NOTES

CHAPTER ONE THE BIG ONE

1. Although "the self" and "personality" are not major topics in the field of neuroscience, some brain scientists and psychologists have discussed the relation of self and personality to the brain. Most discussions of the self and the brain have focused on conscious aspects of the self. My approach, in contrast, gives as much if not more weight to unconscious or implicit aspects. The attempts of others to relate personality to the brain have mostly treated personality as a set of fairly static traits. I'm attempting to construct a way of thinking about personality as a set of brain processes that are in constant flux due to their capacity to learn and remember. The relation of the terms *self* and *personality* is considered in chapter 2. For discussions of self and the brain by others see: Popper and Eccles 1977; Gazzaniga 1985; Gazzaniga 1998; Stuss 1991; Brothers 1997; Arbib 1999; Llinas 2001; Damasio 1999; Feinberg 2000; for personality (or temperament) and the brain see: Gray 1991; Schore 1994; Davidson 1992; Kagan 1994; Kagan 1998; Zuckerman 1991.
2. The two other major possible alternatives to the synaptic view are that the self is mediated by intrinsic properties of individual neurons (rather than connections between them) or that it is mediated by large aggregates of neurons that act globally as a field or Gestalt (rather than by particular connections between specific ones). The aggregate field theory has generally not been given much credence in recent years, in part due to experiments that argue against it (Sperry and Miner 1955) and in part due to the fact that the synaptic approach has been successful. In contrast, the notion that intrinsic properties of neurons are important is indisputable (Llinas 1988; Llinas 2001). However, in order for intrinsic properties of any cell to be expressed in psychological functions of the brain it is necessary for that cell to interact with others by way of synapses. This is discussed further at the end of chapter 3.
3. Pinker 1994; Pinker 1997; Dawkins 1996; Wilson 1999.
4. Tellegen et al. 1988.
5. Kagan 1999; Kagan 1998.
6. Pinker 1997; Harris 1998; Gazzaniga 1992.
7. Harris 1998. Also see the Nurture Assumption Website (*http://home.att.net/~xchar/tna/*). For rebuttals to Harris, see: Gardner 1998; Kagan 1999; LeDoux 1998.
8. O'Connor et al. 2000; O'Connor and Rutter 2000.
9. Blanchard and Blanchard 1972.
10. This does not mean that the rat's innate fear of cats is programmed solely by genes in the absence of any environmental influence. Fear circuits, like other circuits, get

wired by a combination of genetic programming of synaptic connections and various environmental influences. As a result, although the ability to respond to the cat doesn't require experience with the cat, it may require other kinds of experience to get the amygdala properly wired. Unfortunately, relatively little is known about amygdala development.

11. The actual sequence of events was startle and then freeze.
12. Explanations like this are the business of evolutionary psychology (Tooby and Cosmides 2000). See chapter 4 for a critique of this field.
13. Bolles and Fanselow 1980; Blanchard and Blanchard 1972.
14. Blanchard and Blanchard 1972; LeDoux 1996.
15. LeDoux 1996.
16. The bodily responses that occur during fear reactions include the so-called flight-fight responses. Actually, a better term might be the freeze-flight-fight response, since freezing often occurs first. Supporting physiological changes include a redistribution of blood away from the skin and gut and toward the brain and muscles, since the latter will need energy during the upcoming fight or flight. These changes in blood flow account for the alterations in blood pressure and heart rate that occur, as well as for the alterations in skin temperature. Hormones are also released from various organs that support these processes. For a more extensive discussion, see LeDoux 1987.
17. Rushdie 1990.
18. Sperry 1966; Sperry 1984; Gazzaniga 1970; Popper and Eccles 1977; Gazzaniga and LeDoux 1978; Gazzaniga 1988; Szentagothai 1984; Gazzaniga 1985; Gazzaniga 1992; Crick and Koch 1990; Stoerig 1996; Penrose 1989; Singer 1998; Edelman and Tononi 2000; Edelman 1993; Crick 1995; Damasio 1999; Llinas 2001; Zeki and Bartels 1999.
19. Horgan 1996.
20. By this statement, I'm not denying the existence of consciousness in other animals but only saying that the unique kind of consciousness we have is probably not present in other animals, owing to the fact that our brain is different from most others in terms of its size (relative to body weight) and complexity (especially in the frontal neocortex). I'll have more to say about these issues later, especially in chapters 7 and 8.
21. As pointed out in the previous note, I'm not denying that other animals have some kind of conscious awareness, and instead am only saying they don't have the kind of conscious awareness that comes from having a human brain. In particular, their capacity for self-reflectance is probably missing. They are not strictly speaking unconscious in the sense of being asleep or knocked out. They are instead unconscious in the sense of not being self-aware in the way humans are. I will avoid saying which animals are conscious and which are not and instead emphasize that only humans are conscious in the way humans are conscious. Animal consciousness is discussed in some detail in chapters 7 and 8.
22. Bargh 1990; Bargh and Barndollar 1996; Bargh and Chartrand 1999; Greenwald and Banaji 1995; Bowers and Meichenbaum 1984; Greenwald 1992; Jacoby and Woloshyn 1989; Kihlstrom 1987; Kihlstrom 1990; Meichenbaum and Gilmore 1984; Merikle 1992; Öhman and Soares 1994; Öhman 2000; Rozin 1976; Shevrin et al. 1992; Nisbett and Wilson 1977; Erdelyi 1985; Wilson et al. 2000; Wilson (in press).
23. Rozin 1976; Shevrin and Dickman 1980; Kihlstrom 1987; Kihlstrom 1990.

24. Popper and Eccles 1977; Stuss 1991; Sperry 1984; Gazzaniga 1985; Brothers 1997; Arbib 1999; Llinas 2001; Damasio 1999; Feinberg 2000.
25. Damasio 1999; Gazzaniga 1998.

CHAPTER TWO SEEKING THE SELF

1. James 1890.
2. Hall et al. 1998; Mischel 1993.
3. Title of a song by the saxophonist King Curtis.
4. The conference was sponsored by the Vatican Observatory and the Center for Theology and Natural Sciences. The proceedings were published, Russell et al. 2000.
5. The theological problem raised by this discussion, of course, is one of figuring out when God acts and when He doesn't.
6. Christian 1977.
7. Christian 1977.
8. Flew 1964.
9. Quoted in Walter 1953.
10. I'm grateful to Stephen Happell and Nancey Murphy for their helpful suggestions on the content of this paragraph. They were participants at the Vatican conference in Poland.
11. For a summary of how Descartes's views came to be so influential, see Rorty 1979.
12. Bremmer 1993; Snell 1960.
13. Flew 1972.
14. Plato, cited in Flew 1964.
15. Flew 1964; Flew 1972.
16. Happel 2000.
17. For contemporary discussions of the mind-body possibilities, see: McGinn 2000; Humphrey 1992, 2000; Metzinger 1995; Searle 1992, 2000; Dennett 1991; Churchland 1984; Block 1995; Chalmers 1996; Clark 1998. For a Website with a bibliography on the mind-body problem, see: *http://www.u.arizona.edu/~chalmers/biblio.html*.
18. Chalmers 1996.
19. Although I believe that my mind (and yours) is the product of a physical system, I don't outright reject other ways of thinking about the mind. Reductionism is a good approach to brain research, but isn't necessarily a good principle for guiding us through daily life, say, when we are wooing a partner, raising children, climbing, or descending, the corporate ladder, or hiring a plumber. These activities, of course, all depend on and are even potentially explainable in terms of brain mechanisms, but when scientists or lay persons do these things, they don't necessarily need to know about the neurobiological underpinnings involved. Of course, facts about how the brain operates can work their way into everyday activities (people freely take drugs to control anxiety or depression, eliminate aches and pains, or to manage epilepsy or Parkinson's disease). But there's nothing special about brain research in this regard, as our culture is constantly changing on the basis of developments in the humanities as well as the sciences. Literature, for example, offers ideas that are often useful to people in their lives, and may even be helpful in understanding how the mind, through the

