed among all North American snakes[14].

Genetic covariances result from pleiotropy (whereby genes affect multiple traits) or linkage disequilibrium (nonrandom association of alleles at different loci) or both. The data presented here cannot be used to distinguish between these causes, but the genetic covariances probably result from linkage disequilibrium rather than pleiotropy, because the coupling is between morphological and behavioural traits. In either case, theory indicates that genetic covariances can be maintained by selection for particular combinations of traits[18]. The concordance between the correlations detected in this study and those observed across snake species indicates that selection favouring particular combinations of colour pattern and behaviour may be at least partially responsible for the maintenance of the genetic covariances reported here. Other phenomena that may contribute to the maintenance of genetic correlations include assortative mating, secondary contact of divergent populations, small effective population size and population subdivision[19].

Selection favouring combinations of traits could also help to explain the extensive colour pattern polymorphism within populations of *Thamnophis ordinoides*. If the fitness of an individual with a particular colour pattern depends on its antipredator behaviour, then it may be that individuals with different combinations of colour pattern and antipredator behaviour have comparable fitnesses. A similar argument has been used to explain polymorphism in heterogeneous environments where different genotypes choose to occupy the microhabitat in which they are most fit. Under these conditions genetic polymorphisms within populations can be maintained with small genetic loads[6].

The post-selection distribution of all characters will be a result not only of direct selection on those characters, but also of selection on genetically correlated characters. In this way, genetic covariances can constrain the rate[20] and even determine the direction[21] of short-term evolutionary change. Thus, genetic correlations between morphology and behaviour imply a high degree of genetic integration of the entire phenotype, indicating that phenotypic evolution is more complex than even multivariate analyses of a single category of characters would indicate. □

Received 17 April; accepted 26 October 1989.

1. Lande, R. *Evolution* **33,** 402-416 (1979).
2. Falconer, D. S. *Introduction to Quantitative Genetics* (Longman, London, 1981).
3. Lande, R. & Arnold, S. J. *Evolution* **37,** 1210-1226 (1983).
4. Emerson, S. B., Travis, J. & Blouin, M. *Evolution* **42,** 68-78 (1988).
5. Hutter, P. *Genetica* **72,** 193-198 (1987).
6. Taylor, C. E. *Genetics* **80,** 621-635 (1975); **83,** 887-894 (1976).
7. Giesel, J. T. *Evolution* **24,** 98-119 (1970).
8. Lees, D. R. *J. Zool., Lond.* **176,** 341-352 (1975).
9. Steward, R. C. *J. Zool., Lond.* **181,** 395-406 (1977); *Biol. J. linn. Soc.* **24,** 285-293 (1985).
10. Jones, J. S. *Nature* **298,** 749-750 (1982).
11. Stewart, A. J. A. *Biol. J. linn Soc.* **27,** 79-101 (1986).
12. Cavener, D. *Behav. Genet.* **9,** 359-365 (1979).
13. Lynch, C. B., Sulzbach, D. S. & Connolly, M. S. *Am. Nat.* **132,** 521-537 (1988).
14. Jackson, J. F., Ingram, W. III & Campbell, H. W. *Am. Nat.* **110,** 1029-1053 (1976).
15. Brown, J. F. *Psych. Forsch.* **14,** 199-232; 249-268 (1931).
16. Brodie, E. D. III *Am. Nat.* **134,** 225-238 (1989).
17. Pough, F. H. *Copeia* **1976,** 834-836 (1976).
18. Lande, R., *Genet. Res.* **44,** 309-320 (1984).
19. Endler, J. A. *Natural Selection in the Wild* (Princeton University Press, 1986).
20. Grant, P. R. *Ecology and Evolution of Darwin's Finches* (Princeton University Press, 1986).
21. Zeng, Z. *Evolution* **38,** 363-374 (1988).
22. Becker, W. A. *Manual of Quantitative Genetics* (Academic Enterprises, Pullman, Washington, 1984).
23. Arnold, S. J. & Bennett, A. F. *Anim. Behav.* **32,** 1108-1118 (1984).
24. Garland, T. Jr. *Evolution* **42,** 335-350 (1988).
25. Via, S. *Evolution* **38,** 896-905 (1984).

