CHAPTER 5

THE LOWER BOUNDS
OF COGNITION: WHAT
DO SPINAL CORDS
REVEAL?

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1. Introduction

Why should philosophers care about the spinal cord? After all, philosophers have mostly cared about neurons at all only insofar as they are relevant to the mind–body problem, and the assumption that the brain is where all the cognitively interesting action lives is practically built into the jargon of philosophy. Thus, the mind–body problem is often recast as the mind–brain problem, and generations of philosophers have cut their teeth on the mind–brain identity thesis. Similarly, the compound term mind–brain is frequently used by authors to signal their materialist bona fides, as in “We are interested in how consciousness arises in the mind–brain.” This association of mind with brain is not just built into the discipline of philosophy, for neuroscience institutes from Johns Hopkins to Sydney to UC San Diego to Lausanne all have some permutation of mind and brain in their titles.

Ironically, the ubiquitous example used by philosophers to illustrate the mind-brain identity thesis is that pain is identical to C-fiber stimulation. Yet, as Puccetti (1977) pointed out long ago, this is hardly a good candidate for mind–brain identity given that C-fibers are located well outside the brain, existing as a subset of the
sensory neurons that project to the spinal cord. Even more incongruously, many
philosophers who haven't bothered to study their neuroanatomy have relocated
C-fibers inside the brain in their expositions of the identity thesis, and this error
has made it into at least one textbook that is still in print.

Complementary to this mind–brain association is the idea that the parts of the
nervous system that lie outside the brain, including the spinal cord and the periph-
eral sensory and motor neurons, are mere conduits of sensory information and
motor commands. As such, they constitute the trunk and branches of an extensive
signal relay system that conveys sensory and proprioceptive information to the brain
and relays its signals back to the muscles and organs. The implicit assumption for
those who identify mind with brain is that there is no cognition outside the brain.
Indeed, behavioral responses to sensory stimuli that do not involve brain media-
tion are often called "spinal reflexes," suggesting that they are fixed, automatic, and
noncognitive in nature.

In this chapter, we review animal research that challenges this picture by show-
ing the (rat) spinal cord to be a flexible and interesting learning system in its own
right, and we discuss the consequences of these findings for philosophical under-
standing of the relationship between learning, cognition, and even consciousness.
Although spinal plasticity has been hinted at for many years, the extent of its flex-
ibility remains underappreciated. In a sense, it is hardly surprising that the nervous
tissue in the spinal cord should have many of the same self-organizing and adaptive
capacities as nervous tissue in the brain, but our view is that a full appreciation of
the sophistication of the spinal cord raises some important questions about cogni-
tion. To answer our opening question, philosophers who master the details of these
results will be in a much better position to discuss mind–body relationships.

It is currently fashionable in the philosophy of mind to discuss the "extended
mind" hypothesis that the human mind literally extends outside the boundaries
of the human organism into our interactions with "cognitive technologies," such
as writing and the World Wide Web (Clark, 2003). Given such a radical envi-
ronment, our discussion of whether cognition extends into the spinal cord may seem
rather timid. We think that a good look at the actual science of spinal cord learning
has more potential for helping philosophers understand the boundaries of cogni-
tion than any number of thought experiments about cognitive technologies. In the
end, we do not argue that the spinal cord is either cognitive or conscious. We do
argue, however, that the sophistication of spinal learning mechanisms in the rat,
and presumably in all mammals and most other vertebrates, places certain long-
held assumptions about the concepts of cognition, mind, and consciousness under
the spotlight. (See Rockwell, 2005, for another biologically inspired but more rad-
ical approach to extended mind.)

