11 Learning and Memory Without a Brain

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Over the past 40 years, the study of animal learning has gone through a radical metamorphosis. Animal learning of the 1960s variety was largely dominated by researchers who took Watson's behaviorist dictates as scripture. They hoped to explain complex behavior in simple stimulus-response (S-R) terms while avoiding reference to unobservable constructs. They were, in retrospect, the killjoys of creativity, always suspicious of anything new and wary of most internal or theoretical constructs. Against this backdrop came the cognitive approach, opening the doors to a new faith that promised greater tolerance for internal constructs.

Today we often take for granted that all higher vertebrates process information in a limited-capacity device (short-term memory or STM) (Wagner et al. 1973), that attention guides the learning process (Mackintosh 1975), and that the organism can deduce the connection between its behavior and an outcome in its environment (Maier and Seligman 1976). Yet 30 to 40 years ago, each of these claims was greeted with skepticism, with many researchers questioning whether explanations of animal behavior required such cognitive constructs.

I began my research career well indoctrinated in the cognitive approach, and I championed its benefits (e.g., Moye et al. 1981; Rescorla et al. 1985; Grau 1987a). However, in recent years, my collaborators have led me down an alternative path that I initially thought led in an incomprehensible direction, a course that sought evidence of learning and memory within a vertebrate that effectively lacks a brain. As we will see, this path led to a new vista, one that has forced me to question my cognitive faith. It seems that many of the behavioral effects I thought were best described in cognitive terms can be observed in the absence of a brain. I now find myself in the unfortunate position of the killjoy, questioning the application of cognitive constructs to infrahuman species.

Memory within the Spinal Cord

The starting point for this work was a series of studies that explored how the body (and mind) regulate pain. I had shown that exposure to a mildly painful stimulus engages an inhibitory mechanism that reduces behavioral reactivity to subsequent noxious stimuli, a phenomenon known as antinociception. For example, in rats, exposure to a few brief tail shocks inhibits tail withdrawal from radiant heat (the tail-flick test). This response is mediated by a nociceptive (pain signal) reflex that is organized within the spinal cord; it is a spinal reflex that can be readily elicited after the lower spinal cord has been surgically disconnected from the brain. Exposure to moderate shock appears to inhibit nociceptive reactivity (antinociception) by engaging neural mechanisms within the brain that inhibit spinal nociceptive reflexes through descending pathways. I argued that the memory of the aversive event helped maintain the antinociception after shock exposure. Specifically, I argued that the central representation of the aversive event in short-term memory continues to drive the antinociceptive systems after exposure to shock, producing an antinociception that lasts 10 minutes or more.

Short-term memory in humans is generally envisioned as a kind of rehearsal buffer where information can be temporarily maintained (e.g., Atkinson and Shiffrin 1968). Because it is thought to have a limited capacity, it is subject to distraction; new information can disrupt the rehearsal of items already in STM, causing the memory of them to decay rapidly. Interesting, a distracting stimulus also disrupts memory in other animals, which suggests that they too process information in an STM-like device (Wagner 1981).

Assuming this perspective, I reasoned that if the central representation of an aversive event in STM maintains the activation of the antinoci-

ceptive systems, then displacing this memory with a distractor should cause the antinociception to decay more rapidly. As predicted, presentation of a visual distractor (a flashing light) after shock decreased the duration of the subsequent antinociception (figure 11.1A) (Grau 1987a).

To derive predictions from this memory-oriented perspective, I had to assume that the magnitude of antinociception observed depends on the hedonic value of the representation; the more aversive the memory, the greater the antinociception. Given this, suppose rats are exposed to a moderately intense shock (1 mA) followed by a weak shock (0.1 mA) distractor. The weak shock should displace the memory of the strong shock. By introducing a "better end" (Kahneman et al. 1993), I should be able to reduce the magnitude of antinociception observed. As predicted by this memory-based account, subjects that experienced a weak shock after moderate shock exhibited less antinociception (figure 11.1B) (Grau 1987b). Normally, adding shock increases the magnitude of antinociception. Because adding a weak shock had the opposite effect, the finding appeared to provide particularly strong evidence that memorial systems mediate the generation of antinociception.