ACKNOWLEDGEMENTS. I thank S. J. Arnold, A. F. Bennett and D. and M. Brodie for providing space and equipment during this study, N. L. Reagan for help in collecting specimens and data, and S. J. Arnold, F. J. Janzen, R. Lande and M. Wade for comments on the manuscript. This research was supported by grants from Sigma Xi and the NSF to E. D. B. III, the NIH to S. J. Arnold and the NSF to A. F. Bennett.

Independent hemispheric attentional systems mediate visual search in split-brain patients

Steven J. Luck, Steven A. Hillyard, George R. Mangun & Michael S. Gazzaniga*

Department of Neurosciences, M-008, University of California, San Diego,
La Jolla, California 92093, USA
* Dartmouth Medical School, Hanover, New Hampshire 03756, USA

THE primate visual system is adept at identifying objects embedded within complex displays that contain a variety of potentially distracting elements. Theories of visual perception postulate that this ability depends on spatial selective attention, a mechanism analogous to a spotlight or zoom lens, which concentrates high-level processing resources on restricted portions of the visual field[1,2]. Previous studies in which attention was pre-cued to specific locations in the visual field have shown that the spotlight has a single, unified focus[2,3], even in the disconnected hemispheres of patients who have undergone surgical transection of the corpus callosum[4,5]. Here we demonstrate that an independent focus of attention is deployed by each of the surgically separated hemispheres in a visual search task, such that bilateral stimulus arrays can be scanned at a faster rate by 'split-brain' subjects than by normal control subjects. The attentional system used for visual search therefore seems to be functionally and anatomically distinct from the system that mediates voluntary orienting of attention.

In visual search tasks of the type studied here, an array of items is presented and the subject must decide whether or not a specified target item is present in the array. When the target and distractor items share the same basic features but differ in combinations or conjunctions of these features, attention must be focused on each item in turn (serial search) until the target is found or the array is exhausted[1,6]. Reaction time therefore increases linearly with the number of items in the array (the set size), and the slope of the function relating reaction time to set size indicates the amount of time required to scan each item (the search rate).

Several studies in normal subjects have indicated that the

TABLE 1 Mean per cent correct for the control subjects and split-brain patients

		Set size		
		2	4	8
Control	Unilateral	98.5 (1.6)	97.6 (2.4)	94.9 (2.9)
	Bilateral	99.6 (0.6)	99.4 (0.7)	97.9 (2.4)
Split	Unilateral	99.4 (0.8)	96.1 (5.5)	92.2 (1.6)
	Bilateral	98.3 (1.1)	98.4 (0.8)	97.2 (4.0)

Standard deviations are shown in parentheses.

attentional spotlight is unitary and cannot be divided effectively between spatially disparate regions of a stimulus array[2,3], except under certain conditions[7,8]. If visual arrays are scanned by serial movements of such a unitary attentional spotlight, then the search rate should be about the same whether the items are all presented to one visual hemifield or are equally distributed between the left and right hemifields. Alternatively, if each hemisphere in the split-brain patient controls its own attentional spotlight and can scan its respective hemifield independently of the other hemisphere, then the search rate should be faster for bilateral arrays than for unilateral arrays having the same set size. Presumably, intact interhemispheric communication should force normal subjects to maintain a single focus of attention for both unilateral and bilateral stimulus arrays[9].

The performance of commissurotomy patients J.W. and L.B., whose case reports have been described elsewhere[10,11], was compared with a group of six neurologically normal young adults using the visual search paradigm shown in Fig. 1. The stimulus arrays consisted of rectangles constructed from a red square and a blue square, with blue on top for the distractors and red on top for the target. Subjects pressed a left-hand button if the target was in the left visual field (LVF), and a right-hand button if the target was in the right visual field (RVF); neither button was pressed on target-absent trials. Our design included set sizes 2, 4 and 8, presented either unilaterally or bilaterally (bilateral stimuli were divided equally between the LVF and RVF). Stimulus order was completely randomized. To ensure that the items were processed only by the contralateral hemisphere, subjects were instructed to fixate a central point at all times and eye position was verified by measurement of the electro-oculogram. For all patients and control subjects, horizontal eye movements averaged one degree of visual angle or less, which was smaller than the distance between the fixation point and the stimulus arrays.

