jections has been mapped 4 to 6 weeks after optic nerve crush [M. G. Yoon, *J. Physiol. (London)* **257**, 621 (1976); R. L. Meyer, *J. Comp. Neurol.* **189**, 175 (1980)]. Ultrastructural studies show that a few retinotectal synapses reappear 3 to 4 weeks after optic nerve crush [L. R. Marotte and R. E. Mark, *Exp. Neurol.* **49**, 772 (1975); M. Murray, *J. Comp. Neurol.* **168**, 175 (1976)]. The recovery of synaptic density to a normal level, however, appears to take several months [M. Murray and M. A. Edwards, *ibid.* **209**, 363 (1982)].
8. P. H. O'Farrell, *J. Biol. Chem.* **250**, 4007 (1975). The modifications we used are described by P. Strocchi *et al.* [*J. Neurochem.* **37**, 1295 (1981)]. The lysis buffer and the gels contained 6 percent Ampholine, *p*H ranges 3.5 to 5, 5 to 7, and 3.5 to 10 (LKB) in the ratio 2:2:1.
9. W. M. Bonner and R. A. Laskey, *Eur. J. Biochem.* **46**, 83 (1974).
10. A rapidly transported 24K protein which is selectively enhanced in the frog regenerating optic nerve does not ordinarily migrate out of isoelectric focusing gels into SDS slab gels unless high concentrations of urea are present in the latter [J. H. P. Skene and M. Willard, *J. Neurosci.* **1**, 419 (1981)]. In our two-dimensional gels, the absence of a 26K protein that is differentially regulated by the tectum may be accounted for by such solubilization properties. Alternatively, the isoelectric point of the 26K protein may be outside the range of our gel system.
11. In other experiments (M. G. Yoon, L. I. Benowitz, F. Baker, in preparation), we crushed both optic nerves and ablated only one tectum. The differences in rapidly transported proteins seen in the two nerves of these animals were found to be similar to the differences between R+T and R−T groups described in the present report. This result suggests that diffusible factors deriving from the one intact tectum are not causing the two retinas to express similar proteins, and implies that the differences between R+T and R−T nerves reported here are likely to be mediated by surface-contact events.
12. Supported by National Institute of Neurological and Communicative Disorders and Stroke grant R01NS16943 (to L.I.B.) and by grants MT 4994 from the Medical Research Council and A 9756 from the Natural Sciences and Engineering Research Council of Canada (to M.G.Y.). We thank F. Baker for technical assistance.

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Stimulation of Catecholamine Secretion by Choline

Holz and Senter (*1*) observed that choline, in concentrations (1 m*M*) an order of magnitude greater than those present in tissues after choline or lecithin administration, can stimulate the secretion of catecholamines from primary cultures of bovine chromaffin cells, apparently by interacting with a nicotinic cholinergic receptor. Based on this finding, they propose that choline may exert its effects (presumably including the stimulation of adrenomedullary secretion) by acting as a partial nicotinic agonist.

This possibility has already been examined experimentally (*2*). Rats received a large oral dose of choline chloride (20 nmoles/kg) or placebo and urines collected during the next 24 hours were assayed for catecholamines. Administration of the acetylcholine precursor was associated with a several-fold increase in urinary epinephrine, and potentiated the increases in epinephrine secretion caused by treatments known to accelerate splanchnic nerve firing (for example, insulin; phenoxybenzamine). However, increases in urinary epinephrine after choline were not observed among rats previously subjected to bilateral adrenal denervation, even though the denervated adrenals continued to be perfused with amounts of choline similar to those that reach the intact organs. That the failure of the denervated organs to respond to the choline was not caused by loss of nicotinic receptors was shown by the fact that they did retain the ability to secrete epinephrine when animals received nicotine itself. In related studies (*3*), exogenous choline was found to induce the enzyme tyrosine hydroxylase in intact adrenal medullas, but not in the chromaffin cells of previously-denervated adrenals.

These observations provide strong support for the view that the effects of exogenous choline on adrenomedullary function require that the choline first be converted to acetylcholine within splanchnic nerve terminals.

It seems clear that, in high enough concentrations, choline itself can activate certain acetylcholine receptors (*4*). However, it remains to be demonstrated that this ability is at all related to the physiological effects seen after choline or lecithin adminstration.

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Failure to Integrate Information from Successive Fixations

In our report (*1*), we described an experiment providing evidence for an integrative visual buffer, a briefly lasting memory in which visual information from successive eye fixations is integrated and stored in proper spatial registry. We have encountered difficulty in replicating our original result in two new experiments. These experiments were motivated in part by a concern that physical persistence of the phosphor on our graphics display device may have contributed to the integration effect.

In the first experiment, a display consisting of 25 amber light–emitting diodes (LED's) were arranged in a 5 by 5 array. These LED's decay completely within nanoseconds when extinguished. All aspects of the experiment were as before (*2*), except that the LED display was mounted in a wooden frame over the face of the display device used in the original experiment. In addition, the LED's were both a different color (amber as opposed to white) and larger in diameter than the original dots. With this device, subjects were unable to identify the location of the missing dot accurately, a result unlike that of the previous experiment.

Because of the differences between the LED display and the display used in the original experiment, we attempted a replication using the same display device as in the original report (*3*), but with a filter placed over the display screen that dramatically attenuated the long-persistence component of the phosphor. Again, subjects were unable to perform the task at a level that reliably exceeded their control condition performance.

In the original report, we claimed that screen persistence could not have accounted for our results. We based this claim on several tests that space limitations prohibit describing here. The new results suggest either that our original tests were not sufficient and that the integration reported previously (*1*) was spurious, or that the result reported earlier is restricted to a very narrow range of stimulus conditions (that is, to stimuli with particular colors and forms).

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1. J. Jonides, D. E. Irwin, S. Yantis, *Science* **215**, 192 (1982).
2. Subjects fixated a central point. A randomly chosen 12 of the 25 dots appeared for approximately 150 msec in the right periphery. Subjects then executed a saccade to the array location; during this time, no dots were present in the display. After the eyes reached the saccade goal, another set of 12 dots was displayed for 20 msec. Subjects then attempted to identify the matrix location in which no dot appeared.
3. A Digital Equipment Corporation VT-11 graphics display device with P-4 phosphor. According to the manufacturer's specifications, the phosphor decays to 1 percent of its initial brightness within 0.5 msec, and to 0.1 percent within 20 msec.
4. We thank R. Abrams and J. Sullivan for their help in constructing and implementing the display used in experiment 1.

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Stimulation of catecholamine secretion by choline

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