A few years later, Mary Meagher examined whether incoming nociceptive signals can engage antinociceptive systems within the spinal cord in the absence of the brain (Meagher et al. 1993). To address this issue, she cut the spinal cord of rats at the second thoracic vertebra (T2). This operation produces a condition in which the rat can move about using its front paws, but is paralyzed below its midsection. She found that the moderate shocks used to study the activation of antinociceptive systems in intact subjects had little effect on tail-flick latencies after the spinal cord was transected. However, exposure to severe shocks, which were more intense and longer, caused a dramatic increase in tail-flick latencies.[1]

We assumed that this antinociception reflected an unconditioned response, a passive reaction to the noxious stimulus that was independent of learning and memory, processes everyone knew required a brain. Given this, a shock distractor should not cause the antinociception observed in a spinally transected rat to decay more rapidly. Rather, in this case, more shock should produce greater antinociception. To test this, one group (Int Shk → Nothing) of spinal rats was exposed to three intense shocks that Meagher had shown induce a strong antinociception. Another group (Nothing → Mod Shk) received moderate shock alone, which produces only a weak antinociception in spinalized rats. Because the moderate shock was apparently detected, but has only a weak effect, it was used as the distractor in this study (Grau et al. 1990).

We expected that the distractor would have an additive effect and, if anything, augment the antinociception produced by intense shock. Contrary to our expectations, rats that received the moderate shock distractor after intense shock (Int Shk → Mod Shk) exhibited an antinociception that decayed more rapidly (figure 11.1C), a result that formally mirrors the results obtained in intact rats (figure 11.1B).

Because these findings ran counter to my theory (Grau 1987a), we sought additional evidence. Indeed, this has remained a common feature of all our studies; because we generally doubt the processing capacity of spinal cord neurons, we routinely hold this system to a higher standard. In the present case, this led us to examine the effect of reversing shock order. According to the memory hypothesis, a distractor should be effective only if it is presented after the target event. Presenting a shock distractor prior to a strong shock should have no effect. Again, the results were consistent with our memory-oriented perspective (figure 11.1D). Spinalized rats that experienced the shock distractor before the intense tail shock (Mod Shk → Int Shk) exhibited a robust antinocicep-