The reaction time functions for the control and split-brain subjects are shown in Fig. 2. The control subjects' search functions were very similar for unilateral and bilateral arrays; reaction times were somewhat faster overall for bilateral arrays, but

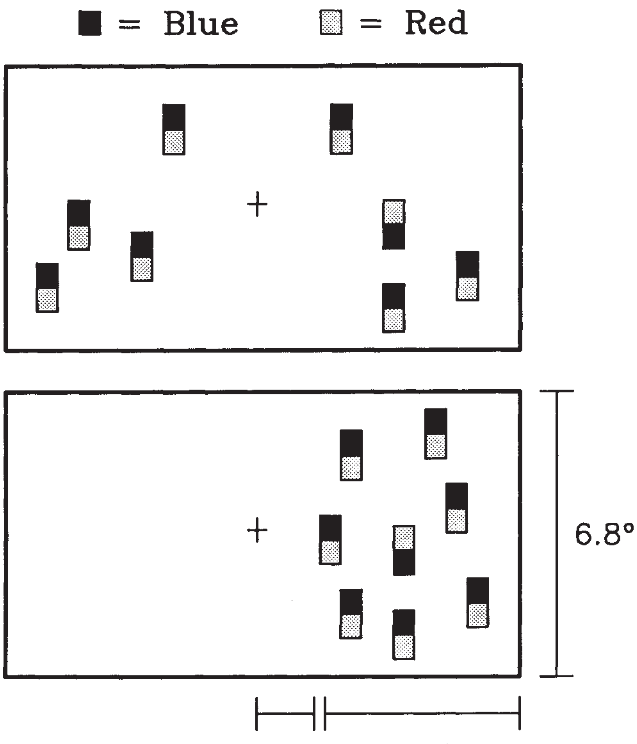

FIG. 1 Examples of stimuli used for the visual search task. The top panel is a bilateral array and the bottom panel is a unilateral array with the same set size (8 items). Each item subtended 0.4×0.8 degrees of visual angle. The items were placed in random positions within rectangular zones on the left and right sides; these zones measured 3.3×6.8 degrees and were displaced 1.1 degrees laterally from the fixation point. The arrays were presented for a duration of 2.5 s, with an interstimulus interval that varied randomly between 3.35 and 3.65 s. Unilateral and bilateral arrays of the various set sizes were randomly intermixed, and ~50% contained a target. Only one target could occur per trial. Each subject received a total of 792 trials.

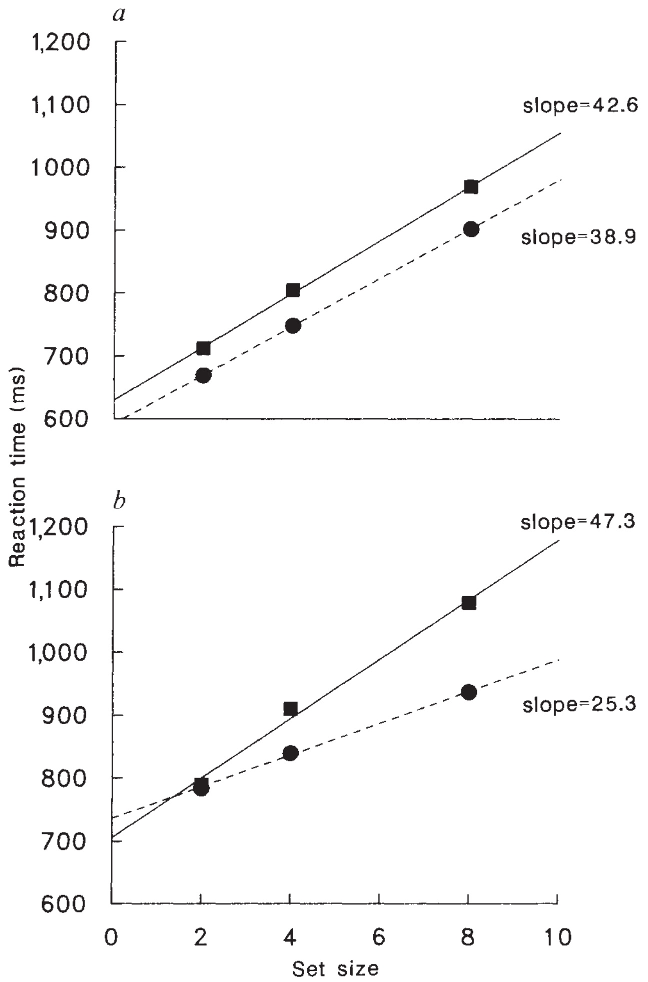

FIG. 2 Group mean reaction times as a function of set size for *a*, 6 normal control subjects and *b*, 2 split-brain patients. ■, Unilateral arrays; ●, bilateral arrays. In the control group, the search functions were essentially parallel for the unilateral and bilateral arrays, indicating a single focus of attention. For the split-brain patients, however, the slope was about twice as large for unilateral arrays as for bilateral arrays, suggesting that each hemisphere can search its corresponding hemifield independently of the other hemisphere.