An additional reason for philosophers to care about the spinal cord concerns
questions about the relationship between behavioral evidence and cognitive attribu-
tions. At various stages of our careers, we have each advocated taking a cognitive
approach to animal behavior. But data that Grau and Meagher have collected over
the past two decades have challenged our thinking about these issues. In particu-
lar, these findings suggested that some of the data taken to demonstrate cognitive processing in nonhuman animals could also be obtained in the absence of a brain. Specifically, spinal systems appear to be sensitive to distraction, exhibit cue competition, and show a form of learned helplessness. Distraction and cue competition are typically connected with "attention," a central notion in cognitive science, whereas learned helplessness has been linked to a cognition of no control (Maier and Jackson, 1979; Maier and Seligman, 1976). We are not arguing that these phenomena as manifested in the spinal cord are identical to what's found in intact organisms when brains are fully engaged. We are saying, however, that apparent evidence of attentional phenomena must be treated carefully if a strong case for mental state attribution is to be made. A central issue here is just how similar a pattern of results has to be to demonstrate a level of functional equivalence that makes the use of cognitive terms anything more than a handy metaphor that helps scientists remember the overall pattern of the data.

We also believe that concepts of learning deserve more attention from theoreticians, including philosophers of the neural and behavioral sciences. Organisms may have multiple mechanisms for encoding relations between events in the environment and between those events and their own behaviors. A properly neuroscientific approach to those mechanisms will seek to characterize them at the "neurofunctional" level (Grau and Joynes, 2005a, 2005b) rather than, as was traditionally the case among psychologists, in terms of experimental methods. These issues are relevant to philosophers interested in issues of multiple realization of functional kinds. Aside from its philosophical interest, the research described here may have important implications for managing spinal cord injuries (Grau and Hook, 2006), as we'll explain further later.

2. Antinociception and Conditioned Antinociception

The spinal cord has long been a focus of investigation of pain researchers. The Melzack-Wall gate theory (Melzack and Wall, 1965; Wall and Melzack, 1962) located a significant part of the regulatory control of vertebrate pain signals in the dorsal horn of the spinal cord, where peripheral A and C fibers relaying signals from nociceptors (neurons functionally specialized to sense noxious stimuli) converge with descending neurons from the brain. Traditionally, pain inhibitory effects due to learning, memory, and other cognitive processes were assumed to come top-down from the brain. Grau's initial research on spinal mechanisms of nociception was conducted within this conceptual framework.

The first unexpected result emerged from some studies on shock-induced antinociception (a form of stress-induced analgesia). It had been shown in several laboratories in the early 1980s that exposure to a mildly aversive shock can induce an inhibition in pain reactivity (Grau, 1987a). Pain reactivity was often measured using
the tail-flick test, which assesses the latency at which a rat withdraws its tail from a radiant heat source. Tail flick is a reflexive response organized by neurons within the spinal cord (i.e., a spinal reflex). Because the response is spinally mediated, a tail withdrawal from noxious heat can be elicited after a mid- (thoracic) spinal cord transection. This transection completely eliminates all sensory–motor communication between neurons within the lower spinal cord and the brain, producing a form of paraplegia. Consequently, brain mechanisms remain unaware of stimuli presented below the transection. Because rats normally rely on their forelimbs to guide locomotor activity, a paraplegic rat has little trouble moving about its home cage, shows little evidence of pain or distress, and continues to eat and drink on its own.

In normal, intact, rats the tail flick reflex is modulated by brain systems through descending fibers that regulate the incoming pain signal. Grau (1987a) had worked to develop a model of when these descending inhibitory mechanisms are engaged, suggesting an account linked to Wagner’s “Sometimes Opponent Processes” (SOP) model of automatic memory processing (Wagner, 1981). Grau had found that mildly painful electric shock could reduce reactivity to a subsequent noxious stimulus; for example, a few brief shocks to the tail would result in slower responses to a subsequent application of heat as measured by the tail-flick latency. This phenomenon of “antinociception” (reduction of nociception) seemed explainable in terms of brain-based short term memory of the initial painful event (the shock) causing modulation of subsequent nociception (from the heat stimulus). In casual terms, it was as if the memory of the aversive event maintained the pain inhibition, driving the descending circuits for 10 minutes or more.