brain, makes us who we are. Dostoevsky, for one, had lots of interesting ideas about the importance of unconscious processes in mental life. Nonscientific approaches (literature, poetry, psychoanalysis) and nonreductionist sciences (linguistics, sociology, anthropology) can, I believe, coexist with and complement neuroscience. For example, new facts about how the brain works may help anthropologists understand human evolution, and new discoveries in anthropology or other social sciences might lead neuroscientists toward novel experiments on the mind. In a similar vein, as I said above, a spiritual view of persons doesn't have to be mutually exclusive with a neural view. Though I'm not particularly religious, I know scientists who are, and even some with a mystical side. Reduction is often treated disparagingly by those outside science. This is partly because people like to think of themselves in terms of their own self-awareness, and they don't like the idea that the self might exist at some level other than at the level of conscious awareness. Reduction also has a bad name because carried to its logical extreme, it would require that we, for example, describe poetry in terms of subatomic particles. This is the so-called absurd kind of reduction that we have to avoid. But I'm looking for nonabsurd reductions, reductions that make sense, and I believe it is reasonable to begin to think of the self in terms of synapses.

20. Philosophers can and have helped in the area of mind and brain by analyzing the mental in ways that can be pursued by brain researchers. Jerry Fodor's philosophical analysis of what constitutes a mental module, a self-contained mental system, has been very useful in stimulating research and discussion in neuroscience (Fodor 1983). There have been proponents (Tooby and Cosmides 2000; Gazzaniga 1992) and detractors (Elman et al. 1997; Fuster 2000). Ned Block's view that the reason it is so hard to think about brain and consciousness is that different kinds of consciousness are often confused and mixed together is also helpful (Block 1995). His analysis gives rise to a distinction between phenomenal and access consciousness, with one being about subjective experience and the other about control processes that regulate mental and behavioral states. Although subjective experiences are difficult to investigate scientifically, control processes, like attention, are amenable to experimental study. Regardless of whether Block's distinction is ultimately right, it helps researchers think in concrete terms about which aspects of consciousness are most profitably pursued in the brain, given current understanding and research tools. Also important to keep in mind is a distinction made by John Searle and others between the search for the neural correlates of consciousness and the search for the mechanisms of consciousness (Searle 2000). That is, many brain events may occur during a conscious experience, but not all of these will be related to the generation of that experience. Pat Churchland has written philosophy for neuroscientists on several occasions, sometimes in collaboration with neuroscientist Terry Sejnowski (Churchland 1986; Churchland and Sejnowski 1992). And Nick Humphrey, a neuroscientist turned philosopher, has made the interesting point that maybe clever thinking about the way the brain works, not just about how the mind works, may be a key to progress (Humphrey 2000).
21. Referenced in Dennett 1976.
22. Strawson 1959.
23. Both quotes appeared in Strawson 1959.
24. Dennett 1976.

25. Rawls is quoted in Dennett 1976.
26. Nagel is quoted in Dennett 1976.
27. Gallagher 2000; Sorabji 2001.
28. Gallagher 2000.
29. Dennett 1991; Dennett 1988; Neisser and Fivush 1994.
30. Gallagher 2000.
31. Foucault 1978; Gergen 1990; Butler 1990; Lutz 1988; for review, see Strauss and Quinn 1997.
32. Social constructivists emphasize the relativistic nature of reality and assume that there is no one underlying reality waiting to be discovered by scientists. Some even reject the notion that people exist as psychological beings, and thus argue for an elimination of psychology. For a sampling of writings about this topic, see: Gross et al. 1996; Martin and Sugarman 2000; Gergen 1997; Sass 1992.
33. Kolm 1985.
34. James 1890; Elster 1985; Neisser 1988.
35. Gallagher 1996; Rochat 1995; Damasio 1999; Bermudez 1996.
36. Neisser 1988.
37. Nagle 1974.
38. Others might disagree. For example, Leslie Brothers, a neuroscientist turned philosopher, has embraced Strawson's idea that persons are defined by their conscious states and has combined it with the social theories of George Herbert Mead and Rom Harré, and added a bit of evolutionary psychology to it (Brothers 1997). Like Strawson, Brothers says that a person is a "being with a mental life, an 'owner' of conscious subjective experience." Following Mead and Harré, she proposes that "Self consciousness arises in the process of social experience" and *"Only brains in a social context can generate the kind of consciousness that includes 'I.'"* In the tradition of evolutionary psychology, she notes, "Human beings are biologically prepared to subscribe to the concept of a person just as we are biologically prepared to learn a language." For reasons that should be obvious by now, I disagree with Brothers's idea that consciousness is the main key to persons, but more about that later. On the other hand, I agree with Brothers that it is important to understand the role of brain mechanisms in social interactions, but I'd like to try to reach the social level by climbing up to it from the neurobiology of specific brain networks rather than start at the social level and try to find brain correlates. I also agree with the idea that we should look, to the extent possible, for evolutionary mechanisms, though I'm less enthusiastic about evolutionary psychology (as opposed to evolutionary biology) than she is. That is, I believe the brain rather than the mind per se evolves.
39. See the discussion of Brothers, Harré, and Mead in the previous note.
40. Boring 1950; Gardner 1987.
41. Boring 1950; Gardner 1987.
42. Watson 1925.
43. Ryle 1949.
44. Gardner 1987.
45. Bruner et al. 1956.
46. Miller 1956.

