Recovery of function after brain damage: A theory of the behavioral deficit

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The present discussion assumes that recovery of function is not an event but rather a process. When it occurs, this process bridges the gap between the occurrence of the brain injury and the reinstatement of a particular behavior disrupted by the brain injury. Accordingly, to understand the process of recovery of function, one must first appreciate the point from which the process begins, that is, the behavioral deficit. It is the thesis of the present discussion that one effect of neural injury is to cause the individual to shift behavioral control to neural systems not directly affected by the neurological insult. The behavioral deficit is then the result of the individual's attempting to learn new behaviors to compensate for those normally mediated by the damaged neural system. Recovery of function, on the other hand, represents a return to the neural system directly affected by the injury and the utilization of whatever of this system may be spared.

If certain animals (e.g., rats) are trained on a two-choice brightness discrimination and then subjected to visual neocortical ablation, the preoperatively acquired behavior is completely disrupted (Horel, Bettinger, Royce, & Meyer, 1966; Lashley, 1935; LeVere & Davis, 1977; LeVere & Morlock, 1973, 1974). However, if the lesioned animals are given postoperative retraining on the discrimination, then the brightness habit may be reinstated and in approximately the same number of training trials required to initially learn the behavior. This result is but one of the many examples of a general phenomenon in neuropsychology—namely, the initial effects of brain injury are more severe than the subsequent long-term effects. The phenomenon has been summarily labeled “recovery of function” and its widespread acceptance can be attributed to the range of postlesion behaviors with which it is typically, if sometimes inappropriately, associated. Yet, even if recovery of function is more correctly restricted to just the reinstatement of only those particular behaviors disrupted by the neural lesion, there still remain enough instances of the phenomenon to confirm its validity.

Because recovery of function is so robust, because it is often taken to demonstrate unique characteristics of neural tissue (e.g., vicariation), and because it is a classic result, it has generated considerable debate and theory (see texts edited by Finger, 1978; Stein,

Rosen, & Butters, 1974). Unfortunately, the research generated by this debate has done little more than reaffirm the phenomenon, and the theory has produced correspondingly little explanation. The thesis of the argument I will present here is that the reason for this debate is that recovery of function research and theory have, by and large, attempted to investigate and explain something that has not occurred, i.e., the recovery of neurological and behavioral losses following brain injury.

To begin to develop this thesis, consider again the example of the disruption and subsequent recovery of a preoperatively learned brightness discrimination in rat. Typically, the behavioral deficit is considered to be a loss because it is assumed that visual behavior is principally mediated by neocortical neural systems. While the evidence is extremely slim (for a full discussion, see Meyer & Meyer, 1979), this usually reduces to a belief that, initially, the memory engrams or neural traces of the preoperatively learned discrimination are stored in visual neocortex. Following the visual lesion, these memory engrams or neural traces are necessarily destroyed, and the lesioned animal thus exhibits a postoperative behavioral loss because there is no memory of what is required and/or how to perform what is required.

However, since recovery of function is possible, the assumption that the behavioral deficit is a loss necessarily requires yet another assumption. This is that the nervous system possesses sufficient plasticity to allow remaining neural centers to recognize and carry on the functions of the neural centers destroyed by the brain lesion. Accordingly, with vicariously functioning neural systems, the brain-injured animal is able to relearn, that is, recover, the lost brightness discrimination. In general terms then, this explana-

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tion supposes that recovery of function is essentially similar to the way in which the behavior was initially established, with the single, important exception that different reorganized and vicariously functioning neural centers are involved.

On the surface, there would appear to be a number of reasons to accept the assumption that the behavioral deficit following brain injury represents a loss and that the recovery of this loss depends upon neural reorganization. Not the least of these is that post-trauma behavioral evaluation typically indicates not even the slightest vestige of the disrupted behavior. Moreover, and more importantly, there is expanding evidence that the central nervous system possesses a number of very impressive dynamic properties (LeVere, 1975). Among these are neural regeneration (see NANCDS, 1975), the electrophysiological consequences of early sensory manipulations (Layton, Corrick, & Toga, 1978; Walsh & Cummins, 1976), and compensatory and collateral sprouting (Cotman, 1978; Kerr, 1975), all of which demonstrate that even the adult mammalian central nervous system is anything but stagnant.

The problem is, however, that demonstrating that the central nervous system possesses certain dynamic properties does not also demonstrate that the central nervous system is capable of reorganization. In this regard, neural regeneration principally reestablishes pretrauma synaptic contacts (NANCDS, 1975), the electrophysiological effects of early sensory manipulations are quite permanent and do not occur when similar sensory manipulations are performed in the adult animal (Layton et al., 1978; Walsh & Cummins, 1976), and the sprouting that occurs is typically between neural systems that normally have some organizational affinity (Cotman, 1978; Kerr, 1975). These and similar considerations led LeVere (1975) to suggest that the morphological changes following CNS injury might be more akin to peripheral regeneration and, as such, represent reinnervation and not reorganization. Or, from another view, if the nervous system is "plastic" enough to accommodate something more than reinnervation, then it is difficult to understand why the frog with rotated eyes is never able to capture its own prey.

Thus, it would seem that a fundamental postulate necessary to account for the recovery of behavioral losses, that is, neural reorganization, is equivocal at best. But perhaps the behavioral deficit following a neural insult need not always represent a loss. And, if the behavioral deficit is not a loss, then there is no need to postulate unique and tenuous biological mechanisms to account for recovery of function. It is the purpose of the present discussion to take exactly this position and explore an alternative explanation of the effects of brain injury and the recovery from these effects. To do this, we will do three things. First, we will outline certain recent evidence that reaffirms

the proposition that recovery of function is incompatible with neurological and behavioral losses. Second, we will offer an explanation of the behavioral deficit, given that the neural mechanisms and, necessarily, the disputed behavior are not lost. And, finally, we will provide evidence to support this explanation of the nature of the behavioral deficit and recovery of function.