2.1. Distractor-like Effects

Cognitive psychologists (beginning, e.g., with Atkinson and Shiffrin, 1968) have conceptualized short-term memory in humans as a kind of limited-capacity buffer where information is temporarily maintained but is subject to distraction by the intrusion of new information. Wagner (1981) showed that short-term memory in rats was also subject to distraction. Such a model predicts that if one could displace the memory of shock, the pain inhibition should disappear. Supporting this, Grau (1987a) showed that presenting a visual distractor (a flashing light) after a few brief shocks caused the shock-induced antinociception to rapidly decay. He then pushed the hypothesis a bit further, asking whether adding a better end could reduce the antinociception. (See Kahneman, Fredrickson, Schreiber, and Redelmeier, 1993, for a report of this phenomenon in humans.) The better end was generated using a weak shock that produced few signs of pain and little antinociception. If the memorial account is correct, a weak shock distractor should displace the memory of an earlier moderate shock and again cause the antinociception to decay more rapidly. Grau (1987b) confirmed that this occurred in intact animals. (See Grau, 2002, for a review of these and other experiments that appeared to confirm a role for short-term memory in antinociception in rats.)
Grau had assumed that the pain modulatory effects observed after moderate shock depended on brain systems, which influenced tail-flick latencies through descending fibers. Supporting this, moderate tail shock has no effect on tail-flick latencies when communication to the brain is cut by a thoracic transection (Meagher, Grau, and King, 1990). However, Meagher showed that when shock intensity is dramatically increased, it can generate antinociception in spinally transected rats (Meagher, Chen, Salinas, and Grau, 1993). Apparently, intense nociceptive input can directly engage mechanisms within the spinal cord that inhibit tail withdrawal from radiant heat. It was assumed, however, that this was a simple unconditioned response that had nothing to do with memory—at least not in the cognitive terms assumed by Grau (1987a). At this point, a difference of opinion arose between Grau and Meagher. Grau believed that when isolated from the brain, spinal mechanisms should not exhibit memory-like effects. Here, presenting a weak shock distractor should not cause the antinociception to decay more rapidly. If anything, increasing the duration of shock exposure should amplify the antinociception. In contrast, Meagher argued that spinal systems might exhibit simple forms of learning and memory-like phenomena similar to those observed in even simpler invertebrate organisms by Eric Kandel and colleagues (Carew, Hawkins, and Kandel, 1983; Dale, Schacher, and Kandel, 1988; Walters, Carew, and Kandel, 1981). If so, then the presentation of the weak shock could function as a distractor, displacing the memory of the more intense shock.

To begin to explore this issue, they performed an experiment modeled after the weak shock distractor study performed in intact rats. In this experiment, spinally transected rats received a brief intense shock followed by a weaker shock that produced little antinociception in transected subjects. Much to Grau's surprise, the weak shock distractor caused the antinociception to decay more rapidly, a pattern identical to that observed in intact rats (Grau, Salinas, Illich, and Meagher, 1990). Suspecting an artifact, Grau examined whether the temporal order of the stimuli mattered—a distractor should only have a disruptive effect when it is presented after the target event. When spinally transected rats received a weak shock before an intense shock, it had no effect; when the weak shock followed the intense shock, it caused the antinociception to decay more rapidly, as predicted by a memory-oriented account. Here, then, was a process that seemed formally equivalent to what had been found in the intact rats, albeit requiring higher levels of stimulation, but with no brain involvement at all.

2.2. Conditioning

Learning can also impact pain, and again, these effects were assumed to require a brain. Clearly, this is the case in many situations. For example, when an auditory or visual cue is paired with a moderately aversive shock, the cue acquires the capacity to elicit a conditioned antinociception on the tail-flick test (Famselaw, 1986). This effect reflects a form of Pavlovian conditioning, wherein the auditory or visual cue
serves as the conditioned stimulus (CS) and the moderate shock acts as the unconditioned stimulus (US).

When a visual or auditory cue is employed, the brain must play a role. But what if a cutaneous cue was used instead? Under these conditions, perhaps lower level mechanisms within the spinal cord could support a simple form of conditioned antinociception. Grau and colleagues explored this possibility using mild shock to a rear leg as a CS and a strong shock to the tail as the US in spinaly transected rats. The CS was applied either to the rats' left or right hind legs in one of two conditions: paired with a strong shock to the tail (CS+), or unpaired (CS−). During the conditioning phase, rats received either the CS+ or the CS− 30 times. One hour later, tail-flick latency was tested during reexposure to the CS. Shocks delivered to the same leg previously used for the CS+ produced longer tail flick latencies (i.e., an antinociceptive effect) than shocks to the CS− side, the same outcome observed in intact rats. Evidently, even the spinal cord is sensitive to CS−US relations (for a review of this literature see Patterson, 2001). Furthermore, as predicted by standard learning theory, the CS+/CS− difference extinguished across test trials. Conditioned antinociception appears not to require the involvement of the brain.