47. Gardner 1987.
48. See Shevrin and Dickman 1980; Kihlstrom 1987; Erdelyi 1985; LeDoux 1996; Wilson et al. 2000; Wilson (in press); Bargh 1990; Bargh and Chartrand 1999; Greenwald and Banaji 1995; Zajonc 1984; Loftus and Klinger 1992; Bowers 1984; Bowers and Meichenbaum 1984; Öhman 2000; Debner and Jacoby 1994.
49. Gardner 1987.
50. Gazzaniga 1995.
51. LeDoux 1984; LeDoux 1996; Zajonc 1984; Ekman and Davidson 1994.
52. Hilgard 1980.
53. Hall et al. 1998; Boring 1950.
54. Hall et al. 1998.
55. Kagan 1994; Hall et al. 1998.
56. Freud 1915.
57. Hall et al. 1998.
58. Hall et al. 1998.
59. *Personality* is usually a broader term since *self* usually means "conscious self." But in my scheme, *self* is the broader term since only people are persons but all organisms have selves.
60. Rogers is quoted in Hall et al. 1998, p. 463.
61. Markus and Kitayama 1991.
62. Munroe 1955.
63. Bargh 1990; Greenwald and Banaji 1995; Bargh and Chartrand 1999; Wilson et al. 2000; Wilson (in press).
64. See Squire et al. 1993; Schacter 1987; Cohen and Eichenbaum 1992.
65. The effects of emotion and stress on memory are described in some detail in LeDoux 1996 and will also be discussed in later chapters.
66. Popular trait theories are those of Raymond Cattell and Hans Eysenck. See chapters 7 and 8 of Hall et al. 1998 for a summary.
67. Tellegen et al. 1988.
68. See Zuckerman 1991; Gray 1982; Gray 1991; Kagan 1994; Kagan 1992; Kagan 1998; Eysenck and Eysenck 1985; Davidson 1992.
69. Schwartz et al. 1999.
70. Kagan 1994; Kagan 1992; Kagan 1998.
71. Mischel 1993; Mischel 1990.
72. Carlson 1993; Zuckerman 1991.
73. Interview with Bob Dylan, *Newsweek,* October 13, 1997.
74. Roth 1986.
75. Epstein 1995.
76. James 1890.
77. Virginia Woolf, *Orlando,* chapter 6.
78. Klee 1957.
79. The main exception is biological trait theory, especially Eysenck's trait theory. It has been proposed that the trait called neuroticism is related to overactivity in the brain's fear/anxiety system, whereas extraversion is due to overactivity in the pleasure or reward system (see Gray 1982, 1991; Zuckerman 1991). For a critique of trait theory, see Mischel 1993.

80. For other views of the self and the brain, see the list of citations below. For the most part, these have tended to emphasize the conscious aspects of the self. My view, in contrast, includes unconscious as well as conscious aspects. Others that include a role for unconscious aspects of the self include Antonio Damasio (Damasio 1999) and Michael Gazzaniga (Gazzaniga 1985, 1992, 1998; Gazzaniga is working on a book, *The Last to Know,* which emphasizes unconscious processing in the construction of consciousness). For ideas about the brain and the conscious self, see: Popper and Eccles 1977; Stuss 1991; Sperry 1984; Gazzaniga 1985, 1992, 1998; Brothers 1997; Arbib 1999; Llinas 2000; Damasio 2000; Feinberg 2000.

CHAPTER THREE THE MOST UNACCOUNTABLE OF MACHINERY

1. For a summary, see LeDoux 1987.
2. The divisions of the forebrain include: the thalamus, hypothalamus, basal ganglia, limbic system, old cortex, and neocortex.
3. Ariëns Kappers 1909; Papez 1937; MacLean 1949; MacLean 1952; Nauta and Karten 1970.
4. Nauta and Karten 1970; Northcutt and Kaas 1995; Karten and Shimizu 1991.
5. Lettvin et al. 1959; Camhi 1984.
6. Camhi 1984; Suga 1990; Gould 1982.
7. For an alternative view that emphasizes evolutionary pressures on the whole brain rather than on specific systems, see Finlay and Darlington 1995.
8. Killackey 1990; Preuss 1995.
9. Brodmann 1909; Economo and Koskinas 1925; Campbell 1905.
10. Gazzaniga et al. 1996; Feinberg and Farah 1998; Ramachandran and Blakeslee 1998.
11. Nonneuronal cells communicate with each other but not the way neurons do. The electrochemical process of synaptic transmission is unique to nervous tissue.
12. Cell theory discussion based on Shepherd 1998, Jacobson 1993, and Microsoft Encarta 2000.
13. Based on chapter 3 in Shepherd 1988.
14. Shepherd 1988, p. 41.
15. Jones 1961, p. 32.
16. Jones 1961, p. 34.
17. Freud 1887–1902.
18. Jones 1961, Freud's biographer, says that though Freud dropped the anatomical terms, the principles that guided his psychological theories were underneath it all based on his early training in anatomy and physiology.
19. Sherrington 1897.
20. For a summary of Sherrington's early work on reflexes, see Sherrington 1906.
21. Shepherd 1988, p. 65.
22. Shepherd 1988, p. 42.
23. Rozental et al. 2000.
24. Kuffler and Nicholls 1976.
25. Zigmond et al. 1999; Kandel et al. 2000.
26. Chen et al. 2000.
27. Muscles don't have dendrites, but have their own special kind of receptive area that is contacted by the axon terminal.

28. Based on Winson 1985.
29. Boring 1950.
30. Boring 1950.
31. Gregory 1981.
32. Shepherd 1988.
33. Shepherd 1988.
34. Shepherd 1988.
35. From Jacobson 1993.
36. The space between neurons is filled with fluids that are in essence part of a vast continuous sea of liquid in which all the neurons of the nervous system are bathed. This sea is made up of so-called cerebrospinal fluid, and it occupies the so-called extracellular space.
37. Based on Kuffler and Nicholls 1976.
38. In fact, the postsynaptic cell has to receive convergent inputs within a matter of milliseconds, otherwise the inputs will not sum together and will not produce an action potential. Since the inputs are added up in the cell body, they can arrive from many different dendrites, as long as they produce electrical responses that reach the cell body at about the same time.
39. Electrical transmission is made possible by the existence of special contacts between cells called gap junctions (Rozental et al. 2000). These are actually physical contacts and are exceptions to the notion promoted by the neuron theory that cells are physically separate. These turn out to be important in synchronizing hippocampal GABA cells (Fukuda and Kosaka 2000).
40. Based on Bloom and Laserson 1985.
41. Cooper et al. 1978.
42. GABA cells sometimes have long axons and communicate between brain regions, but mostly they have short axons that end on nearby cells.
43. But even the time-course distinction between fast transmitters and modulators can be blurred. Most transmitters work with a variety of receptors. GABA, for example, has A and B receptors. While the A receptor mediates the fast effects we've been talking about, when GABA binds to B receptors its action is slower and more prolonged. Glutamate, too, has some late, longer-lasting effects when it binds to some of its receptors. Another fast transmitter is acetylcholine. When it binds to its nicotinic receptor, it does its fast transmitter thing, but when it binds to its muscarinic receptor, it works slowly. So it is often best to think of transmitters and receptors together when drawing conclusions about the kind of transmission involved.
44. Shepherd 1998.
45. See note 43 above.
46. The main exception involves the cholinergic neurons of the basal forebrain, which complement the brain stem cholinergic systems.
47. This will be discussed in chapter 10.
48. Shepherd 1998; Cooper et al. 1978.
49. Selkoe and Kosik 1983.
50. Babic 1999; Yamada et al. 1999.
51. This will be discussed in detail in later chapters, especially chapters 8 and 10.