A Fundamental Definition

Before addressing these topics, it is first necessary to precisely define recovery of function. This is necessary because recovery of function is often associated with a variety of postoperative behaviors, particularly compensation, which is definitionally incorrect (Riese, 1944). Recovery of function is defined here as the postlesion reinstatement of the specific behaviors that were disrupted by the brain injury. Compensation, on the other hand, is a substitutive process in which nothing is recovered but a new and sometimes grossly different behavior is acquired to attenuate the behavioral deficiencies produced by the brain injury.

As an extreme clinical example, consider the aphasic who is taught to use a typewriter to compensate for an inability to form coherent vocal patterns. Clearly, through this compensatory learning, the individual may be able to communicate and enjoy a certain control of interactive language functioning. But, just as clearly, the individual has not recovered from the effects of the neurological insult that disrupted the speech patterns in the first place. Moreover, and critically more important, I will argue shortly that it is this very process of compensation which actually underlies the behavioral deficit because it interferes with the postlesion expression of spared neural mechanisms.

Losses, Disruptions, and Recovery of Function

With this definition in mind, we may turn to the question of whether recovery of function is compatible with neurological and behavioral losses. Basically, our position is that there are two polar effects of brain injury. On the one hand, there are losses that are permanent. On the other hand, there are disruptions that are not necessarily permanent and may be recovered. For example, there is a general performance deficit associated with striate ablations in rats (Cooper, Freeman, & Pinel, 1967; Lashley, 1930; LeVere & Mills, 1977) that is permanent and persists even though postoperative training will normalize the absolute (Cooper et al., 1967) and relative (LeVere & Mills, 1977) brightness thresholds. Or, as another example, consider the destriate rat's ability to recover both a preoperatively learned brightness discrimination and a horizontal-vertical line discrimination. However, notwithstanding this rather impressive postlesion visual capability, the striate

decorticate rat is nonetheless unable to discriminate oblique line patterns (Lavond, Hata, Gray, Geckler, Meyer, & Meyer, 1978). Apparently, there is a loss of form vision but only a disruption of the ability to detect differences in brightness, even the very localized differences in brightness needed to discriminate horizontal and vertical lines (Lavond et al., 1978).

In somewhat more detail, a recent series of experiments from our laboratory directly addressed the question of whether the disruption of a brightness discrimination by visual neocortical lesions represented a loss of function. Our research was predicated on the fact that a behavior, particularly a learned behavior, is not a unitary thing. Rather, a behavior is a constellation of processes, all of which must occur for the overt expression of the behavior. Thus, even though there may be considerable sparing, it is still possible that a behavioral deficit might be precipitated by the loss of some individual component of the behavior in question.

For example, the disruption of a preoperatively learned brightness discrimination following visual decortication might reflect the destruction of, the loss of, the memory engrams or memory traces of the preoperatively learned discrimination. Or, the memory traces might be spared and the behavioral deficit might reflect the loss of the neural mechanisms associated with the motivation to perform the discrimination. Or, both the memory and the motivational mechanisms might be spared and the behavioral deficit reflect a loss of the ability to utilize that which is spared. If any of these should occur, then the recovery that is observed might easily depend upon reorganization and vicarious functioning of certain individual neural centers or systems. And this would be independent of the fact that other components of the learned behavior were spared.

To investigate this possibility, we first tested whether the postoperative behavioral deficit might be due to a loss of the preoperatively acquired memory traces of the brightness discrimination (LeVere & Morlock, 1973, 1974). The procedure was essentially a proactive interference paradigm in which rats were postoperatively tested on the preoperatively learned brightness discrimination or a reversal of the preoperatively learned brightness discrimination. The results showed that those animals retrained on the reversal of the brightness discrimination were significantly impaired relative to those animals retrained on the original brightness discrimination. Definitionally, this sort of proactive interference could only occur if the preoperatively established memory engrams or memory traces were not only spared but were also able to influence postoperative behavior. Thus, the disruption of a preoperatively learned brightness discrimination by striate ablation must not

be a function of the loss of preoperatively established memory engrams.

Turning to the question of whether the behavioral deficit might represent the loss of the motivation to perform, LeVere, Davis, and Gonder (1979) trained three groups of normal rats on the same brightness discrimination but under different levels of footshock motivation. Subsequent to visual decortication, these animals were tested on a 10-trial extinction procedure using the same preoperative training paradigm but without footshock. The obvious purpose of this extinction test was to determine whether or not there was a loss of the motivation to run in the Yerkes apparatus following striate lesions. The results clearly indicated that not only would all of the animals run from the startbox and enter the goal area, but also that response latency was directly related to the amount of footshock received during preoperative training. From these data, we conclude that the neural mechanisms associated with the motivation to perform the discrimination, like the memory engrams of the discrimination, are not lost following visual neodecortication.

LeVere et al. (1979) also tested whether the decorticate rat was able to utilize what was learned in one brightness discrimination when trained on a subsequent brightness discrimination. In this experiment, all training was subsequent to visual decortication, and the question of utilization was evaluated by training rats on two similar brightness discriminations but under different motivations. We chose to change motivational state to test utilization because our previous research (LeVere & Davis, 1977) demonstrated that changing motivation between preoperative learning and postoperative recovery would eliminate the reversal impairment described by LeVere and Morlock (1973, 1974). However, the results of the present experiment clearly showed that the destriate rat is as adept as its normal counterpart at utilizing what is learned in one discrimination during training in another discrimination. And again, we must conclude that the behavioral deficit produced by visual neodecortication is not the loss of some preoperative ability.

In summary, whatever it is that disrupts a preoperatively acquired brightness habit following visual neodecortication, it is not the loss of the memory engram of the habit, because this is spared. Nor is it the loss of the motivation to perform the habit, because this is spared. Nor is it the loss of the ability to utilize what is spared, because this is also spared.