**Latent Inhibition and Overshadowing**

Given evidence that spinal neurons are sensitive to a CS−US relation, Paul Illich (Illich, Salinas, and Grau, 1994) explored whether this system could support some more complex Pavlovian phenomena, such as latent inhibition and overshadowing. Latent inhibition is the phenomenon whereby preexposure to a CS reduces its associability with the US. Intuitively, it is as if the organism has ceased attending to the CS because of the preexposure (habituation) and therefore fails to notice that the preexposed cue predicts the US. Overshadowing is observed when a compound CS, formed from two cues that differ in noticeability (salience), is paired with the US. Even though the less salient cue can support conditioning when it is presented alone, subjects typically fail to learn about it when it is presented in compound with a more salient cue.

To test for latent inhibition in the spinal cord, two groups of spinally transected rats were preexposed to the CS alone prior to conditioning. For one group (CS+ preexposed), the preexposed cue was subsequently paired with the tail shock US. For the other group (CS− preexposed), the preexposed cue was presented in an unpaired fashion with the US during conditioning. In both cases, stimulation to the left or right hind leg served as the CS (counterbalanced across groups). A third group (no preexposure) remained untreated during the preexposure phase. As expected, rats that received no stimulation during the preexposure phase exhibited longer tail-flick latencies during the CS+ (i.e., they predictably showed conditioned antinociception). Preexposure of the CS− had no effect (i.e., conditioned antinociception was observed when subjects were conditioned using stimulation to the opposite leg). But rats that experienced the CS+ alone prior to training exhibited little conditioned antinociception after the training regime was applied using
the same side as the preexposure—that is, the stimulus preexposure produced latent inhibition.

To test for overshadowing, Illich and colleagues manipulated stimulus salience by having two different intensities of CS. One group of spinally transected rats was conditioned using the less intense CS (B) paired with the US, and, as expected, they showed conditioned antinociception in the form of longer tail-flick latencies compared with controls. Another group of spinally transected rats were given B in conjunction with the more intense CS (A). When these animals were subsequently tested with B alone, their tail-flick responses were not significantly different from controls. An additional control showed that the intense cue only disrupted learning about the less salient cue when the stimuli were presented in compound. These results suggested that spinal systems support overshadowing as well as latent inhibition.

The discovery of analogs to latent inhibition and overshadowing in the spinal cord was astonishing, for when brain mechanisms are involved, these phenomena are often accounted for in terms of attentional mechanisms. If one was dealing with an intact organism, one might be tempted to say that the rat's attention was directed away from the less salient cue and toward the more salient one when both are present, so that only the attended association with the US was learned.

We need to acknowledge that whatever mechanisms are operating in the spinal cord, they don't have all the functional capacities associated with Pavlovian conditioning in intact animals. Indeed, detailed analyses of the underlying mechanisms suggest that it may abide by simpler rules, and we assume that it is unable to support some complex phenomena. For instance, the capacity of the hippocampus to learn about temporal and spatial ordering greatly increases the range of relationships among stimuli beyond those that can be learned by the spinal cord alone. Likewise, in intact animals latent inhibition is sometimes characterized as involving a context-CS association (Wagner, 1981), but there is currently no evidence that spinal neurons can learn about contextual cues (although this has not been thoroughly investigated). The observed inhibition in the spinal cord is probably due to a simple nonassociative, single-stimulus habituation-like effect (the simplest version of latent inhibition). Despite these differences, Grau and Joyner (2005a, 2005b) have argued that results from the spinal cord suggest that it is important to keep a clear conceptual distinction between the learning phenomena associated with an experimental method, namely, Pavlovian conditioning, and the variety of neuro-functional mechanisms that can encode stimulus-stimulus relations in multiple ways. A similar point holds for instrumental (response-outcome) conditioning.

