hypnosis condition were primarily responsible for the increase in output, and hypnotic suggestion was no more potent than task motivating suggestion for those lower in hypnotic ability (Fig. 2).

A two-way analysis of variance based on the total increase in items indicates a significant main effect for condition [$F(1, 50) = 5.63$, $P < 0.03$] and a significant interaction of condition with hypnotic susceptibility [$F(1, 50) = 4.31$, $P < 0.05$]. When just the correct information was considered, the interaction between condition and hypnotic ability was significant as well [$F(1, 50) = 4.95$, $P < 0.05$]. Using new errors as the dependent measure yielded a significant main effect for condition [$F(1, 50) = 5.38$, $P = 0.03$], but the interaction in this case was not statistically significant [$F(1, 50) = 3.10$, $P = 0.08$]. Even though hypnotizable subjects in the hypnosis condition showed a statistically significant increase in accurate recall, this increase was small in absolute terms. No subject in even this most responsive group retrieved more than five new correct items (mean = 1.40), and six of them failed to produce any new correct information at all. The cost of correctly recalling these few items was considerable, since it was accompanied by almost three times as many errors as were made by subjects in any other condition. We have replicated this pattern of results on a new sample of 56 subjects (*9*).

The probability of correctly recalling new items under hypnosis seems directly related to the number of items a subject is willing to report as memories, a finding that could be interpreted as being due to a shift in report criterion. That is, the increase in correct recall may not represent increased sensitivity to memory traces, but may instead result from less caution by subjects in what they are willing to report as memories. This criterion shift could be attributed to various demand characteristics, social cues, and expectations engendered by the hypnotic situation.

Another possible explanation for the effect of hypnosis on memory depends less on a shift in the report criterion than on the frequency with which the individuals' criterion for memorial judgment is subjectively met. Hypnosis may heighten the sense of recognition associated with even falsely recalled items, in effect "fooling" a central processor or editor responsible for memorial judgments (*10*). It may be that one of the criteria upon which this sense of recognition is based is the vividness with which the subject is able to envision those items generated as possible memories during recall attempts. If hypnosis enhances the vividness of mental imagery (*11*), perhaps the vividness with which the subject is able to envision these possibilities becomes compelling. Under these circumstances, the editor could mistake vividly imaged possibilities for memories of the stimuli; the enhanced vividness could lead to a false sense of recognition and hence the inflated output as well as the surprising certainty that subjects have about their hypnotically enhanced recall (*12*).

The role that affect may play in the relationship between hypnosis and memory has not been explored in this investigation, but may be relevant to the use of hypnosis in forensic settings. Nonetheless, our observations of hypnotically enhanced recall should give pause to those advocating the use of hypnosis in situations in which the veridicality of information is of prime concern.

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9. All subjects were hypnotized, with those unresponsive to hypnotic suggestion serving as controls. The susceptible subjects reported significantly more information in response to hypnotic suggestion than the unsusceptible subjects [$t(27) = 2.68$, $P < 0.01$]. However, only 10 percent of the new information reported by either group was accurate. While hypnosis produced a small but significant increase in new information [$t(27) = 1.90$, $P < 0.05$], it also produced a significant increase in new errors [$t(27) = 2.67$, $P < 0.01$]. Since this replication examined specific hypotheses, one-tailed tests were used.
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11. Experimental evidence on this point is controversial since most studies base their results solely on self-rating scales. H. J. Crawford, using self-rating scales and an objective performance criterion, reported a shift in the vividness of visual imagery during hypnosis for those in the upper range of hypnotic ability [paper presented at the National Meeting of the Society of Clinical and Experimental Hypnosis, Denver, Colo., October 1979].
12. Pseudomemories, originally developed in hypnosis, may come to be accepted by the subject as his actual recall of the original events; they are then remembered with great subjective certainty and reported with conviction [M. T. Orne, *Int. J. Clin. Exp. Hypn.* **27**, 331 (1979)].
13. We thank S. Vandermeulen and D. Chansonneuve for assistance in collecting and scoring the data; S. Segalowitz, J.-R. Laurence, E. Woody, and M. P. Bryden for their advice and editorial comments; and all members of the University of Waterloo Hypnosis Research Team for their suggestions and encouragement. Supported in part by Social Sciences and Humanities Research Council grant 037-8195.