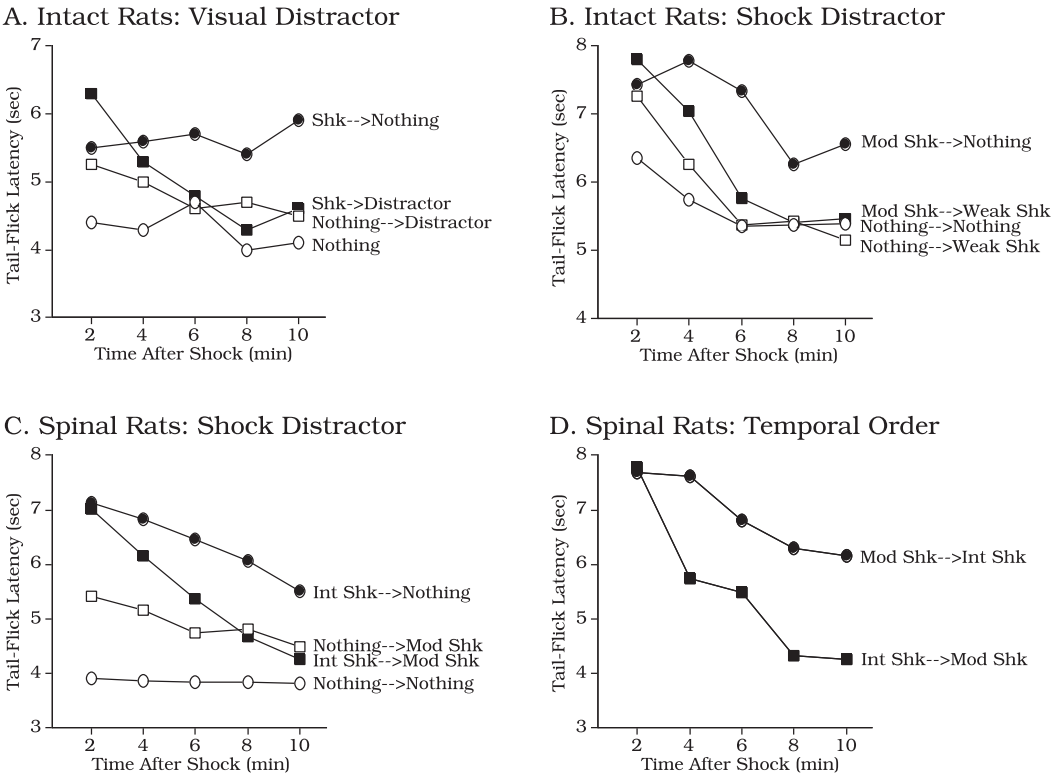

Figure 11.1
A postshock distractor speeds the decay of antinociception in intact and spinal rats. (A) Rats that received shock alone (Shk → Nothing) exhibited longer tail-flick latencies (antinociception) over the 10-minute test period. Presentation of a visual distractor (Shk → Distractor) caused the antinociception to decay more rapidly. (B) Presentation of a weak (0.1 mA) shock distractor after the moderate (1 mA) tail shock (Mod Shk → Weak Shk) also caused the antinociception to decay more rapidly. (C) Exposure to more intense (3 mA) tail shock (Int Shk → Nothing) increased tail-flick latencies in spinally transected rats. Exposure to a moderate (1 mA) tail shock alone had relatively little effect (Nothing → Mod Shk). Presentation of a moderate shock distractor after each intense shock (Int Shk → Mod Shk) caused the antinociception to decay more rapidly. (D) A shock distractor only caused antinociception to decay more rapidly when it was presented after intense shock (Int Shk → Mod Shk); rats that experienced the same amount of shock, but in the opposite temporal order (Mod Shk → Int Shk), exhibited a longer-lasting antinociception. (Adapted from Grau 1987a,b and Grau et al. 1990.)

tion, while rats that received exactly the same amount of shock, but in the opposite order (Int Shk → Mod Shk), did not (Grau et al. 1990).

Pavlovian Conditioning and Attention

Subsequent studies have explored whether nociceptive mechanisms within the spinal cord are sensitive to Pavlovian relations. In intact rats, a conditioned antinociceptive response can be established by pairing a conditioned stimulus (the CS+) with an aversive tail shock (the unconditioned stimulus, or US). After a few pairings, the CS+ generates an antinociception on the tail-flick test relative to another cue (the CS−) that was presented in an explicitly unpaired fashion (figure 11.2A).

To study Pavlovian conditioning within the spinal cord, Juan Salinas used cutaneous electrical stimuli that were applied to the left or right hind legs (figure 11.2B). These served as our CSs, one of which (the CS+) was paired with an intense tail shock 30 times while the other (the CS−) was presented in an unpaired fashion. An hour later, we tested tail-flick latencies during each CS. Subjects exhibited longer tail-flick latencies during the CS+ (conditioned antinociception), and this effect extinguished over the course of testing (Grau et al. 1990).

In intact subjects, preexposure to the CS alone prior to training generally undermines the acquisition of a conditioned response to that CS, a phenomenon known as latent inhibition. A common account of this effect assumes that learning is regulated by attentional mechanisms (e.g., Mackintosh 1975). From this perspective, the repeated presentation of a CS alone decreases its capacity to command attention and thereby undermines the rate of learning. Again, most researchers believe that attention is the province of the brain and, consequently, an explanation couched in attentional terms seemingly would predict that preexposure to the CS alone would have little effect on an intraspinal learning mechanism. Yet once more our preconceptions proved wrong, for we found that the presentation of the CS alone prior to training undermined conditioning in spinalized rats (Illich et al. 1994).