3. Instrumental Conditioning

If the spinal cord exhibits Pavlovian conditioning, what about instrumental learning? In instrumental conditioning, delivery of a reinforcer (positive or negative) is
made contingent on the organism's behavior, subsequently altering the probability of that behavior. Whereas in Pavlovian conditioning the key relation is between two stimulus events (the CS and US), in an instrumental paradigm learning depends on the relationship between a response (R) and an outcome (O). Most assume that this form of learning requires a brain, but here, too, recent data suggest otherwise.

To separate the role of the animal's own behavior in conditioning from the contribution of the reinforcing stimuli alone, it is necessary to use a yoked design, where one group of animals (the master group) is reinforced contingently on their own actions, and the yoked group receives the reinforcement on exactly the same schedule. The subsequent change in behavior in these two groups is compared to a third group of controls who receive no reinforcement at all. If outcome for the master group is significantly different from both the yoked and unreinforced groups, then it is reasonable to attribute the difference to the instrumental relationship between the animal's own behavior and the reinforcement.

Others (Buerger and Chopin, 1976) had previously explored whether spinal neurons are sensitive to response–outcome (R–O) relations, but this work had been dismissed on methodological grounds (Church and Lerner, 1976). Grau, Barstow and Joyner (1998) developed a set of procedures that overcame the limitations of past studies to provide evidence that spinal neurons are capable of a simple form of instrumental learning. In their paradigm, spinalically transected rats are placed in an apparatus that allows both hind legs to hang free. Master rats are given a shock to one hind leg whenever that leg is in an extended position. Subjects in a second group are experimentally coupled (yoked) to the master subjects and receive shock at the same time as their master partner, independent of leg position. Master rats soon learn to keep the shocked leg lifted, effectively minimizing net shock exposure. This learning is not observed in the yoked subjects. To discount alternative interpretations of these results, Grau tested the subjects under common conditions with controllable leg shock. Previously trained rats (master) quickly reacquired the task, exhibiting positive transfer relative to a control group that had never been trained. Surprisingly, yoked subjects that previously received uncontrollable shock failed to learn, and this was true independent of whether they were tested on the same or opposite leg (for a recent review, see Grau et al., 2006).

Further work has shown that uncontrollable nociceptive stimulation has a lasting (>24 hours) inhibitory effect on spinal learning and impacts recovery after a spinal contusion injury. In the latter study, the spinal cord was bruised, rather than cut, using a device that emulates a human spinal injury. This produces a nearly complete paralysis that wanes over the course of a few weeks as subjects regain some hind limb function. Many spinal cord injuries in humans are accompanied by tissue damage that can provide a source of uncontrolled nociceptive input. Using an animal model, Grau et al. (2004) showed that uncontrollable nociceptive stimulation after injury impairs recovery. Importantly, nociceptive input has no adverse effect on recovery when it is controlled by the subject. These results imply that independent of philosophical debates, understanding how spinal cord neurons process signals could have important clinical implications. Indeed, the emergence of
techniques to span a spinal injury with neural bridges has brought to the fore a potentially greater challenge—encouraging the new neurons to select the appropriate pattern of synaptic connectivity. Selecting appropriate connections is a process that will depend on a form of dynamic tuning that is shaped through the process of learning. Rewiring the spinal cord will depend on its capacity for learning.

In intact animals, it is well established that uncontrollable stimulation can induce a lasting impairment in learning and performance, a phenomenon known as learned helplessness. A key variable in this literature is the perception of control—subjects that receive the same amount of aversive stimulation, but can control its presentation, generally exhibit far fewer ill effects (Peterson, Maier, and Seligman, 1993). Moreover, a history of controllable stimulation can “immunize” subjects against the adverse effect of uncontrollable shock. Conversely, the presumed cognition of no control induced by uncontrollable shock can be reversed by exposing rats to controllable stimulation (“therapy”). Is the same true for spinal mechanisms? Eric Crown ran the analogous experiments in spinally transected rats and showed, as before, that uncontrollable shock disrupted subsequent learning (Crown and Grau, 2001). Subjects that had previously received controllable shock did not exhibit a learning deficit after uncontrollable shock. Conversely, coupling behavioral training with a pharmacological manipulation that fostered learning had a therapeutic effect that reversed the learning deficit. Once again, the overall pattern bears a remarkable similarity to the results obtained in studies of learned helplessness.