52. Stutzmann et al. 1998; Stutzmann and LeDoux 1999.
53. Gibbs 2000; Dell and Stewart 2000.
54. See note 39.
55. Quirk et al. 1995; Rolls 1999; Ono and Nishijo 1992; Collins and Pare 2000; Maren 2000.
56. Breiter et al. 1996; Morris et al. 1996; Morris et al. 1998; Whalen et al. 1998; LaBar et al. 1998.
57. Li et al..1996; Lang and Pare 1997; Collins and Pare 1999.
58. Chapman et al. 1990; Weisskopf and LeDoux 1999.
59. Quirk et al. 1995; Collins and Pare 2000; Maren 2000.
60. Woodson et al. 2000; Szinyei et al. 2000; Smith et al. 2000.
61. Li et al. 1996; Collins and Pare 1999.
62. Stutzmann et al. 1998; Stutzmann and LeDoux 1999.
63. McEwen and Sapolsky 1995.
64. Stutzmann et al. 1998.
65. Bogerts et al. 1993; Convit et al. 1995; de Leon et al. 1988; Fukuzako et al. 1996; Sheline et al. 1996; Starkman et al. 1992; Yehuda et al. 2000; Coplan et al. 1998; Young et al. 1994.
66. Corodimas et al. 1994; Conrad et al. 1999; Makino et al. 1994; Shors et al. 1992.
67. Llinas 1988.

CHAPTER FOUR BUILDING THE BRAIN

1. This section on early development is based on Purves et al. 1996.
2. Nottebohm 1989; Gould et al. 1997; Gould et al. 1999; Fuchs and Gould 2000.
3. Rodier 2000.
4. Chan and Jan 1999; Reichert and Simeone 1999.
5. Schlaggar and O'Leary 1991.
6. Rakic 1995.
7. Schlaggar and O'Leary 1991; Shatz 1992; Rakic 1992.
8. Miyashita-Lin et al. 1999.
9. Based on Raper and Tessier-Lavigne 1998.
10. Terman and Kolodkin 1999.
11. Edelman 1987; Changeux and Danchin 1976.
12. Jerne 1967; see also Gazzaniga 1992.
13. Changeux and Dehaene 1989.
14. Edelman 1987.
15. Edelman 1987.
16. Based on text from the home page of the Neuroscience Institute, of which Gerald Edelman is the director (*www.nsi.edu*), and from a summary of Edelman's views by Flanagan 1994.
17. Changeux and Danchin 1976; Innocenti 1991.
18. For a summary, see Oppenheim 1998.
19. For review of regressive events, see O'Leary 1992.
20. Rakic et al. 1986.

21. Bourgeois et al. 1994.
22. Quartz and Sejnowski 1997.
23. Huttenlocher 1979.
24. See Quartz and Sejnowski 1997 and Katz and Shatz 1996. For one thing, it is very difficult to measure accurately the density of synapses in a brain region given that the region itself is changing size over time. Also, unless the synapse changes are related to specific cell types, it is hard to know what the implications would be. Finally, the relation of structural measures (like the number of synapses) to functional ones (is the synapse working?) is hard to assess. In early development, synapses are functional before they have the "look" of synapses, and these would go uncounted.
25. O'Leary 1992.
26. For further discussion, see Quartz and Sejnowski 1997.
27. Hubel and Wiesel 1962; Hubel and Wiesel 1963; Hubel and Wiesel 1965; Hubel and Wiesel 1972.
28. Apologies to visual scientists for this simplistic description of visual pathways.
29. For a summary see: Katz and Shatz 1996; Shatz 1996; Stryker 1991.
30. Antonini and Stryker 1993.
31. The experiment actually involved the injection of the tracer into cells in the visual thalamus area called the lateral geniculate nucleus. In this region, cells are organized in layers devoted to one eye or the other. By recording the action potentials elicited by stimulation of one eye, it is possible to find the layers and then to inject a cell in that layer with the chemical.
32. Actually, the tracer is actively transported to the terminal by natural processes that go on in the cell all the time. These are taking things made by the cell body and shipping them throughout the cell.
33. Quartz and Sejnowski 1997.
34. Neville 1990.
35. Neville and Lawson 1987.
36. Based on Katz and Shatz 1996.
37. Rakic 1977; Horton and Hocking 1996.
38. Galli and Maffei 1988; Wong et al. 1993.
39. Even when endogenous activity is blocked, the clusters develop if the nerves headed for the brain from the two eyes are electrically stimulated separately. This kind of stimulation simultaneously activates many fibers from a given eye to the brain, tricking the brain into thinking that it received lots of activity at the same time from one eye (see Stryker and Harris 1986; Crair 1998).
40. Chiaia et al. 1992.
41. Crair 1999.
42. Hebb 1949.
43. This phrase comes from Carla Shatz.
44. Katz and Shatz 1996; Shatz 1992; Shatz 1996; Stryker 1991; Purves 1994.
45. However, recall that cortical cells initially receive inputs from both eyes. So the cortical cell will actually receive correlated input from each eye, but at different times. How then can one eye come to dominate? Although each cell gets inputs from both eyes, the two eyes never quite have equal inputs, leading one eye to dominate slightly. Hebbian plasticity builds upon this preexisting bias, wiring the connection between the cortical cell and its more efficient inputs. While Hebbian plasticity may be enough to wire up a particular cell, more is needed to establish the cell-specific clusters, the so-called ocular dominance columns, in the cortex. Ken Miller of UCSF has some interesting proposals on this; see Miller 1994 and Wimbauer et al. 1997. As a result, one eye or the other will come to be more efficient in driving a cortical cell. Miller's work on this was pointed out to me by Tony Movshon of NYU. Hebbian plasticity thus takes care of the problem of how inputs from one eye come to control an individual cell, but leaves open the question of how cells that are responsive to one eye come to cluster together. For this, it is generally assumed that there are factors that allow presynaptic inputs that are nearby and that are active at the same time to link up.

46. Glanzman et al. 1990; Martin and Kandel 1996.
47. Tsien 2000; Bliss and Collingridge 1993; Purves et al. 1996; Brown et al. 1988.
48. Katz and Shatz 1996.
49. Katz and Shatz 1996; Johnson 1998; Schuman 1999.
50. Oppenheim 1998.
51. Lorenz and Tinbergen 1938; Lorenz 1950; Tinbergen 1951.
52. Lehrman 1953.
53. Terrace 1984.
54. Terrace 1984.
55. Watson 1925; Skinner 1938; Hull 1943.
56. Chomsky 1957.
57. Gardner 1987.
58. Keil 1999.
59. Garcia and Koelling 1966.
60. For discussion, see chapters by H. S. Terrace, P. P. G. Bateson, and J. L. Gould and P. Marler in the book edited by Marler and Terrace 1984.
61. Pinker 1994.
62. Pinker 1997.
63. Pinker 1997.
64. Bickerton 1980.
65. Elman et al. 1997; Quartz and Sejnowski 1997.
66. See Gopnik 1997; Korenberg et al. 2000; Ridley 1999.
67. Pinker 1994; Gopnik 1997; Korenberg et al. 2000; Bickerton 1980; Ridley 1999.
68. Ekman 1999.
69. Cosmides and Tooby 1999; Barkow et al. 1992.
70. Cosmides and Tooby 1999; Barkow et al. 1992; Spelke 1994; Carey and Spelke 1994; Povinelli and Preuss 1995.
71. Cosmides and Tooby 1999; Barkow et al. 1992.
72. Gould 1997.
73. Gould 1991.
74. Gould quoted in Gazzaniga 1992.
75. Premack 1985.
76. Pinker 1997; Pinker and Bloom 1990; Cosmides and Tooby 1999.
77. Rose and Rose 2000.