Another effect that is often described in attentional terms is overshadowing. In an overshadowing experiment, subjects experience a compound cue composed of two elements. These elements are chosen to differ in their salience, or noticeability. One element (*B*) is very salient while the other (*X*) is much less salient. A control group is included to show that subjects can learn about the less salient element (*X*) when it is presented alone and paired with the US. But when *X* is presented jointly with the more salient cue *B*, and the two are paired with the US, intact subjects later exhibit a conditioned response to *B*, but not to *X*. It appears that the more salient element overshadows the less salient cue. A popular account assumes that subjects naturally attend to the more salient element. Because they fail to attend to *X* during training, they fail to learn about it.

We reasoned that learning within the spinal cord could be governed by a simple rule that depends solely on the number of CS–US pairings (contiguity). At this level of the nervous system, there may be no cue competition. Certainly, attention does not exist, at least in the usual, cognitive, sense of the term. Paul Illich evaluated these possibilities by training spinalized rats with a compound CS (Illich et al. 1994). Stimulation of one hind leg at the usual intensity served as the *X* element. A more salient element (*B*) was provided by stimulating the opposite (contralateral) leg at a higher intensity. As usual, rats that experienced *X* alone paired with the US exhibited conditioned antinociception relative to a group that experienced *X* and the US unpaired. More important, rats that experienced *X* in compound with *B* during training did not exhibit a conditioned antinociception when *X* was presented alone. It appears that the concurrent presenta-

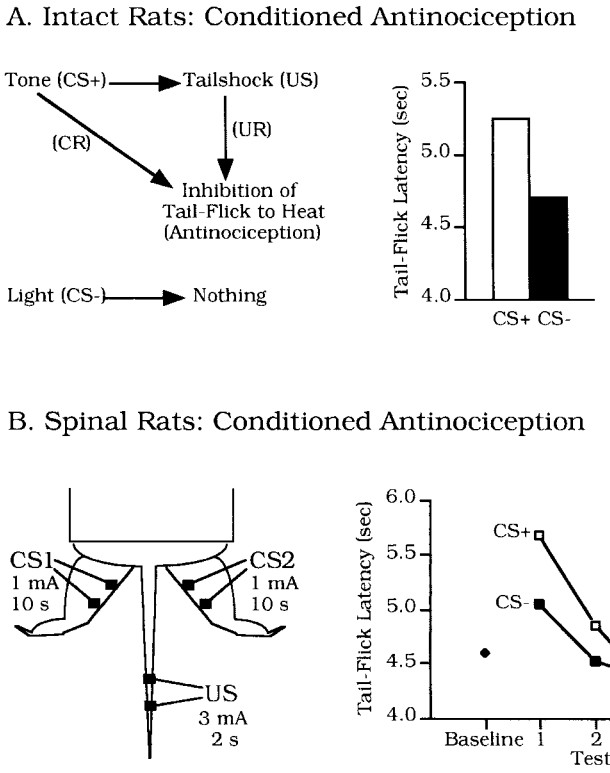

Figure 11.2
Intact (upper panels) and spinalized (lower panels) rats exhibit longer tail-flick latencies during a conditioned stimulus (CS) that has been paired with tail shock (conditioned antinociception). (A) In intact rats, conditioned antinociception can be demonstrated by pairing an auditory cue (a tone) with tail shock (the unconditioned stimulus, US). (B) After a few pairings, rats exhibit longer tail-flick latencies during the paired cue (the CS+) relative to an unpaired cue (the CS−) that was presented an equal number of times. (C) Conditioned antinociception can be demonstrated in spinalized rats by pairing stimulation of one hind leg (the CS) with an intense tail shock (the US). (D) After 30 CS-US pairings, rats exhibited longer tail-flick latencies during the CS+, relative to the CS−. Repeated presentation of the CSs alone during testing caused the CS+/CS− difference to extinguish. (Adapted from Grau et al. 1990.)

tion of *B* with *X* undermined the conditioned response produced by *X*. Neither latent inhibition nor overshadowing necessarily require a brain.