As with the Pavlovian conditioning, some caution is warranted. The learning capacities of the spinal cord are much more restricted than those of intact animals. In examining instrumental learning within the spinal cord, there are several criteria that must be met (adapted from Grau, Barstow, and Joynes, 1998):

A. **Minimum Criteria (Instrumental)**

1. Instituting a relationship between the response and an outcome produces a change in behavior (performance).
2. The effect is neurally mediated.
3. The modification outlasts (extends beyond) the environmental contingencies used to induce it.
4. The behavioral modification depends on the temporal relationship between the response and the outcome.

B. **Advanced Criteria (Operant)**

5. The nature of the behavioral change is not constrained (e.g., either an increase or decrease in the response can be established).
6. The nature of the reinforcer is (relatively) unconstrained (a variety of outcomes can be used to produce the behavioral effect).

The experiments performed by Grau and colleagues (Grau, Barstow, and Joynes, 1998; Grau, et al., 2006) have addressed the first four criteria, providing solid evidence that
spinal neurons are sensitive to R–O relationships. At the same time, it’s clear that many examples of operant behavior in intact animals exhibit a range of flexibility that seems well outside of what the spinal cord might accomplish. Two key differences are the degrees to which the response and available reinforcers are much more constrained in the spinal cord (a failure to meet criteria 5 and 6). A third factor may involve the ease with which behavior can be inhibited by circuits in the brain. Intact rats can exhibit a range of behavior, can be trained with a variety of outcomes (though, here too, biological limits impose constraints), and can inhibit their actions—capacities that seem well outside the limits of spinal learning. Still, even though full operant conditioning appears to require mediation by circuits in the brain, Wolpaw and Lee (1989) showed that learned operant responses in monkeys were preserved after spinal transection, locating the operant memory itself in the spinal cord.

4. Philosophical Implications

More than three and half centuries ago, Descartes argued that animals are automata: reflex-driven machines with no intellect or other cognitive capacities. A little more than two centuries later, T. H. Huxley traced the philosophical development of the idea that animals are automata, giving special attention to spinal cord reflexes (Huxley, 1874). Huxley reported a series of experiments on a frog, which showed that much of its reflexive behavior was preserved even when its spinal cord had been severed, or large portions of its brain removed. He argued that equivalence between the behavior of an intact frog and a frog with its brain removed implied that consciousness was superfluous to the explanation of either.

We have reviewed research showing that spinal neurons belonging to the nociceptive system are sensitive to both Pavlovian and instrumental relations, and they exhibit a number of phenomena that when studied in normal, intact organisms, including human beings, are frequently described in cognitive or attentional terms. These phenomena include a distractor effect, latent inhibition and overshadowing, and learned helplessness effects. Thus, like Huxley, we have suggested a kind of equivalence between spinal mechanisms and cerebral mechanisms. Rather conveniently for his thesis, Huxley ignored the fact that a brain-damaged frog is much less reactive to its environment than it was before (Crowley and Allen, 2008). We have indicated ways we think spinal mechanisms are much more restricted in their capacities than brain mechanisms. Nevertheless, it is clear that any view that treats learning and memory as brain-bound processes must confront these surprising findings about the adaptive capabilities of the rat spinal cord.