78. For example, see Edwards and Pap 1959.
79. Spelke 1994; Carey and Spelke 1994; Marcus 1999; Pinker 1994, 1997; Piattelli-Palmarini 1989.
80. Wexler 1999.
81. Fodor 1983; Gazzaniga 1992; Tooby and Cosmides 2000; Mody et al. 1997; Denenberg 1999.
82. Keil 1999.
83. There is also a domain-independent learning system in the brain (the explicit or declarative memory system). However, this system is involved in recording facts and experiences independent of rewards and punishment, and though it might be thought of as a universal learning system, it does not appear to play an essential role in the kinds of learned behaviors that the behaviorists studied.
84. Elman et al. 1997.
85. Quartz and Sejnowski 1997.
86. Barton 1997.
87. Brothers 1997.
88. Barton 1997.
89. Neisser 1998.
90. Alcock 1998.
91. Arnold 1980.
92. Alcock 1998; Wimer and Wimer 1985.
93. Described in Alcock 1998; based on Holden 1980.
94. Tellegen et al. 1988.
95. Described in Harris 1998. Harris also argues that genetic influences are underestimated by heritability scores, noting that the correlation between parents and children on personality traits is sufficiently weak that genes they share might fully account for any similarities that exist.
96. Gardner 1998.
97. For summary, see Schuster and Ashburn 1992; Jacobson 1993.
98. For summary, see Jacobson 1993.
99. Harris 1998; Gardner 1998.
100. See Hall et al. 1998.
101. See Bruer 1999.
102. Mooney 1999; Gould and Marler 1984; Doupe and Kuhl 1999; Bottejer and Johnson 1997; Singh et al. 2000; Jarvis et al. 1998.
103. Elman et al. 1997.
104. Bruer 1999.
105. See Tallal 2000; Tallal et al. 1998.
106. Bruer 1999.
107. Gopnik et al. 1999.

CHAPTER FIVE ADVENTURES IN TIME

1. Bartlett 1932; Schacter 1999.
2. Each time the brain learns something, it is changed.

3. Semon 1904; Schacter 1982.
4. Lashley 1929.
5. Lashley 1950.
6. Scoville and Milner 1957.
7. Scoville and Milner 1957; Milner 1962; Milner 1965; Milner 1967; Milner 1972.
8. Squire 1987; Cohen and Eichenbaum 1993.
9. Scoville and Milner 1957.
10. MacLean 1949; MacLean 1952; MacLean 1970.
11. Milner 1962; Corkin 1968.
12. Cohen 1980; Cohen and Squire 1980; Cohen and Corkin 1981.
13. Warrington and Weiskrantz 1973; Graf et al. 1984.
14. Weiskrantz and Warrington 1979.
15. Cohen and Squire 1980.
16. Schacter and Graf 1986.
17. The parahippocampal region consists of the entorhinal cortex, perirhinal cortex, and the parahippocampal cortex, as defined by Witter et al. 1989.
18. See Amaral et al. 1987; Suzuki and Amaral 1994; Witter et al. 1989; Burwell et al. 1995; Van Hoesen and Pandya 1975.
19. Entorhinal cortex, perirhinal cortex, and parahippocampal cortex are included in the parahippocampal region, as defined by Witter et al. 1989.
20. Jones and Powell 1970; Damasio 1989.
21. Mesulam et al. 1977.
22. This section is based on Squire and Kandel 1999.
23. The reason ECT produces memory disturbance is related to the fact that the conversion of short- to long-term memory is disturbed. For discussion, see Squire 1987.
24. McClelland et al. 1995.
25. Winson 1985; Buzsaki 1989; McNaughton 1998; Wilson and McNaughton 1994.
26. Wilson and McNaughton 1994; Nadasdy et al. 1999; Poe et al. 2000; Louie and Wilson 2001.
27. Nadel and Moscovitch 1997.
28. For discussion, see Nadel and Moscovitch 1997; Knowlton and Fanselow 1998.
29. Bontempi et al. 1999.
30. See Tulving 1983.
31. Vargha-Khadem et al. 1997.
32. Squire and Zola 1998.
33. Milner 1970.
34. For summary, see Mishkin and Murray 1994; Murray and Richmond 2001; Squire and Zola 1996, 1998.
35. Eichenbaum et al. 1994.
36. Section title adapted from Nadel and Willner 1980.
37. O'Keefe and Nadel 1978.
38. Olton et al. 1979.
39. O'Keefe and Nadel 1978.
40. Ranck 1973.
41. Muller et al. 1999.

42. See McNaughton 1998.
43. Morris 1984.
44. Cohen and Eichenbaum 1993.
45. Eichenbaum 2000.
46. Wicklegren 1979; Rolls 1990; Schmajuk and DiCarlo 1992; Gluck and Myers 1993; McClelland et al. 1995; Rudy and Sutherland 1992; Rudy and O'Reilly 1999.
47. O'Reilly and Rudy 2001.
48. McClelland et al. 1995.
49. Rudy and Sutherland 1992.
50. Skinner 1938; Skinner 1972; Hull 1943; Hull 1954.
51. Kandel and Spencer 1968.
52. Kandel and Spencer also noted that any approach to cellular physiology that could illuminate synaptic plasticity, even if irrelevant to behavior, might be reasonable at this stage since so little was known.
53. Cowan 1998.
54. Cohen 1974.
55. Pavlov 1927.
56. McAllister and McAllister 1971; Brown et al. 1951; Bolles and Fanselow 1980; Blanchard and Blanchard 1969.
57. Kluver and Bucy 1937; Weiskrantz 1956; Blanchard and Blanchard 1972.
58. LeDoux 1996; LeDoux 2000; Davis 1992; Davis et al. 1997; Kapp et al. 1992; Maren and Fanselow 1996; Maren 2001; Fendt and Fanselow 1999; Weinberger 1995.
59. Pitkänen et al. 1997.
60. LeDoux et al. 1990; Amaral et al. 1992; Herzog and Van Hoesen 1976.
61. Pitkänen et al. 1997.
62. Amorapanth et al. 2000.
63. Romanski and LeDoux 1992a; Doron and LeDoux 2000.
64. Bordi and LeDoux 1992.
65. Quirk et al. 1995; Quirk et al. 1997.
66. Romanski et al. 1993.
67. Quirk et al. 1995; Quirk et al. 1997; Repa et al. 2001.
68. Collins and Pare 2000; Maren 2000.
69. LeDoux 1990; Campeau and Davis 1995; LeDoux et al. 1990; Amorapanth et al. 2000.
70. Muller et al. 1997; Wilensky et al. 1999; Wilensky et al. 2000; Schafe and LeDoux 2000; Bailey et al. 1999; Helmstetter and Bellgowan 1994; Fanselow et al. 1994; Lee and Kim 1998; Maren et al. 1996; Miserendino et al. 1990; Gewirtz and Davis 1997.
71. Fanselow and LeDoux 1999; LeDoux 2000.
72. Thompson and Spencer 1966.
73. Gormezano 1972.
74. Weinberger 1995; Weinberger 1998.
75. For summary, see Thompson et al. 1983; Steinmetz and Thompson 1991; Hesslow and Yeo 1998; Medina et al. 2000.
76. Desmond and Moore 1982.
77. Thompson et al. 1983; Steinmetz and Thompson 1991; Thompson 1986; Thompson and Kim 1996; Hesslow and Yeo 1998.