Instrumental Learning and the Cognition of No Control

More recently, we have begun to explore whether neural systems within the spinal cord can encode the relationship between a response and an environmental outcome (reinforcer), a form of learning known as instrumental conditioning. In these studies, Doug Barstow spinalized rats and then exposed them to a response-reinforcer relation using the apparatus illustrated in figure 11.3A. In this apparatus, when a shock is applied to one hind leg, it elicits a flexion response that can be monitored by means of a contact electrode taped to the rat's paw. In the master condition, a leg shock is given whenever the contact electrode touches the underlying salt solution. In this situation, intact rats will quickly learn to maintain the leg in an up (flexed) position, thereby decreasing net shock exposure. What is amazing is that spinalized rats can also learn to perform this response, exhibiting a progressive increase in duration of flexion as a function of training (figure 11.3B) (Grau et al. 1998; Grau and Joynes 2001).

A critic could charge, however, that this reflects an unconditioned response to shock. For example, perhaps shock causes a tetanuslike effect that produces a cumulative increase in muscle tension. To address this possibility, a yoked control was included. Each yoked rat was experimentally coupled to a master rat and received the same shock, but independent of leg position (noncontingent shock). If the change in flexion duration reflects an effect of shock per se, these subjects should also exhibit an increase in flexion duration, but this was not observed (figure 11.3B). Learning, as evidenced by an increase in flexion duration, was observed only when there was a response-reinforcer contingency; remove the contingency and the learning disappears.

To meet the criteria for instrumental learning, we must also show that the experience has an effect that outlasts the environmental contingencies used to produce it, that the consequence of learning can be observed when subjects are later tested under common conditions (Grau et al. 1998). Robin Joynes addressed this issue by testing subjects with response-contingent shock (figure 11.3C). Now all of the subjects had an opportunity to decrease net shock exposure by exhibiting a flexion response. We found that previously trained subjects (master) exhibit some savings and reacquire the instrumental response more rapidly than a control group that had never been shocked (unshocked). Amazingly, yoked rats fail to learn and this is true even if they are tested on the opposite (contralateral) leg (Joynes et al., submitted). It appears that prior exposure to noncontingent shock undermines instrumental behavior, a behavioral deficit reminiscent of the phenomenon of learned helplessness (Maier and Seligman 1976; Overmier and Seligman 1967).

Researchers studying the consequences of noncontingent shock in intact animals have suggested that learned helplessness occurs when the organism develops a cognition of no control (Maier and Seligman 1976; Maier and Jackson 1979). If the cognition of no control mediates the deficit, then behavioral manipulations that alter this cognition should influence the deficit. For example, showing the subject that it can control its environment in some situations might protect it from becoming helpless. Conversely, experiencing a contingent response–outcome relation could reverse the cognition of no control and thereby attenuate the deficit. As predicted, training with contingent shock before (immunization) or after (therapy) inescapable shock attenuates learned helplessness in intact subjects (Seligman et al. 1968, 1975). Notice that the addition of escapable shock training actually increases the