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Transported Proteins in the Regenerating Optic Nerve: Regulation by Interactions with the Optic Tectum

Abstract. *The transport of specific proteins in regenerating optic fibers of goldfish depends on the presence or absence of the optic tectum. When optic fibers were allowed to contact the tectum, amounts of rapidly transported proteins having molecular weights between 120,000 and 160,000 increased, and a species of molecular weight 26,000 reverted to normal levels. When nerves were prevented from contacting the tectum, the amount of the 26,000–molecular weight protein remained high for months. Amounts of other transported proteins, in particular a group of acidic components of molecular weight 44,000 to 49,000 that increase greatly at early stages of regeneration, proved to be independent of the tectum.*

In neurons, many constituents of the nerve terminal membranes are synthesized in the cell body and conveyed to the terminals in the rapid phase of axonal transport (*1, 2*). Thus, during the development or regeneration of neural connections, changes in the complement of rapidly transported proteins might be expected, in response to the cell's shifting requirements for components involved in such processes as axon elongation, target recognition, and synaptogenesis. Growth-related changes in rapid axonal transport have now been demon-

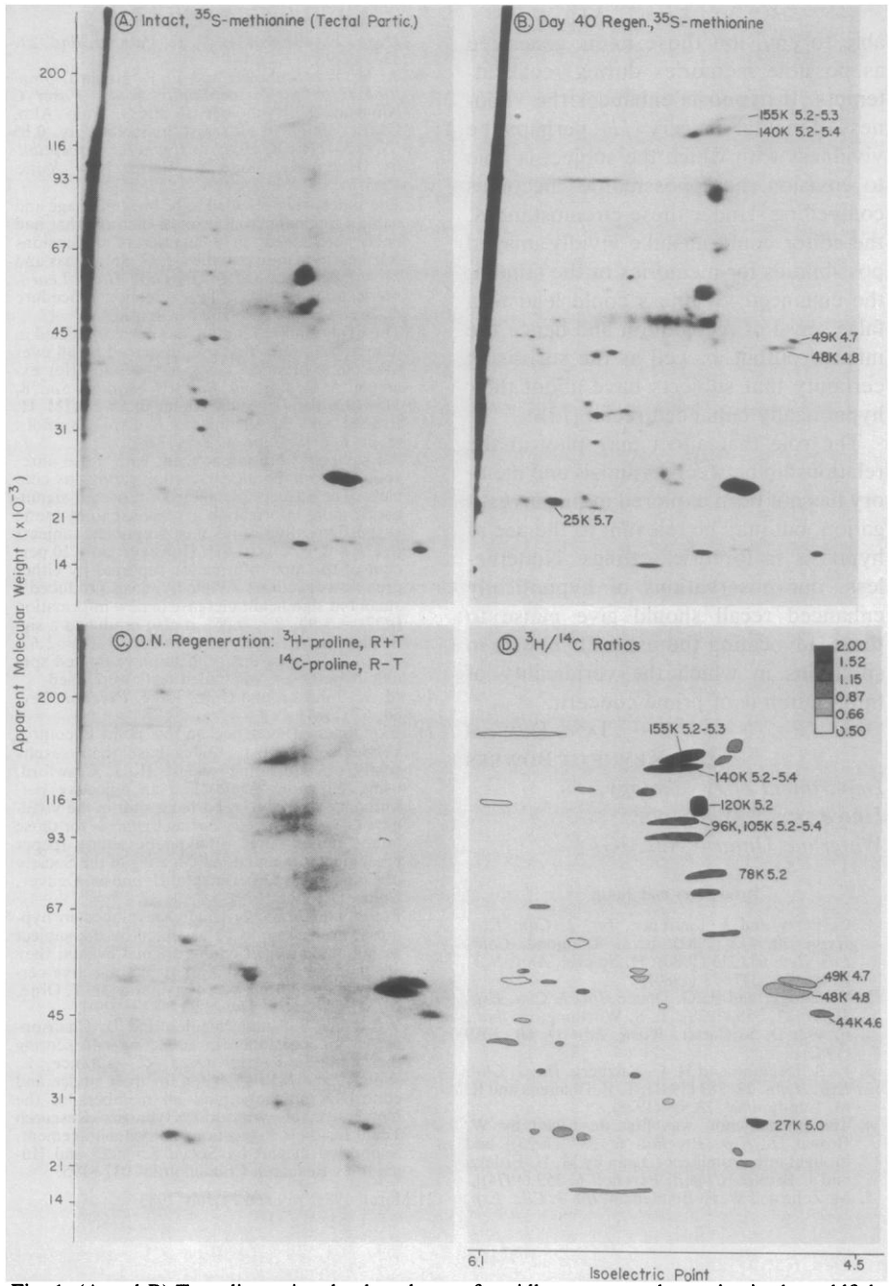

Fig. 1. (A and B) Two-dimensional gel analyses of rapidly transported proteins in the goldfish (A) intact and (B) regenerating visual pathways. Optic nerves were crushed unilaterally in seven goldfish (Comet; length, 10 cm); 40 days later both eyes were labeled with 100 μCi of [^{35}S]methionine (NEG-009T, 1000 Ci/mmole, New England Nuclear) in 4 μl of 0.01*M* phosphate-buffered saline (PBS). Optic tecta were dissected out after 16 hours, homogenized in 0.30*M* sucrose, 50 