there was no significant difference between the slopes of the reaction time functions for the two types of arrays (ANOVA, $P > 0{\cdot}4$). For the split-brain patients, however, the slope of the search function was about twice as steep for the unilateral arrays as for the bilateral arrays. This 2:1 ratio of slopes would be expected if each hemisphere had an independent serial scanning mechanism, such that bilateral arrays were scanned with two spotlights and unilateral arrays were scanned with only one.

Multiple regression analyses of the single-subject reaction times confirmed that bilateral search slopes were significantly faster than unilateral search slopes for both split-brain patients ($P < 0.001$ for J.W.; $P < 0.04$ for L.B.), but not for any of the control subjects. Moreover, these differences were not due to speed/accuracy trade-offs: accuracy actually tended to be higher for bilateral arrays than for unilateral arrays, and this pattern was present for both the control group and for the split-brain patients (Table 1).

These findings indicate that the serial scanning of visual arrays for conjunction targets is conducted independently in the disconnected hemispheres of split-brain patients. Visual search can therefore be added to the list of perceptual and mnemonic processes that can be performed in parallel by the right and left hemispheres after commissurotomy[12]. But our results stand in contrast to several previous studies indicating that attentional processes may be shared by the separated hemispheres through intact subcortical pathways[13-15]. In particular, Holtzman and his colleagues reported that split-brain patients cannot divide the attentional spotlight in tasks that use symbolic cues to manipulate the direction of attention[4,5], which contrasts with our evidence that attention can be divided during visual search. These divergent results point to the existence of two functionally and anatomically distinct systems that participate in visual selective attention: whereas the separated cerebral hemispheres can function independently in scanning arrays of objects during visual search, the advance orientation of attention by means of a symbolic precue seems to be a joint exercise of the separated hemispheres, presumably mediated by intact subcortical structures.

This proposed dichotomy is consistent with studies of normal subjects[16] showing that attentional orienting produced by symbolic precues does not improve the identification of feature conjunctions as effectively as the attentional processes deployed during visual search[1] or during the direct (peripheral) precuing of location[17,18]. Our results are also in accord with earlier studies of split-brain subjects indicating that the two hemispheres can process information in parallel during simple binary decision[9,19] and spatial memory tasks[20,21].

Although the search rates from the present experiment indicate that attention may operate independently in the disconnected hemispheres during the item-by-item search process, other aspects of the data provide evidence for some sharing of resources in this task. For the split-brain patients, the intercept of the search function was higher for bilateral arrays than for unilateral arrays, whereas the intercept was significantly lower for bilateral arrays in the control subjects. This finding indicates that increasing the processing load in one of the separated hemispheres may slow the overall target detection speed of the other hemisphere, although this interference does not affect the processing stage that conducts the serial target search. ☐

Received 17 July; accepted 9 October 1989.