The severe critic may, however, point out that all of the above data are behavioral observations of presumed underlying neurobiological events. And, while these observations are consistent with the suggestion that recovery of function is incompatible with losses,

it is nonetheless possible that overt behavioral assessment is simply not adequate to the detection of a neurological loss and subsequent reorganization. Without implying the validity of this position, it may be mentioned that the present conclusions are, in fact, supportable with the microelectrode.

Among these data, perhaps the most well known are the electrophysiological effects of early sensory deprivation. The initial data were convincingly provided by Hubel, Wiesel, and their associates (Dews & Wiesel, 1970; Hubel & Wiesel, 1965, 1970; Wiesel & Hubel, 1963) and involved suturing closed the lids of one eye of a cat during the first few weeks of life. In adult cats that were allowed normal visual experience during their early development, approximately 80% of the single cells in the visual neocortex were binocularly responsive. However, in adult cats that were allowed only monocular visual experience during their early development, this binocular responsiveness dropped to roughly 7%. And what is important for the present discussion is that this abnormal electrophysiology persists even when the deprived cat is allowed 4 years' normal visual experience, even when the normal eye is sutured closed in adulthood, and even when there is some behavioral adaptation (Layton et al., 1978; Walsh & Cummins, 1976). Clearly, this neurological loss of binocular responsiveness is anything but transitory.

Even more compelling evidence that brain injury may induce permanent neurological losses and that recovery of function is not dependent upon mitigation of these losses is provided by a recent experiment reported by Spear and Baumann (1979). Spear and his colleagues were concerned with the recovery of visual abilities following destruction of visual neocortical areas 17, 18, and 19 in cat. Previous research (Baumann & Spear, 1977; Wood, Spear, & Braun, 1973) demonstrated that this visual recovery was strongly dependent upon the integrity of the lateral suprasylvian area (LS). What interested Spear and Baumann in their most recent experiment, however, was whether or not destruction of primary visual neocortex induced electrophysiological changes in area LS and, more importantly, whether these changes were ameliorated when behavioral recovery of function occurred. In normal cats, the single units that may be recorded from area LS show well-defined visual fields that are directionally sensitive to moving stimuli. Subsequent to primary visual neocortex ablation, there is a marked drop in the number of LS units showing directional sensitivity and a decrease in the proportion of cells that respond to the ipsilateral eye. Thus, there is a clear loss of certain of the electrophysiological properties of the single units in area LS following primary visual lesions. However, what is critical about this loss is that it is unmodified when the cat ultimately recovers the ability to per-

form visual discriminations. Or, from another point of view, the process of recovering the ability to perform visual discriminations is not dependent upon, or the result of, recovering some neurological loss in a neocortical area shown to be critical to the recovery process. To quote Spear and Baumann: "These results suggest that functional reorganization plays little or no role in recovery from visual cortex damage in adult cats. Rather the recovery of form and pattern discrimination ability appears to be based upon the functioning of residual neural processes in the LS area which remain after visual cortex damage."

In conclusion, then, it would seem that brain injury may result in either or both disruptions and losses. And, more importantly, disrupted behaviors are, or may be, temporary and ultimately recoverable, but losses, whether measured behaviorally or neurologically, are just that—losses.

Disruptions, Optimization, and the Nature of the Behavioral Deficit

On the surface, the proposition that recovery of function depends upon what is spared is obviously an uninteresting truism. However, there is a deeper aspect to this proposition. This is that, not only is recovery of function dependent upon morphological sparing, but also, because we postulate that the nervous system is incapable of reorganization and vicariation, recovery of function is dependent upon functional sparing.

The problem is, however, that adopting this posture forces a rather strange paradox. This paradox is that if recovery of function depends upon sparing of those neurological mechanisms controlling the behavior, then why is there a deficit at all? In fact, on purely definitional grounds, it would seem that sparing could not be the mechanism underwriting recovery from behavioral deficits. I do not believe that this is so. Rather, I believe that there is a simple explanation of why there is a behavioral deficit even if the critical neural mechanisms are spared. The nucleus of this explanation is that when a behavioral deficit occurs following brain injury, it occurs because the individual attempts to compensate for the effects of the neural lesion by utilizing alternative unaffected neural systems rather than what is spared of the lesioned neural system.

To explain this, I will first suggest that, above all, individuals are optimizers. By this, I mean that given the opportunity to do so, the individual will behave on the basis of those sensory, cognitive, and motor systems that perform best. Next, I will suggest that brain injury is not without some consequence that will degrade some sensory, cognitive, or motor function. From these two points, it would not seem unreasonable to suppose that the brain-injured individual's first response to brain injury would be

an attempt to avoid any behavior dependent upon the neural centers directly affected by the brain injury and to behave on the basis of those neural centers least affected by the brain injury. And, in turn, this will necessarily produce a behavioral deficit, simply because the neural centers controlling the disrupted behavior are not utilized.

To develop this position further, consider the two possible outcomes of compensation. Compensation may be either successful or unsuccessful; but, in either case, it will be incompatible with recovery of function. On the one hand, if compensation is successful and if the individual is able to obtain goals by compensating for the disrupted behavior, then obviously there is no need for recovery of function to occur. And I submit that recovery of function will not occur. In fact, if the requirement for obtaining the preoperative goals are not strict, there may even be no observable behavioral deficit. Consider in this regard the aphasic who, unable to speak certain words, uses alternative sentence constructions to avoid the unspeakable words but still convey his or her intended meaning (Luria, 1970) or, in the extreme case, the split-brain lesion, the severity of the resultant behavioral dysfunction (Gazzaniga, 1970) of which we are only now beginning to fully appreciate.