m*M* tris-HCl, *p*H 7.4, 3 m*M* dithiothreitol, and 3 m*M* unlabeled methionine, and centrifuged at 17,000*g* (20 minutes; 0° to 4°C). Particulate fractions were solubilized in lysis buffer, and the proteins were separated first on isoelectric focusing gels, then on 5 to 15 percent linear gradient sodium dodecyl sulfate (SDS)–polyacrylamide slab gels (*8*). Gels were fluorographed (*9*) and exposed to preflashed Kodak XAR-5 film. Proteins showing marked increases in the regenerating example relative to the normal control are labeled in (B). (C) Fluorogram of two-dimensional gel in which rapidly transported proteins from nerves regenerating with tecta present (R+T; 12 fish) and with tecta absent (R−T; 12 fish) were differentially labeled and co-separated on two-dimensional gels. Intraocular labeling was done 35 days after surgery with 100 μCi of [^{3}H]proline (NET-285, 5 Ci/mmole; New England Nuclear) in the R+T group and 8 μCi of [^{14}C]proline (NEC-285, 290 mCi/mmole; New England Nuclear) in the R−T group (all injections were in 4 μl of PBS and were equimolar in proline). Optic nerves were dissected out 5 hours later, combined from all 24 animals, homogenized, and fractionated as described above, and the proteins co-separated on two-dimensional gels. (D) Isotope ratios for proteins in (C). A template prepared from the fluorogram was used to cut labeled spots out of the gel; ^{3}H and ^{14}C activity was then determined by means of a liquid scintillation system (*5*). The ratios of ^{3}H to ^{14}C for proteins were normalized by the overall ratio of the two isotopes measured in the total sample. Normalized ratios (see key, upper right) reflect the extent to which particular molecular species are selectively increased or decreased in the R+T group relative to the R−T group. Errors in isotope ratio are ≤ 15 percent in all cases (points with low counts, and therefore greater counting errors, are not presented).