78. Ito 1984.
79. Marr 1969; Eccles 1977; Ito 1984; Ito 1989.
80. Llinas and Welsh 1993.
81. Lisberger 1996; Lisberger 1998.
82. Steiner 1973.
83. Steiner 1973.
84. Grill and Norgren 1978.
85. As with any so-called innate function, a role for environmental factors cannot be ruled out (see discussion in chapter 4).
86. Garcia and Koelling 1966.
87. Garcia 1990.
88. See Chambers 1990; Yamamoto et al. 1994; Lamprecht and Dudai 2000.
89. Although some CS-US integration may occur in the hindbrain, any integration here is not believed to be sufficient to mediate CTA.
90. Berrige 1999.
91. Lamprecht and Dudai 1996; Lamprecht et al. 1997; Dunn and Everitt 1988; Lamprecht and Dudai 2000.
92. Schafe et al. 1998.
93. Manns et al. 2000.
94. Moyer et al. 1990; Moyer et al. 1996; LaBar and Disterhoft 1998; Huerta et al. 2000.
95. Chun and Phelps 1999.
96. Blanchard and Blanchard 1969; Bolles and Fanselow 1980.
97. Kim and Fanselow 1992; Phillips and LeDoux 1992; Maren and Fanselow 1996; Frankland et al. 1998; Selden et al. 1991.
98. Schacter 2001.
99. De Leon et al. 1995.
100. Damage to implicit systems does not destroy personality solely by affecting implicit memory functions. All systems operate on the basis of synaptic connections that are epigenetically specified during early development (that is, that are constructed from genetic and environmental influences) and then altered each time the neural system involved is active and engages in some form of learning. For more information on development, see chapter 4. For more information on learning-induced synaptic changes, see chapter 6.

CHAPTER SIX SMALL CHANGE

1. Ramón y Cajal 1909–1911.
2. Hartley 1749.
3. James 1890.
4. Freud 1887–1902.
5. Hebb 1949.
6. Comment made by Bruce McNaughton at a Neurobiology of Learning and Memory Conference in Park City, Utah, sometime in the late 1990s. McNaughton had been a student at McGill.
7. The Hebb learning rule was formalized by Stent 1973.
8. Jacobson 1993.

9. Ramón y Cajal 1911 proposed a mechanism called neurobiotaxis, but this notion has long been viewed as unrealistic (see Kandel and Spencer 1968).
10. Konorski 1948.
11. For summary, see Kandel 1976. Particularly important were early studies by Larrabee and Bronk 1947; Lloyd 1949; Brock et al. 1952.
12. Brock et al. 1952.
13. Eccles 1953.
14. This reflex is studied by measuring muscle twitches in response to electrical stimulation of the skin or nerves carrying sensory information from the skin to the spinal cord.
15. Thompson and Spencer 1966.
16. Hawkins et al. 1987.
17. Kandel and Spencer 1968.
18. For a review of some of the major invertebrate systems that have been studied over the years, see Beggs et al. 1999.
19. Lømo 1966.
20. Interview with T. Lømo in the *Journal of NIH Research* 1995.
21. Bliss and Lømo 1973.
22. Interview with Bliss in the *Journal of NIH Research* 1995.
23. It's not exactly right to say they failed. Spinal cord researchers found that they could produce changes in synaptic transmission lasting up to several hours. However, it took very strong and prolonged stimuli to produce these effects, and the potentiation then drifted away. In contrast, in the hippocampus, much milder stimuli did the trick and the responses remained for very long periods, as long as weeks in some studies where LTP was recorded in awake behaving animals.
24. Iriki et al. 1987; Castro-Alamancos et al. 1995; Bear and Malenka 1994; Huang and Kandel 1998; Weisskopf et al. 1999; Randic et al. 1993; Pennartz et al. 1993; Kombian and Malenka 1994.
25. Sanes and Lichtman 1999.
26. Bliss and Collingridge 1993; Lynch 1986; McNaughton and Barnes 1990.
27. McNaughton et al. 1978.
28. Levy and Steward 1979.
29. Kelso et al. 1986; Malinow and Miller 1986; Wigström et al. 1986. It's important to note that the postsynaptic cell does not have to fire action potentials to be active in the Hebbian sense. Its membrane potential just needs to become less negative.
30. For summaries of early research on LTP mechanisms, see Lynch 1986; Bliss and Collingridge 1993; Nicoll and Malenka 1995; Malenka and Nicoll 1999.
31. Nowak et al. 1984; Mayer and Westbrook 1987; Bliss and Collingridge 1993; Malenka and Nicoll 1999; Nicoll and Malenka 1995.
32. Brown et al. 1988.
33. Husi and Grant 2001.
34. The actual duration is not known since brain slices can't be kept alive much longer than several hours in a dish.
35. Huang et al. 1996.
36. For a discussion of different forms of LTP, see: Bliss and Collingridge 1993; Nicoll and Malenka 1995; Johnston et al. 1999; Morgan and Teyler 1999.