On the other hand, the attempt to compensate for the effects of brain injury may be unsuccessful and not lead to goal attainment. In this instance, one of two additional outcomes must occur, depending upon whether the neural mechanisms critical to the disrupted behavior were destroyed. If the critical neural mechanisms were destroyed, then, as previously discussed, there will be a permanent behavioral loss. Alternatively, if the critical neural mechanisms were spared, then, as previously discussed, recovery of function may occur. However, it is important to emphasize that even if there is sparing and even if recovery of function ultimately does occur, there is still an initial behavioral deficit. The central theme of the present theory is that this behavioral deficit is due to the initial attempt of the brain-injured individual to compensate for the neural lesion, and only after this attempt proves fruitless—that is compensation is unsuccessful or some other event again changes what is optimal (subsequent brain injury)—may the injured individual utilize those sensory, cognitive, or motor systems that were directly affected by the neural injury. Moreover, it is only at this time that the spared neural centers and/or spared neural systems can influence postlesion behavior to allow recovery of function to occur.

To bring these abstractions together, we may return to the classic Lashley paradigm previously described. It will be remembered that a preoperatively learned brightness discrimination can be completely disrupted by visual decortication but that the rat can

postoperatively recover the behavior. With regard to this procedure, it is important to emphasize two things. First, the laboratory two-choice brightness discrimination is a highly controlled and restricted learning situation in which the animal can attain reward only by responding to the information provided by the visual discriminative cues. Second, even the normal animal initially expends a number of training trials and commits a number of errors before learning occurs, and there is general agreement that, during these initial trials, the animal is doing something it ought not to be doing—that is, its behavior is not under visual stimulus control. It is not terribly important here whether the rat begins to attend to the visual cues by switching in the appropriate analyzer (Sutherland & Mackintosh, 1971) or by eliminating inappropriate response tendencies (Estes, 1973) or whatever. The important thing is that, initially, the normal animal does not respond to the available visual information and, in operational terms, exhibits a behavioral deficit.

Subsequent to visual decortication, the rat's behavior seems to parallel that which occurred during the initial preoperative training. And one might postulate, as Cooper and his associates have (Bauer & Cooper, 1964; Bland & Cooper, 1970; Goodale & Cooper, 1965), that this is exactly what happens because the neocortical lesion produces a neurological loss and, postoperatively, the animal must relearn the brightness discrimination. Thus, the initial preoperative errors, the original preoperative learning, the postoperative behavioral deficit, and the subsequent recovery of function are all postulated to represent a similar learning process. The problem is, however, that more recent data suggest that the postoperative behavioral deficit does not represent a loss of memory, a loss of the motivation to perform the preoperatively learned behavior, or a loss of the ability to utilize previously learned behaviors (LeVere & Davis, 1977; LeVere et al., 1979; LeVere & Morlock, 1973, 1974). Also, the effect of amphetamine (Braun, Meyer, & Meyer, 1966; Ritchie, Meyer, & Meyer, 1976) and the effect of the RNA antimetabolite 8-azaguanine (Davis & LeVere, 1979) both demonstrate that whatever the recovery process is, it is not a rerun of preoperative acquisition.

However, while we would argue that relearning does not account for recovery of function, the present theory does not deny that attempts to learn influence recovery of function. Nor does the present theory deny that these attempts to learn are precipitated by the occurrence of brain injury. Indeed, we would strongly assert that this is exactly what happens. But there is a very critical difference in what is presently being suggested with respect to this learning. This is that, rather than alleviating the behavioral deficit precipitating recovery of function, attempted new

learning—that is, compensation—will actually cause the behavioral deficit by retroactively interfering with the expression of what is spared. Returning to the Lashley paradigm, removing the rat's visual cortex is certainly not without some deleterious effect on the visual information available to the lesioned animal (see, e.g., Spear, 1979). Since we believe that the rat will attempt to attain reward on the basis of the best available sensory information, it would seem reasonable to suggest that the rat will initially attempt to perform on the basis of that information not directly affected by the striate lesion. But, as previously noted, the laboratory brightness discrimination apparatus is a very restricted situation, so that any attempt to perform on the basis of nonvisual sensory information will fail, and it will fail independent of the inherent quality of this other nonvisual information. And, similar to the animal's attempts to respond on the basis of nonvisual information during the initial preoperative training, this will produce a behavioral deficit. Thus, the major difference, the only real difference, between the initial trials of preoperative acquisition and the postoperative behavioral deficit is that, in the latter case, the incorrect responding is precipitated by an attempt on the part of the animal to compensate for the effect of the visual lesion. And, it is only after this attempt to compensate proves inadequate that the rat will return to utilize what is spared of the visual system. Furthermore, it is only at this time that whatever visual mechanisms that were spared by the lesion can affect the animal's postoperative behavior and allow recovery of function.

In summary, this theory then suggests that compensation is the antithesis of recovery of function and one of the primary factors responsible for the postoperative behavioral deficit. Accordingly, to maximize recovery of function—that is, recovery of those specific preoperative behaviors disrupted by a particular brain injury—one must minimize compensation. While this notion that learning interferes with recovery of function may seem somewhat heretical, it is not necessary to accept it, or reject it, on faith. Rather, the theory does enjoy some considerable empirical support, as we will next attempt to demonstrate.

Some Data

There are two cornerstones to this theory of the nature of the behavioral deficit and its alleviation following brain injury. The first is that a major effect of neurological insult is to induce a shift in behavioral control to those neural systems not directly affected by the injury. Second, this shift of behavioral control necessitates that the individual compensate by attempting to learn new methods to attain the goals previously attained by behaviors mediated

through the damaged, but now unutilized, neural system. What follows is a summary of the evidence, which we interpret as indicating that: (1) lesions will cause shifts in the neural systems that control behavior, and (2) postlesion learning, that is, attempts to compensate, will interfere with recovery of the spared functions of an injured neural system.