strated in the regenerating visual pathway of lower vertebrates (*3–6*). In the goldfish, rapidly transported components having molecular weights of 24,000 to 27,000 (24K to 27K), 44K to 49K, and 210K are selectively turned on between days 8 and 21 of regeneration (*5*). By 1 month—at the beginning of tectal reinnervation (*7*)—the amounts of these proteins begin to decline, and the greatest relative increases occur in proteins having molecular weights between 110K and 140K (*5*). We examined whether the transition occurring at about 1 month is due to a regulatory effect of the principal target tissue, the optic tectum, on the retinal ganglion cells' program of protein metabolism and transport. Our results indicate that interactions between the optic fibers and the tectum alter the pattern of rapidly transported proteins in the retinotectal pathway.

Rapidly transported proteins in the goldfish regenerating visual pathway were compared with those of the intact optic pathway in two-dimensional gel fluorograms (*8, 9*) (Fig. 1). In agreement with our previous one- and two-dimensional gel analyses (*5, 6*), the retinotectal pathway regenerating for 40 days after being crushed had increased transport of proteins migrating with apparent molecular weights and isoelectric points (*p*I) of 155K, 5.2 to 5.3; 140K, 5.2 to 5.4; 49K, 4.7; 48K, 4.8; and 25K, 5.7.

In order to study whether this change in protein transport is regulated by the target tissue, we compared the rapidly transported proteins in optic nerves regenerating in fish having both tecta present (regeneration with tecta, R+T) with those in nerves regenerating in fish having both tecta removed at the time of the optic nerve crush (regeneration without tecta, R−T). At each time point, retinal proteins synthesized in R+T and R−T groups were differentially labeled by intraocular injections of [^{3}H]- and [^{14}C]-proline. Five hours later, at which time most labeling in the optic nerve is associated with rapidly transported components (*1, 2, 5*), nerves from R+T and R−T groups were combined and homogenized. Proteins in the particulate fraction were separated by one- or two-dimensional gel electrophoresis (*5, 6, 8, 9*). As described earlier (*5*), the normalized ratio of the two isotopes incorporated into particular proteins reflects the extent to which those proteins are selectively turned on or off in each experimental case.

The results of two-dimensional gel analysis show that the labeling pattern of rapidly transported proteins in regenerating optic fibers is influenced by the

optic tectum (Fig. 1, C and D). In this experiment, in which fish whose optic nerves had been regenerating for 35 days were used, ^{3}H-labeled proteins from the R+T group and ^{14}C-labeled proteins from the R−T group were separated simultaneously on two-dimensional gels. The labeling of proteins that migrate at 140K, 5.2 to 5.3, and 155K, 5.2 to 5.4, increased markedly when the tecta were present (Fig. 1D). Increases in the labeling of minor proteins in the R+T group in comparison with the R−T group are also shown (Fig. 1D).

Our results also demonstrate, however, that the metabolism of major rapidly transported proteins other than the 140K to 155K species is not influenced by interactions between the ingrowing optic fibers and the tectum. The heavily labeled group of acidic proteins between 44K and 49K is an example of constituents that are labeled to a similar degree in R+T and R−T groups. This group of proteins was shown in previous work (*6*) to increase during early stages of regeneration by a factor of 100 or more in comparison with normal intact optic nerves.

A replication of this experiment with isotopes reversed, that is, with a ^{14}C-labeled R+T group and a ^{3}H-labeled R−T group 36 days after surgery, again showed that rapidly transported proteins at 140K to 155K, 5.2 to 5.4, increased in the R+T group relative to the R−T group, and there was no difference in the labeling of the 44K to 49K acidic proteins. A two-dimensional gel analysis of day 35 material separated in a more basic *p*H range (5.7 to 7.7) showed a number of proteins that did not enter our acidic two-dimensional gel system, among which was a heavily labeled group of proteins between 160K and 230K, $pI >$ 7, that was labeled more heavily in the R−T group than in the R+T group.