37. For a discussion of the relation of short-term memory in psychology and biology, see Dudai 1989, 1996, 1997; Squire and Kandel 1999.
38. Davis and Squire 1984.
39. Huang et al. 1996.
40. Squire and Kandel 1999.
41. Some of the relevant kinases include calcium/calmodulin protein kinases (CaMKII), protein kinase C, and tyrosine kinase. The actions of CaMKII are particularly well worked out. When calcium enters through the NMDA receptor, it forms a chemical complex with calmodulin (calcium/calmodulin) that then activates the alpha form of calcium/calmodulin kinase. This kinase, which is inactive until it interacts with the calcium/calmodulin complex, remains active even when calcium levels return to normal, since it can engage in autophosphorylation (it activates itself). See Lisman 1994; Elgersma and Silva 1999; Mayford et al. 1996; Mayford and Kandel 1999.
42. Soderling 1996; Shi et al. 1999.
43. Huang et al. 1996; Malgaroli and Tsien 1992; Bekkers and Stevens 1990.
44. Malgaroli and Tsien 1992; Bekkers and Stevens 1990.
45. Hawkins et al. 1994; O'Dell et al. 1994.
46. Malenka and Nicoll 1999.
47. Included are protein kinase A (PKA) and mitogen-activated protein kinase (MAP kinase). The steps involved in PKA activation are believed to include the following. First, calcium entry through NMDA receptors leads to the formation of the calcium/calmodulin complex. In early LTP, this leads to activation of CaKII, but for late LTP, calcium/calmodulin has to activate the cAMP cascade by initiating the production of cAMP by adenyl cyclase (this is part of the classic cell cycle, an important metabolic process that occurs in all cells in the body—that is, energy is released when adenyl cyclase makes cAMP from ATP). cAMP then activates PKA. Once activated, the regulatory subunit is removed from the catalytic subunit, and PKA moves to the cell nucleus. See Huang et al. 1996; Elgersma and Silva 1999; Mayford et al. 1996; Mayford and Kandel 1999.
48. For summary of genetic tools, see: Silva et al. 1997; Mayford et al. 1995; Mansuy 1998; Mayford and Kandel 1999; Tsien 2000.
49. For example, see McHugh et al. 1996.
50. Silva et al. 1997; Mayford et al. 1995; Gerlai 2000; Mayford and Kandel 1999; Tsien 2000; Tsien et al. 1996; Tang et al. 1999; Huerta et al. 2000.
51. Elgersma and Silva 1999; Silva et al. 1998; Kandel and Pittenger 1999; Mayford and Kandel 1999; Tsien 2000.
52. See Sanes and Lichtman 1999; Sweatt 1999; Kennedy 1999.
53. Sanes and Lichtman 1999.
54. Sweatt 1999; Kennedy 1999.
55. Frey and Morris 1997; Martin et al. 1997.
56. Lee et al. 1980; Chang et al. 1991; Desmond and Levy 1986; Engert and Bonhoeffer 1999; Toni et al. 1999.
57. Steward and Schuman 2001.
58. Morris et al. 1986.
59. For review, see Martin et al. 2000.

60. Staubli et al. 1989; Shapiro and Caramanos 1990; Bannerman et al. 1995; Cain et al. 1996; Shors and Matzel 1997; Keith and Rudy 1990.
61. Shors and Matzel 1997; Keith and Rudy 1990; Gallistel 1995.
62. Martin et al. 2000.
63. Tsien et al. 1996.
64. Tang et al. 1999.
65. Martin et al. 2000; Silva et al. 1997; Mayford et al. 1995; Gerlai 2000; Mayford and Kandel 1999.
66. Silva et al. 1998.
67. Abel et al. 1997; Mayford et al. 1996.
68. For discussion, see Shors and Matzel 1997.
69. Martin et al. 2000; Nicoll and Malenka 1995; Malenka and Nicoll 1999; Bliss and Collingridge 1993; Grover and Tyler 1990; Bortolotto et al. 1999; Bortolotto et al. 1999; Bekkers and Stevens 1990; Staubli et al. 1990; Weisskopf and Nicoll 1995.
70. Linden 1994; Bear and Malenka 1994; Ito 1996.
71. Shors and Matzel 1997; Gallistel 1995; Keith and Rudy 1990.
72. For review, see Teyler and DiScenna 1987; Teyler 1992; McNaughton and Barnes 1990; Moser 1995; Staubli 1995; Martinez and Derrick 1996; Morris et al. 1989; Morris 1992; Morris 1994; Martin 2000.
73. See Shors and Matzel 1997; Martinez and Derrick 1996; Martin et al. 2000; Barnes 1995; Eichenbaum 1995; Eichenbaum 1996.
74. Barnes 1995; Eichenbaum 1995.
75. Romanski et al. 1993.
76. An award given by the Society for Neuroscience.
77. Stevens 1998.
78. Barnes 1995; Eichenbaum 1995; Eichenbaum 1996.
79. Brown et al. 1988.
80. Rodrigues 2001; Miserendino et al. 1990; Gewirtz and Davis 1997; Walker and Davis 2000; Tang et al. 1999; Fanselow and Kim 1994; Maren et al. 1996; Lee and Kim 1998.
81. Schafe and LeDoux 2000; Schafe et al. 2000.
82. Schafe et al. 1999; Bourtchouladze 1998.
83. Josselyn et al. 2001.
84. Silva et al. 1992; Abel et al. 1997; Brambilla et al. 1997; Mayford and Kandel 1999; Tsien et al. 1996; Tang et al. 1999; Huerta et al. 2000; Silva et al. 1998.
85. Huang and Kandel 1998; Huang et al. 2000.
86. For a summary of other studies of amygdala LTP, see Maren 1999; Chapman et al. 1990; Chapman 2001.
87. Lamprecht et al. 1997; Rosenblum et al. 1997; Berman et al. 1998; Berman et al. 2000.
88. Weisskopf et al. 1999.
89. Schafe et al. 2001; Blair et al. 2001.
90. Tsien 2000; Paulsen and Sejnowski 2000; Magee and Johnston 1997; Johnston et al. 1999.
91. Huang and Kandel 1998; Huang et al. 2000; McKernan and Shinnick-Gallagher 1997.

92. Fanselow and LeDoux 1999; Cahill et al. 1999; Schafe et al. 2001; Blair et al. 2001.
93. Nader et al. 2000. For the history of reconsolidation research, see Sara 2000.
94. For review, see: Beggs et al. 1999; Sahley 1995; Crow 1988; Alkon 1989; Jing and Gillette 1995.
95. Some of Kandel's main colleagues and collaborators in the *Aplysia* work have included James Schwartz, Irving Kupferman, Vince Castellucci, Tom Carew, Robert Hawkins, Tom Abrams, Jack Byrne, Sam Schacter, Steve Sigelbaum, David Glanzman, Craig Bailey, Mary Chen, and Kelsey Martin. For summaries of the Kandel lab research on *Aplysia,* see Hawkins and Kandel 1984; Hawkins et al. 1987; Kandel 1989; Bailey et al. 1996; Kandel 1997.
96. Byrne et al. 1993; Cleary et al. 1998; Lechner and Byrne 1998.
97. Summarized in Dudai 1989.
98. For a discussion of why the learning in the *Aplysia* may not really be so simple, see Glanzman 1995.
99. In spite of the enormous progress, though, there's a gap between the relation of the research on the reduced experimental setups and the whole organism and its ability to learn. For a discussion, see Dudai 1989. Recent attempts have been made to close this gap by making the test paradigms for the simplified experimental setup more like real-life learning (see Hawkins et al. 1998).
100. The actual steps are that when serotonin binds to its receptors, adenyl cyclase is activated. cAMP is then made from ATP. The cAMP in turn activates PKA.
101. One of PKA's jobs is to phosphorylate an ion channel (a place on the cell membrane where ions, like calcium, sodium, and potassium, flow in and out). The ion channel in question is a special potassium channel. When phosphorylated by PKA, this channel closes, keeping potassium trapped inside the cell. The effect of this is that an action potential traveling down the sensory axon to the terminal lasts a bit longer. The reason for this is related to the fact that the exit of potassium out of the cell plays a key role in resetting the cell's electrical properties after an action potential, and this process is prolonged by phosphorylation of the potassium channel. Since calcium flows into the cell during action potentials, more calcium flows into the terminal after shock than before (since the action potential lasts longer). A longer action potential thus means more calcium coming in, which means more transmitter going out. And the more transmitter that goes out of the sensory terminal, the bigger will be the postsynaptic response of the motor neuron.
102. Specifically, what happens is that repeated shocks lead to the removal of regulatory subunits of PKA, leaving the catalytic subunit to translocate to the cell nucleus.
103. Involved are cell adhesion molecules.
104. Glanzman 1995; Murphy and Glanzman 1999; Lechner and Byrne 1998; Bao et al. 1998.
105. McKernan and Shinnick-Gallagher 1997.
106. Huang and Kandel 1998.
107. Huang et al. 1996; Malgaroli and Tsien 1992; Bekkers and Stevens 1990; Staubli et al. 1990; Weisskopf and Nicoll 1995.
108. This section is based on Dudai 1989.
109. Quinn et al. 1974; Tully and Quinn 1985.