The exact mechanisms of spinal learning remain controversial, but the existence of spinal learning should no longer be. Grau et al. (2006) argue that the behavioral evidence for spinal learning was adequate even though some neuroscientists were reluctant to admit it before a role for NMDA (N-methyl-D-aspartic acid) receptors
and LTP (long-term potentiation) in spinal neurons was demonstrated, thereby establishing a parallel to learning in the hippocampus (for further discussion of the role of the NMDA receptor in spinal learning, see Grau et al., 2006). The attitudes of the skeptical neuroscientists, disbelieving of spinal learning until shown evidence for the engagement of specific molecular mechanisms, seems in line with Bickle’s (2003) “ruthless reductionism” (exemplified by his view that memory consolidation has been reduced to the neuromolecular mechanisms of LTP). But much remains to be discovered about spinal learning, and the exact nature of the link between the behavioral results and mechanisms of synaptic plasticity is a long way from being established. It is also worth bearing in mind that newly emerging dynamical systems approaches to neural networks can provide models for associative learning without synaptic plasticity at all (Phattanasri, Chiel, and Beer, 2007). Whatever the outcome of this program of research into spinal cord learning, we do not believe it is necessary to take a strong stand on the question of whether all learning mechanisms will turn out to be constructed from the same molecular or neural components, although we would not be terribly surprised to find that the basic mechanisms are highly conserved by evolution (see also Bickle, 2003, chapter 3).

One foundational question raised by these findings, then, is “What is learning?” Is it a behavioral type or a neurological type? Traditionally, learning theorists (coming from the behaviorist tradition) have characterized it in terms of the procedures they use to study it, for example Pavlovian conditioning or instrumental conditioning—an approach which has the unfortunate consequence of making discovery of the underlying mechanisms seem relatively unimportant. An alternative perspective suggests that these apparently different forms of learning may instead reflect the deployment of similar neural mechanisms in the service of different adaptive functions. Thus, Pavlovian conditioning in the spinal cord seems tightly coupled to regulating nociception, a task where sensitivity to stimulus–stimulus relationships is important, whereas instrumental learning is functionally related to the central pattern generator involved in locomotor activities such as stepping (Edgerton, Roy, de Leon, Tillakaratne, and Hodgson, 1997; Grau et al., 1990) where the task is to adapt behavior to sensory feedback. These distributed learning systems may be organized in a lattice hierarchy that organizes and regulates behavior (Gallistel, 1980), with higher circuits being capable of associating more abstract relationships within the lattice. The project of mapping the details of the relationships among the distributed learning components will require attention to both their functional capacities and the neurobiological mechanisms, hence the neurofunctionalism advocated by Grau and Joynes (2005a, 2005b). From this perspective, although some aspects of learning and memory may be ruthlessly reduced to specific neuromolecular events in synapses, the degree of abstraction at which such reductions occur will not entirely suit the needs of behavioral scientists who want to understand how brain and spinal systems interact to produce adaptive behavior. Learning is neither solely a behavioral type nor solely a neural type.

Are spinal circuits beneath the lower bounds of cognition in this lattice? We don’t know where to draw the line, or even whether it is worthwhile to try to do so. What we find interesting and challenging about these results is the recognition that
neurons, wherever they are found, are capable of adaptively responding to relationships among their inputs. When those relationships are essentially uncorrelated, as in the yoked animals in the instrumental learning experiment already described, the effect on the neural mechanisms is to make future learning more difficult. Conversely, the master animals that had already been trained on one task were subsequently capable of learning a much more demanding task than the unyoked and untrained controls (Crown, Ferguson, Joynes, and Grau, 2002). Thus, the capacity to learn is itself a function of past experience, even in the spinal cord. Furthermore, under normal developmental conditions, spinal cord mechanisms are coupled to brain mechanisms with influence running in both directions. It seems likely that a full understanding of the cognitive and learning capacities of intact organisms will require significant attention to how these neural systems develop and interact.

At the high end of the spectrum, the discovery of spinal learning mechanisms also suggests a reassessment of philosophical arguments about the functional role of conscious experience in working memory for intact organisms (see Allen, 2004, for discussion). For example, it has often been suggested that the intrusion of conscious experience of pain into working memory serves as a signal that facilitates learning about how to behave when confronted with actual or possible tissue damage. But the finding that instrumental conditioning can take place without the involvement of the brain, and arguably therefore without consciousness, shows a need for more specificity about the exact role that brain-based systems associated with consciousness may play in learning about how to avoid noxious stimuli (Allen, Fuchs, Shriver, and Wilson, 2005). Bickle (2003, p. 163) lists learning and memory among the categories of behavior having “an obvious link with consciousness.” Given the results we have described from the Grau laboratory, the obviousness of that link can no longer be simply assumed.

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