The temporal sequence of protein changes associated with tectal interactions was studied at time points ranging from 10 days to 5 months after surgery by double-labeling methods and one-dimensional gel analysis. At the earliest time points examined (days 10 and 15 after surgery), nerves regenerating with and without the tecta present showed only minimal differences in their patterns of rapidly transported proteins (Fig. 2). Beginning on day 24, a prominent labeling increase between 110K and 160K became apparent in the R+T group and reached a maximum of 1.8 times the baseline isotope ratio on day 30 (dark arrow in Fig. 2). This difference declined at later time points and was absent on day 54 and thereafter. In contrast, a protein at 26K was labeled considerably more in the group in which optic fibers had been prevented from contacting the optic tectum (open arrow in Fig. 2); this difference became apparent on day 30 and persisted up to 5 months.

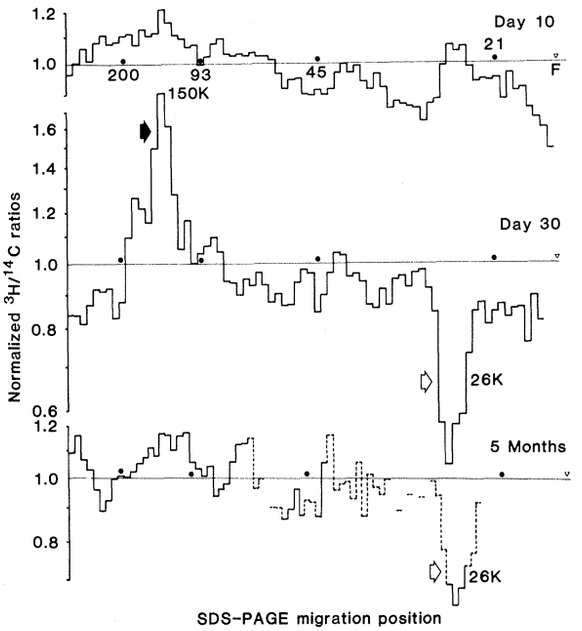

Fig. 2. Time course of tectal regulation of rapidly transported proteins in the regenerating optic nerve. Proteins in nerves regenerating with the tectum present, R+T, and absent, R−T, were differentially labeled with [^{3}H]- and [^{14}C]proline (same concentrations as above; between three and seven fish in each group). Five hours later nerves were dissected out and homogenized, and the proteins from R+T and R−T nerves were separated on 10 percent SDS–polyacrylamide gels (length, 100 mm). Gels were cut at 1-mm intervals and ^{3}H and ^{14}C activities were determined. Normalized isotope ratios reflect the extent to which the metabolism of the proteins is increased (ratio > 1) or decreased (ratio < 1) when regenerating optic nerves contact the contralateral tectum. Statistical errors: 10 to 15 percent for ratios indicated by solid lines and 15 to 20 percent for ratios indicated by dotted lines. Breaks in the ratio profiles are points of low counts on the gel (errors > 20 percent).

Thus, it is possible to distinguish three types of changes in protein metabolism and transport that occur during the reestablishment of neural connections.

1) Changes that are initiated by axotomy but that do not depend on the presence of the target. The most prominent example of this type is the group of acidic 44K to 49K proteins, which are greatly increased at early stages of regeneration and then subside (*5, 6*) whether the tectum is present or not.

2) Changes that are initiated by axotomy and then terminated by interaction with the target tissue. An example of this type is the 26K protein that increases at early stages of regeneration (*5*) but declines to normal levels when contact is made with the tectum (*10*). In nerves prevented from contacting the tectum, high levels of this protein persist for months.

3) Changes that are first initiated by contact with the target. The increased labeling of the 140K to 155K proteins that appears between 3 and 6 weeks when nerves are allowed to contact the tectum is representative of this type of change (*11*).

The nature of the signals that regulate these shifts in protein metabolism and transport is unknown, as is the physiological significance of the various molecular components identified here. These protein species merit further study for understanding the development of neural circuitry at a molecular level.

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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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29 October 1981

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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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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