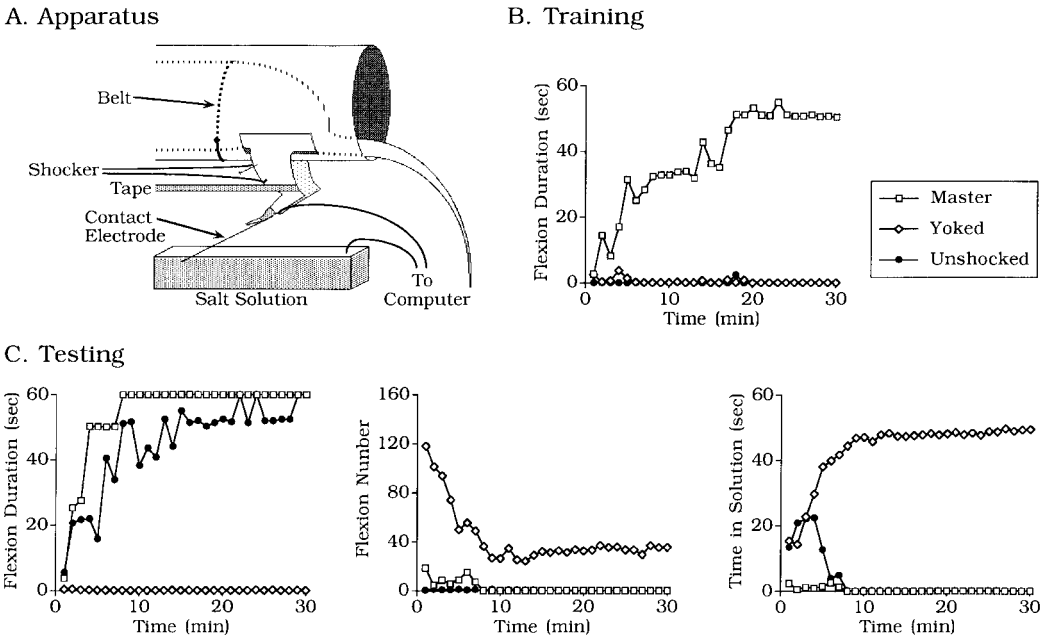

Figure 11.3
(A) The apparatus used to study instrumental learning in spinalized rats. (B) Spinalized rats that received a leg shock whenever the contact electrode touched the saline solution (master) exhibited a progressive increase in flexion duration over the course of the 30-minute training period. Yoked rats that received the same amount of shock independent of leg position did not exhibit an increase in flexion duration. (C) Subjects were then tested under common conditions with response-contingent shock. From flexion duration (left), we can see that rats previously trained with a contingent shock (master) learned more rapidly than a control group that was previously unshocked. Yoked rats repeatedly touched the underlying solution and exhibited the greatest number of flexions (middle). However, this experience with response-contingent shock did not produce an increase in flexion duration (left). As a consequence, yoked rats spent more time contacting the underlying salt solution (right) and received more shock. (Adapted from Grau et al. 1998.)

net exposure to the aversive event. This highlights an essential feature of the phenomenon: It is not the net exposure to shock (aversive stimulation) that is critical, but rather the organism's *perception* of whether the environmental event is controllable or uncontrollable (Maier and Seligman 1976).

Would training with contingent shock affect the development of the behavioral deficit within the spinal cord? To examine this issue, Eric Crown first trained spinalized rats with contingent shock applied to one hind leg (Crown and Grau 2001). Next, subjects received intermittent tail shock at an intensity and duration known to induce a robust behavioral deficit (Crown et al., submitted). Finally, subjects were tested with contingent shock applied to the contralateral hind leg. As usual, rats that experienced noncontingent tail shock failed to learn, but rats that had experienced contingent shock prior to tail

shock acquired the instrumental response at the same rate as the unshocked controls. Evidently training with contingent shock has a protective effect within the spinal cord.

We also examined whether the deficit could be reversed by exposing rats to contingent shock. In this experiment, the deficit was induced by exposing some spinalized rats to intermittent tail shock. Next, a subset of the subjects was given behavioral therapy by training the instrumental response in the presence of naltrexone. Robin Joynes had previously shown that the administration of this opioid antagonist temporarily blocks the expression of the behavioral deficit (Joynes and Grau, in press). Thus, while the drug is present, rats that had previously received inescapable shock were able to learn the instrumental response. Subjects were then tested with response-contingent shock 24 hours later, after the drug had cleared the system. We found that instrumental training in the presence of naltrexone attenuated the behavioral deficit. It seems that instrumental training has a therapeutic effect that helps restore behavioral potential within the spinal cord.

Implications for the Study of Animal Cognition

I admittedly began these experiments as a skeptic, convinced that the spinal cord was largely ineducable. I felt that the behavioral effects I had been studying in intact subjects were best described in terms of cognitive processes. Within a cognitive framework, I had provided evidence that the central representation of an aversive event maintains the activation of antinociceptive systems in intact rats (Grau 1987a,b). To my surprise, the experimental manipulation used to explore this hypothesis generated an identical pattern of results in the absence of the brain (Grau et al. 1990). Similarly, Pavlovian phenomena such as latent inhibition and overshadowing, which are often described in attentional terms, were observed in our spinal preparation (Illich et al. 1994). And experimental designs used to explore the cognition of no control produced remarkably similar results without a brain (Grau et al. 1998).