Brain injury, control shifts, and compensation. As a starting point, consider the effects of bilateral and unilateral spinal deafferentation as reported by Ommaya and Bossom (summarized by Guth, 1974). Following bilateral deafferentation, monkeys are able to recover use of their extremities in approximately 3 to 4 weeks. However, following unilateral deafferentation, the monkeys never use their ipsilateral limbs in any useful fashion. That is, they never do so unless the unaffected contralateral limb is physically restrained, and then the animals are able to recover use of the ipsilateral limb in roughly 3 to 4 weeks. We suggest that there are two reasons that this is rather clear evidence that a neural injury will cause an avoidance of neural systems affected by the injury and a shift in behavioral control. First, there is the obvious empirical observation that the unilateral deafferentated animals never use the limb ipsilateral to the lesion unless physically forced to do so. Second, and more important, recovery of the ipsilateral limb following physical restraint requires 3 to 4 weeks or just about the same amount of time required to recover from bilateral deafferentation. The fact that there is no facilitation of recovery would seem to be rather salient evidence that whatever the monkey is doing during the time it is allowed to use the unaffected contralateral limb, this does not involve the ipsilateral limb. If it did, there should have been some facilitation of the recovery process when the animal was finally forced to use the ipsilateral limb. In other words, when allowed to do so, the neurologically injured animal will attempt to optimize its behavior by avoiding affected neural systems and compensate with behaviors involving unaffected neural systems.

A very similar result was reported at the neocortical level by Lashley (1924) when he studied the effects of precentral gyrus lesions on the performance of learned motor behaviors. In this now-classic experiment, Lashley describes the behavior of several monkeys that were trained prior to bilateral ablation of the precentral gyrus. However, what is of interest here is Lashley's report of a small male *Cebus* monkey that was too wild for preoperative training. This animal received a unilateral lesion of the right motor area before training was begun. Following this lesion, the left leg and arm were not used for about 4 weeks and then the left leg appeared to return to normal and the left arm could support the animal's weight. Eleven weeks after the right unilateral opera-

tion, the paralysis had almost disappeared, but Lashley reported that the left arm was never used unless the right arm was restrained. This is, of course, similar to the effects of spinal deafferentation. Training on a series of puzzle boxes was begun at this time, and during the next 3 months the animal performed quite well, but always using the right arm. Seven months after the first operation, the left motor area was destroyed, which, of course, produced a severe paralysis of the previously used right arm and leg. According to Lashley, this paralysis was as complete as that which occurred on the left side following the first unilateral lesion. After a short recovery period following this second lesion, the animal was given a series of retention tests, and Lashley's description is most succinct: "After the second operation, the right hand was much more affected than the left, which had largely recovered and an almost immediate shift to the left hand in opening all of the puzzle boxes occurred."

Again, as with the effects of spinal cord deafferentation, these results would seem to clearly suggest that, following neurological injury, an animal will utilize the best of what is at its disposal. Moreover, in the data reported by Lashley, it is noteworthy to emphasize that the shift to the less damaged extremity was almost immediate and that the animal was quite able to open the puzzle boxes with its left hand and arm. This in turn would indicate that the destruction of the left motor area spared what was learned with the right limb and that the animal simply utilized the best neural mechanisms available to express that which was spared.

Further evidence concerning optimization and control shifts comes from the effects of unilateral visual neocortical lesions. The typical result of a unilateral striate ablation is a disruption of visual orientation to stimuli in the contralateral visual field (Cooper, Bland, Gillespie, & Whitaker, 1970; Kirvel, Greenfield, & Meyer, 1974; Sprague, 1966). While not quite as salient as this contralateral hemianopia, it is also often reported that the unilateral visual decorticate will exhibit ipsiversive circling. Kirvel et al. attribute this ipsiversive circling to the animal's responding to stimuli in the unaffected visual field. We, of course, concur with Kirvel's interpretation and would suggest that it is a very nice example of the animal's responding to the sensory input that is least affected by the neurological injury. However, both the unilateral hemianopia and the ipsiversive circling may be corrected if the contralateral superior colliculus is ablated. Unfortunately, the superior colliculus lesion is not without its own consequences, and these are to induce a contralateral multimodal sensory deficit, that is, a multimodal sensory deficit on the side opposite to the hemianopia produced by the original unilateral visual lesion (Kirvel et al., 1974). In other

words, what was previously "just" a unilateral visual hemianopia is escalated by the subsequent superior colliculus lesion to a multimodal deficit on the opposite side. From the present notions of an individual as an optimizer, and the effects of neurological lesions, we would suggest that the return of visual orienting is quite understandable because the subsequent superior colliculus lesion changed what was optimal and caused a shift in behavioral control. That is, the return of visual orienting after the superior colliculus lesion is the behavioral expression of an attempt on the part of the animal to base its behavior on the best available sensory information, which, after the superior colliculus lesion, is what was spared by the initial unilateral visual decortication.

Before leaving this particular set of experiments, it is necessary to make one additional point. This is that there have been a number of rather elegant physiological explanations of why the initial visual hemianopia is mitigated by a subsequent contralateral superior colliculus lesion. Not the least of these is that offered by Sprague (1966) to explain his early demonstration of the phenomenon. Moreover, electrophysiological evidence (Goodale, 1973) has provided nontrivial support for the postulation that cortical facilitation and collicular inhibition must remain in balance for appropriate visual orientation. The elegance and worth of these neurophysiological theories are in no way disputed by our present suggestion of optimization following brain injury. Rather, we emphatically emphasize that our position is simply concerned with the behavioral consequences of whatever neurophysiological mechanisms will account for the combined effects of unilateral decortication and subsequent contralateral superior colliculus ablation.