1. Treisman, A. M. & Gelade, G. *Cog. Psychol.* **12,** 97–136 (1980).
2. Eriksen, C. W. & Yeh, Y. Y. *J. exp. Psychol., hum. Percept. Perform.* **11,** 583–597 (1985).
3. Posner, M. I. *Q. J. exp. Psychol.* **32,** 3–25 (1980).
4. Holtzman, J. D., Sidtis, J. J., Volpe, B. T., Wilson, D. H. & Gazzaniga, M. S. *Brain* **104,** 861–872 (1981).
5. Holtzman, J. D., Volpe, B. T. & Gazzaniga, M. S. in *Varieties of Attention* (eds Parasuraman, R. & Davies, D. R.) 375–394 (Academic, New York, 1984).
6. Eglin, M., Robertson, L. C. & Knight, R. T. *J. Cog. Neurosci.* (in the press).
7. McLeod, P., Driver, J. & Crisp, J. *Nature* **332,** 154–155 (1988).
8. Nakayama, K. & Silverman, G. H. *Nature* **320,** 264–265 (1986).
9. Ellenberg, L. & Sperry, R. W. *Neuropsychologia* **18,** 411–418 (1980).
10. Sidtis, J. J., Volpe, B. T., Wilson, D. H., Rayport, M. & Gazzaniga, M. S. *J. Neurosci.* **1,** 323–331 (1981).
11. Bogen, J. E. & Vogel, P. J. in *Les Syndromes de Disconnexion Calleuse Chez l'Homme* (eds Michel, F. & Schott, B.) 227–251 (Hôpital Neurologique, Lyon, 1975).
12. Gazzaniga, M. S. *Neuropsychologia* **25,** 119–133 (1987).
13. Kreuter, C., Kinsbourne, M. & Trevarthen, C. *Neuropsychologia* **10,** 453–461 (1972).
14. Teng, E. L. & Sperry, R. W. *Neuropsychologia* **11,** 131–140 (1973).
15. Lewine, J. D. & Doty, R. W. *Soc. Neurosci. Abstr.* **14,** 2 (1988).
16. Briand, K. A. & Klein, R. M. *J. exp. Psychol., hum. Percept. Perform.* **13,** 228–241 (1987).
17. Treisman, A. M. *Comput. vis. Graphics, Image Proc.* **31,** 156–177 (1985).
18. Prinzmetal, W., Presti, D. E., & Posner, M. I. *J. exp. Psychol., hum. Percept. Perform.* **12,** 361–369 (1986).
19. Gazzaniga, M. S. & Sperry, R. W. *Psychon. Sci.* **4,** 261–262 (1966).
20. Gazzaniga, M. S. & Young, E. D. *Expl Brain Res.* **3,** 368–371 (1967).
21. Holtzman, J. D. & Gazzaniga, M. S. *Neuropsychologia* **23,** 315–321 (1985).

ACKNOWLEDGEMENTS. This research was supported by the NIH and NIMH, by the ONR and by an NSF graduate fellowship to S.J.L.

Recovery of spatial learning deficits after decay of electrically induced synaptic enhancement in the hippocampus

C. A. Castro, L. H. Silbert, B. L. McNaughton* & C. A. Barnes

Department of Psychology, University of Colorado, Boulder, Colorado 80309, USA

A WIDESPREAD interest in a long-lasting form of synaptic enhancement in hippocampal circuits[1,2] has arisen largely because it might reflect the activation of physiological mechanisms that underlie rapid associative learning. As its induction normally requires the 'Hebbian'[3] association of activity on a number of input fibres[4], we refer to the process as long-term enhancement (LTE) rather than long-term potentiation (LTP), to emphasize its distinction from the ubiquitous, non-associative 'potentiation' phenomena that occur at most synapses, including those exhibiting LTE[5]. Among other evidence[6-8] that LTE might actually have a role in associative memory is the demonstration that repeated high-frequency stimulation, which saturated the induceable LTE, caused a severe deficit in spatial learning, although it had no effect on well established spatial memory[9]. These results were consistent with a widespread view that information need only temporarily be stored in the hippocampal formation in order for long-term memories to be established in neocortical circuits[10,11]. In this context, it is important to understand whether the possible underlying synaptic changes are of a permanent character, or are relatively transient. A second question is whether the actual cause of the observed learning deficit is the distruption of the synaptic weight distribution, and/or the limitation of further synaptic change, which presumably results from experimental saturation of the LTE mechanism. Alternatively, the deficit could be a consequence of some unobserved secondary effect of the high-frequency electrical stimulation. Here we demonstrate that learning capacity recovers in about the same time that it takes LTE to decay, which strongly favours the first possibility and supports the idea that LTE-like processes actually underlie associative memory.

Experiments were performed on 12 male F-344 rats (10 months of age). Under deep sodium pentobarbital anaesthesia, all rats underwent bilateral implantation of electrodes[9] for stimulation of perforant path fibres in the vicinity of the angular bundle and for recording the resulting synaptic and postsynaptic field potentials in the fascia dentata[9] (see Fig. 1). Following recovery from surgery, the rats were randomly assigned to three groups. Two groups ($n = 4$) received high-frequency, LTE-inducing stimulation, and the other group ($n = 4$) received only low-frequency test stimuli. The electrical stimulation and physiological recordings were carried out daily over a 34-day period. The

---

* To whom correspondence should be addressed.