Our studies suggest that neural systems within the spinal cord are quite a bit smarter than most researchers have assumed. The spinal cord is not a simple conduit for incoming and outgoing neural impulses, but rather it is a complex information-processing system that can learn from experience. [See Patterson and Grau (2001) for other demonstrations of spinal cord plasticity and Patterson (2001) for a review of earlier studies.] It seems that many basic learning phenomena are not localized to particular brain regions, but instead reflect inherent characteristics of neural systems capable of learning, and that the capacity for learning is widely distributed.

What implications does our work have for the study of comparative cognition? Perhaps the most important is a note of caution. While we can readily design experimental procedures that appear formally similar to those used to study a human cognitive capacity (e.g., STM), similar results do not necessarily imply that the same mechanisms are at work. Organisms have most likely evolved many ways to solve environmental puzzles (Joynes and Grau 1996), just as they have evolved many ways to move about. Sensitivity to distraction alone cannot be taken as evidence for STM. Nor can latent inhibition and overshadowing be taken alone as evidence for attention. The same applies for many of the other behavioral tasks used to infer cognitive processing in infrahuman species.

The way in which sensitivity to distraction, cue competition, and the disabling of behavioral potential by noncontingent shock are represented neurally surely varies across different levels of the nervous system. Similar variability must also exist across species. Yet, at a functional or behavioral level, our experimental manipulations often yield similar results. It appears that some operational principles remain surprisingly stable across different neural architectures. Some persons will read this as consistent with the view of

the behaviorists and their desire to isolate the general principles of learning. Paradoxically, others will see this as favoring a belief common within cognitive psychology, that the operational principles of an information-processing system can be explored with little attention to the underlying architecture. What we must remember, however, is that nature can write a program for behavior in many ways. Sometimes these programs may call upon cognitive functions, but sometimes they will not. In the latter case, our cognitive description of the behavioral effect is just a metaphor.

Do cognitive systems allow operations that noncognitive systems cannot achieve? Perhaps our studies of learning within the spinal cord can provide some hints, for we remain convinced that the brain (and cognition) adds something to our intellect. At the most obvious level, brain systems are tied to a much more elaborate system for detecting and integrating sensory information. Neural mechanisms in the brain (e.g., the hippocampus) allow the brain to span gaps in time (trace conditioning) (Woodruff-Pak 1993; Clark and Squire 1998), to mediate learning in the absence of a physical stimulus (Holland 1990), and to derive new configural representations (Sutherland and Rudy 1989).

Similarly, whereas the spinal cord may have but one response to an instrumental problem, the brain can select from a range of response alternatives that are tuned to particular environmental situations and temporal-spatial relations (Timberlake and Silva 1995). As a result of these elaborations, brain-mediated behavior often appears more flexible and adaptable, a category of behavior Skinner (1938) distinguished from elicited responses (respondents) by the term *operant learning*. [For a discussion of the relation between instrumental and operant learning, see Grau (2000).]

Researchers rely on operant contingencies when they train an animal to select a stimulus that matches a recently presented novel object (delayed matching-to-sample) or to choose the spatial location they first visited (Blough 1959; Kesner and Novak 1982). Operant behavior of this sort presumably requires a brain. It may also require cognition. Perhaps then it is operant behavior that provides the clearest window into animal cognition. Ironically, our best tool for studying cognition may come from the workbench of Skinner, a researcher who rejected the cognitive approach.

Note

1. People describe our moderate shocks as mildly painful. Severe shocks would presumably elicit intense pain, but because the spinal transection prevents the sensory signal from reaching the brain, spinalized rats do not "consciously" experience pain during severe shock.

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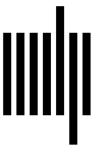

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