However, notwithstanding our emphasis on the behavioral consequences of brain injury, these consequences are nonetheless often lost and/or disregarded in the rush to elucidate the underlying neurophysiology. Because of this, we will describe one final experiment to support our arguments of optimization and control shifts. This final experiment concerns how neocortical lesions will affect overshadowing and has been reported by Nonneman and Warren (1977). We choose this experiment not only because it supports our particular position, but also because, to our knowledge, the technology of the physiology laboratory has yet to overshadow the behavioral data.

Overshadowing, as originally used by Pavlov (1927), refers to the increased conditioning that will accrue to the more salient component of a compound conditioned stimulus. Nonneman and Warren (1977) were interested in determining whether auditory neocortical lesions in cats would modify overshadowing if the visual and the auditory components of a compound conditioned stimulus were individually

equated for saliency. Probe test trials during conditioning with a compound conditioned stimulus indicated that both the normal and the lesioned cat would respond about equally to either the visual or the auditory component if it was presented alone. However, and this is the critical result, when there was a choice between the individual components, the normal and the brain-damaged cat responded quite differently. The normal cat predominantly responded to the auditory stimulus as normal cats usually do, while the auditory lesioned cat predominantly responded to the visual stimulus. We interpret these data as support for our proposition that, when allowed to do so, individuals will guide their behavior on the basis of those neural systems least affected by brain injury, independent of the preoperative disposition of the individual.

In summary, then, it would seem that because the individual will attempt to optimize, one of the effects of brain injury is to induce a shift away from those neural systems that sustained the injury. In fact, to our knowledge, the evidence is quite compelling, and this sort of shift is a dominant result of any experiment that allowed its direct evaluation. In those experiments that did not directly test for a shift in behavioral control, the shift was simply characterized as a behavioral deficit.

Control shifts, compensation, and recovery of function. Turning to the consequences of a shift in behavioral control to neural systems not directly affected by the brain injury, there are but two. First, the animal will not engage in any behavior mediated by the injured neural system, even if what is spared of the injured neural system could accommodate the behavior. Second, the injured individual will necessarily have to compensate, through new learning, for those behaviors that were mediated by the now-avoided neural system. If the compensation is successful, there will be a transient behavioral deficit while this new learning occurs. If the compensation is unsuccessful, there will be either a behavioral loss or a transient behavioral deficit while the individual unsuccessfully attempts to learn alternate, but ineffective, behaviors. Accordingly, it would seem that to maximize the possibility of recovery of function, that is, the recovery of what is spared of the neural system directly affected by injury, one should prevent attempted new learning. We now turn to evidence that we believe supports this proposition.

To begin, we may consider the facilitatory effects of amphetamine. Early research by Meyer, Horel, and Meyer (1963) indicated that the administration of amphetamine would reinstate visual placing in decorticate cats. These results were then extended by Braun et al. (1966) in an investigation involving the postoperative recovery of a preoperatively learned two-choice brightness discrimination. The procedure

was essentially a replication of Lashley's original paradigm but with the exception that some of the decorticate rats were postoperatively injected with amphetamine. The results showed that those animals injected with amphetamine were able to recover the preoperatively learned brightness discrimination significantly faster than the animals injected with physiological saline. By itself, this result may not be terribly surprising, since amphetamine could simply facilitate performance. However, Braun et al. (1966) also demonstrated that the drug had no effect on original learning in visual decorticate rats. This, then, meant that the postoperative facilitatory effects of amphetamine depended upon the existence of an established behavior. On the basis of this, Braun et al. concluded that amphetamine affected recovery of function by facilitating the animal's ability to access memory engrams that were spared by the visual decortication. The arguments that we have presented here are most complementary to this conclusion. Our only contribution is to suggest that the reason the spared memory engrams were more easily accessed was because amphetamine may have prevented the retroactive interference of compensatory learning.

At very high doses, amphetamine is clearly a CNS excitant and may interfere with new learning, but these were not the dosages used by Meyer and his colleagues. However, amphetamine may also interfere with new learning at much lower doses. For example, Tecce and Cole (1974) have shown that even low-level amphetamine will depress CNS activity, produce drowsiness, and lower electrical brain activity (particularly the contingent of negative variation), all of which are incompatible with effective acquisition. Moreover, Nasello and Izquierdo (1969) have demonstrated that hippocampal RNA activity is significantly depressed with repeated administrations of amphetamine. While the drug amphetamine may have numerous effects, the reduction of hippocampal RNA activity is reminiscent of the learning impairments produced by the RNA antimetabolite 8-azaguanine (Dingman & Sporn, 1961; LeVere & Fontaine, 1978). Thus, it would seem that amphetamine may produce conditions that are less than optimal for new learning. And, according to the presently proposed theory, the disruption of new learning will facilitate recovery of function because attempts to compensate will not occur and thus will not retroactively interfere with the expression of spared neural mechanisms.

Unfortunately, how amphetamine affects behavior is debated as rigorously as the notion that recovery of function may or may not depend upon neural sparing. Because of this, the relevance of the amphetamine data to the present notion that the behavioral deficit is an attempt to compensate may be disputed. However, the amphetamine research is not the only

demonstration that the brain-injured animal's attempt to compensate may interfere with recovery of function. In this regard, we turn to a series of experiments utilizing the drug 8-azaguanine.

As previously noted, 8-azaguanine is an RNA antimetabolite that will interfere with both maze learning (Dingman & Sporn, 1961) and two-choice brightness discrimination learning (LeVere & Fontaine, 1978), but will not affect the performance of these tasks once they have been learned. If the behavioral deficit following neocortical injury is a function of the individual's shifting behavioral control to unaffected neural systems, and if this control shift necessitates an attempt to learn to compensate, then 8-azaguanine should facilitate recovery of function because it will interfere with learning and will force the individual to utilize what is spared to control behavior. Davis and LeVere (1979) tested this prediction in a study involving three experiments using normal and visual decorticate rats in a standard two-choice Yerkes brightness discrimination. In these experiments, lesioned animals were administered 8-azaguanine or physiological saline and required to initially learn a two-choice brightness discrimination, recover a preoperatively learned two-choice brightness discrimination, or relearn a preoperatively acquired two-choice brightness discrimination. The results may be summarized quite simply: 8-azaguanine *interferes* with original postoperative learning and postoperative relearning, but *facilitates* postoperative recovery of a two-choice brightness discrimination. Since these data provide rather fundamental support for our present explanation of the consequences of brain injury and the occurrence of recovery of function, some detail is in order.

The first experiment of this study was a systematic replication of LeVere and Fontaine (1978), using visual decorticate rats. Since the drug was to be used to investigate recovery of function following neocortical ablation, we were obligated to perform this replication to determine whether or not the effects of 8-azaguanine were cortically dependent. Procedurally, this experiment was quite simple and tested the postoperative learning and retention of a brightness discrimination when visual decorticate rats were injected with 6 mg of 8-azaguanine or an equal volume of physiological saline. The results exactly replicated those reported by LeVere and Fontaine in that the drug significantly interfered with the acquisition of the two-choice brightness discrimination, but had absolutely no effect on the performance of the discrimination. Thus, we concluded that the effect of 8-azaguanine was not cortically dependent and, more importantly, did not result in any sort of general facilitation following brain injury.

The second experiment of the study tested whether the drug would facilitate the recovery of a preop-

eratively learned two-choice brightness discrimination. The procedure was the traditional Lashley paradigm in which normal rats learn a brightness discrimination, are subjected to posterior visual decortication, and, after a 2-week rest, are returned to the apparatus for postoperative retraining. However, during the postoperative retraining, half of the animals were injected with 6 mg of 8-azaguanine, while the remaining animals were injected with an equal volume of physiological saline. The results were quite striking and exactly followed our prediction. That is, the animals injected with 8-azaguanine recovered the preoperative brightness discrimination significantly faster than the animals given an injection of physiological saline. Moreover, it is most important to remember that this facilitation is just the opposite of the interference produced by the same injection of 8-azaguanine when decorticate rats were required to initially learn a brightness discrimination. We thus believe that the results are rather strong evidence for the notion that the behavioral deficit subsequent to brain injury may reflect a shift to nonaffected neural systems and an attempt to acquire new behaviors to compensate for the neural systems directly affected by the injury. And we believe this because, when new learning is impaired, there is a corresponding moderation of the behavioral deficit as reflected by more rapid recovery of the disrupted preoperative behavior.

However, there is one subtle problem that must be resolved before fully accepting this conclusion. This problem is that, simply because 8-azaguanine will similarly disrupt acquisition in both normal and decorticate rats, it does not also necessarily mean that the drug's facilitation of recovery of function is because it disrupts acquisition. That is, we are suggesting that 8-azaguanine facilitated recovery of function because it interfered with learning and prevented compensation. However, it is possible that the RNA antimetabolite might somehow directly potentiate spared neural mechanisms independent of, or in addition to, its effects on learning. If this were the case, then our speculation that compensation is responsible for the behavioral deficit would not be supported, perhaps even compromised, by the above experiment. Thus, to support the conclusion that the behavioral deficit is the result of an attempt on the part of the animal to compensate for brain injury, we were required to demonstrate that 8-azaguanine would not directly potentiate spared neural mechanisms. The final experiment of the Davis and LeVere study was an attempt to provide this demonstration.

In this final experiment, rats were preoperatively trained on a two-choice brightness discrimination for water reward. Subsequently, the animals were subjected to visual decortication and, after a 2-week rest, returned to the apparatus and trained on the same brightness discrimination but now to avoid foot-

shock. Previous results using this procedure (LeVere & Davis, 1977) demonstrated that, while the memory engrams of the preoperatively learned brightness discrimination for water reinforcement were spared, they did not influence the postoperative performance of a brightness discrimination learned to avoid foot-shock. On the basis of this result, one of two predictions would be made depending upon how 8-azaguanine facilitated recovery of function in the previous experiment. On the one hand, if 8-azaguanine somehow directly potentiated the effectiveness of spared neural traces, then one would expect the drug to facilitate the postoperative performance of the brightness discrimination even if motivation is changed. In this case, our suggestion that the postoperative behavioral deficit was the result of an attempt to compensate for damaged neural systems would have to be revised. On the other hand, since the spared preoperative neural traces do not typically influence postoperative behavior when motivation is changed, then, necessarily, the reinstatement of the brightness discrimination must be a relearning process. In this case, if the only effect of 8-azaguanine is to disrupt acquisition, then the drugged animals should be impaired relative to the saline controls. In this case, our suggestion that 8-azaguanine facilitated recovery of function by interfering with compensation would stand. The results of the experiment were in accord with the latter prediction, and the postoperative administration of 8-azaguanine significantly impaired the recovery of the preoperative brightness discrimination when motivation was changed. Thus, there is apparently no special or unique effect of 8-azaguanine, given the existence of spared neural mechanisms. Rather, we would suggest that the only effect of the drug is to interfere with acquisition, and whether or not this facilitates recovery of function depends upon whether or not compensation, that is, new learning, will retroactively interfere with the postoperative expression of spared neural mechanisms.

We may thus finally summarize our overall position as follows. An individual's initial behavior subsequent to brain injury is an attempt to respond on the basis of the best neural systems that are currently available. This, we suggest, will necessarily preclude those neural systems directly affected by the injury independent of what of these systems may be spared. Because the individual no longer utilizes the neural systems affected by the lesion, there will be a corresponding requirement that the individual attempt to compensate for those behaviors mediated by the damaged neural system. If the compensatory behavior leads to goal attainment, then from the standpoint of meeting his or her needs, the individual has no behavioral deficit. And, accordingly, there is no need for recovery of function to occur and it will not. However, if the compensatory behavior does not

lead to goal attainment, then, from a similar standpoint, there is a behavioral deficit. In this latter instance, if there is sufficient sparing, then recovery of function will be possible. On the other hand, if there is not sufficient sparing, then there will be a behavioral loss. What is important about this viewpoint is that the behavioral deficit following brain injury is not necessarily the loss of the neural mechanisms supporting a particular behavior. Rather, the behavioral deficit is a shift in what neural systems are used to control behavior. In turn, recovery of function represents a process in which the individual returns to the neural system directly affected by the brain injury and begins to utilize what is spared of this system.

A Word on Breadth

There is yet one final consideration. This is whether or not the propositions that have been considered can offer anything to a more general understanding of recovery of function. I believe that they can, and, at the risk of straining what is perhaps an overly long discussion, four examples will be briefly outlined.

First, the present proposition directly addresses and provides an explanation of why some behaviors may be recovered and others may not. Those behaviors that are ultimately recovered are recovered simply because the neural centers mediating their performance were spared by the brain injury. Those behaviors that are not ultimately recovered are not recovered because either compensation was successful or the neural centers mediating their performance were destroyed by the brain injury. While this may seem rather simplistic, I submit that, more often than not, both classic and contemporary explanations of recovery of function do not address the failure to recovery of function. Rather, there is the implication that if we knew enough and if we were able to make the correct manipulations, then recovery of function would always occur. The unescapable and unfortunate fact is that this is just not so.

Second, there are a number of parameters of recovery of function that may be understood in terms of the present theory. Consider, for example, the so-called serial lesion effect. The data indicate that if a brain injury is sustained in successive stages, there is a likelihood that the terminal behavioral deficit will be less than if the injury was sustained all at once. From our present position, we would suggest that this is simply an instance of minimal brain injury not inducing a shift to nonaffected neural systems and a consequent attempt to compensate. That is, the initial brain damage, of course, produces some degradation of the involved neural system. However, because of such things as stimulus and response generalization, this degradation is not sufficient to induce an attempt to compensate for the injury with unaffected neural

systems. Rather, the individual still utilizes what is spared of the damaged neural system and essentially shows no behavioral deficit. The events following the second lesion are similar except that now the animal is behaving from a different base level. That is, having continued to use the affected neural system following the initial lesion, the individual is adequately behaving with a less than optimal neural system. The second lesion, while inducing some further degradation, is, however, again not sufficient to induce an attempt to compensate with unaffected neural systems. Thus, while the effects of each lesion are additive, the degradation associated with each individual insult is never sufficient by itself to cause the individual to shift away from the neural system affected by the lesion. On the other hand, if a similar total injury is sustained in one blow, then the degradation is severe enough to cause the animal to shift away from the affected neural system and to attempt to compensate for this degradation. It is just this shift and compensation which we suggest produces the behavioral deficit following neurological insult.

Following this line of reasoning, one may then explain what I believe is one of the more perplexing results associated with the serial lesion phenomenon. This is that if rats are maintained in the dark between successive unilateral striate lesions, then there is no sparing of visual functioning (Meyer, Isaac, & Maher, 1958). At first glance, it would seem that being kept in the dark should be beneficial to the lesioned animal because it should prevent learning and attempted compensation, and should thus mitigate the behavioral deficit. However, I would argue that being kept in the dark actually forces a shift to nonvisual neural systems and the occurrence of compensation. I would argue this because the visual degradation produced by the unilateral lesion is compounded by the fact that, in the dark, the animal is unable to effectively utilize those portions of the visual system that are spared by the initial unilateral lesion. This, in turn, necessitates that the animal respond on the basis of other sensory information, that is, shift to another neural system, to compensate for the combined effects of the brain injury and the dark environment. And, in fact, this shift to other nonvisual neural systems and this compensation will be successful until subsequent to the second unilateral visual lesion. At this time, the compensatory behaviors evolved with nonvisual neural systems will be inadequate to the performance of the preoperatively acquired visual behavior, and there will be a behavioral deficit. While this is, of course, just one example, we believe that similar reasoning may be applied to other parameters of recovery of function such as infant lesions and overtraining.

Third, from a more practical view, the present theory of recovery of function suggests that the first postoperative therapeutic effort should be an attempt to recover those specific behaviors disrupted by the

brain injury. Furthermore, this attempt should be at the very earliest opportunity in order to minimize the possibility that the individual will shift from the damaged neural system and attempt to compensate. In fact, should the present theory enjoy continued support, it would not seem unreasonable to more fully investigate the possibility of short-term pharmacological prevention of new learning as a therapeutic maneuver to facilitate recovery of function. In this respect, it should also be noted that it is always possible to attempt to compensate for the detrimental effects of neural lesions. However, it is not yet known for certain whether or not recovery of function may be permanently impaired if compensatory behavior patterns are allowed to become firmly established.

It is perhaps appropriate to point out lastly that the present notions of recovery of function have a certain distinct advantage over many other conceptualizations of the effects of brain injury. This advantage is that the behavioral deficit and the recovery process are directly addressed without appealing to any new or vague neural processes. This, then, means that the heart of the theory may be tested without first establishing the validity of any proposed novel neural mechanisms. That is, we are not burdened by the necessity of establishing the existence of, say, functional or structural reorganization before determining their relevance to recovery of function. The inadequacy of much of contemporary research on recovery of function, and what makes the results less than satisfying, is that we are first asked to accept on faith the validity of rather obscure neural processes or events to justify the empirical results. This is not to imply that the ultimate explanation of recovery of function is not at the level of basic brain physiology. Surely it is. But we must be able to investigate and account for the behavioral effects of brain injury and recovery of function without first chasing the elusive Hobbit into Mordor.

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