110. For summaries of fly studies and memory molecules, see Dubnau and Tully 2001 and Yin and Tully 1996.
111. Davis 1996.
112. Staubli et al. 1994.
113. Rogan et al. 1997.
114. Tang et al. 1999.

CHAPTER SEVEN THE MENTAL TRILOGY

1. The phrase "The Mental Trilogy" comes from Hilgard 1980, who traced the three-way partition of mind to eighteenth-century Germany. However, a forerunner of this view is clearly found in Plato's tripartite soul.
2. See Hilgard 1980.
3. It's not completely fair to say that the trilogy was forgotten about during behaviorism. Instead, the processes involved were subsumed under behavioral interpretations. For example, behaviorists were interested in motivation as a driving force in behavior, but not as a mental state.
4. For cognitivists, emotions and motivations became kinds of cognitions, namely, thoughts about one's self in certain kinds of challenging situations. For more on the cognitive theory of emotion, see chapters 2 and 3 in *The Emotional Brain.*
5. Boring 1950.
6. Watson 1913; Watson 1925.
7. Minsky 1985.
8. It is well known that you can basically attend to one thing at a time, except under special circumstances. See Hirst et al. 1980.
9. Baddeley and Hitch 1974; Baddeley 1982; Baddeley 1992.
10. Bartlett 1932.
11. Gardner 1987.
12. Hilgard 1980.
13. Miller 1956.
14. Norman and Shallice 1980.
15. Johnson-Laird 1988.
16. Smith and Jonides 1999.
17. Hirst et al. 1980.
18. Luria 1973.
19. Stuss and Benson 1986; Nauta 1971; Fuster 1997; Lhermitte et al. 1972; Teuber 1964; Goldman-Rakic 1987; D'Esposito et al. 1995; Smith and Jonides 1999; Albright et al. 2000.
20. D'Esposito et al. 1995; Smith and Jonides 1999; Albright et al. 2000.
21. Smith and Jonides 1999.
22. Preuss 1995.
23. Jacobsen and Nissen 1937.
24. Fuster 1973; Fuster 1997; Fuster 2000; Fuster 1993; Levy and Goldman-Rakic 2000; Goldman-Rakic 1999; Goldman-Rakic 1987.
25. For a summary of prefrontal connections, see: Fuster 1989; Goldman-Rakic 1987; Passingham 1995; Groenewegen et al. 1990; Petrides and Pandya 1999; Fuster 1997; Maioli et al. 1998.

26. Ungerleider and Mishkin 1982; Zeki 1993; Van Essen et al. 1992; Van Essen 1985.
27. Van Essen 1995.
28. Ungerleider and Mishkin 1982; Ungerleider and Haxby 1994; Goodale 1998.
29. There is not universal agreement that the parietal area is involved in processing spatial information. Some prefer the view that the parietal region is more involved in planning and making decisions about movements than in the perception of where objects are located. Some of those who dispute this are Paul Glimcher, Carol Colby, Michael Goldberg, and Richard Andersen. See: Platt and Glimcher 1999; Colby and Goldberg 1999; Xing and Andersen 2000.
30. Mesulam et al. 1977; Pandya and Seltzer 1982.
31. Fuster 1973; Fuster 1997; Funahashi et al. 1989.
32. Gnadt and Andersen 1988; Koch and Fuster 1989.
33. Mesulam et al. 1977; Pandya and Seltzer 1982.
34. Miller et al. 1993; Miller and Desimone 1994.
35. Romanski et al. 1999.
36. This simple story involving specialized short-term buffers in the sensory systems and a general purpose working memory mechanism in the prefrontal cortex is somewhat more complicated than the way I have presented it. As discussed later, the prefrontal cortex itself seems to have regions that are specialized, at least to some degree, for specific kinds of working memory functions.
37. Cohen and Servan-Schreiber 1992; Cohen et al. 1999.
38. Vendrell et al. 1995.
39. Cohen and Servan-Schreiber 1992; Cohen et al. 1999.
40. Leung et al. 2000; Peterson et al. 1999; Epstein et al. 1999.
41. Cohen and Servan-Schreiber 1992; Cohen et al. 1999.
42. Goldman-Rakic 1999.
43. D'Esposito et al. 1995.
44. Smith and Jonides 1999.
45. Jacobsen 1935.
46. Reynolds and Desimone 1999.
47. Jiang et al. 2000; Haxby et al. 2000.
48. Asaad et al. 1998; Miller 1999.
49. Chelazzi et al. 1993.
50. Miller 1999; Desimone and Duncan 1995.
51. Knight 1997; Barcelo et al. 2000.
52. Tomita et al. 1999.
53. Kim and Shadlen 1999.
54. Shadlen et al. 1996.
55. Platt and Glimcher 1999; Colby and Goldberg 1999; Batista and Andersen 2001.
56. Batista and Andersen 2001.
57. Fuster et al. 1982.
58. Levy and Goldman-Rakic 2000.
59. Wilson et al. 1993.

60. Romanski et al. 1999.
61. Smith and Jonides 1999.
62. Smith and Jonides 1999.
63. This region is often called the dorso-lateral prefrontal cortex, but we'll refer to it as lateral prefrontal to keep things simple.
64. Smith and Jonides 1999; Bush et al. 2000.
65. Pandya and Yeterian 1996; Fuster 1997; Passingham 1995; Petrides and Pandya 1999; Maioli et al. 1998.
66. Posner 1992.
67. Berger and Posner 2000; Badgaiyan and Posner 1998; Bush et al. 2000; Botvinick et al. 1999; Carter et al. 2000.
68. Another important area is the orbital cortex, as we will discuss in chapter 8.
69. Smith and Jonides 1999; Bechara et al. 1998; Robbins 1996; Owen et al. 1999; Botvinick et al. 1999; Carter et al. 2000.
70. Circuit description below is based on: Douglas and Martin 1998; Durstewitz et al. 1999; Markram et al. 1997; Kritzer and Goldman-Rakic 1995; Cauller et al. 1998; Jones 1984.
71. Arnsten 1998.
72. Arnsten 1998; Robbins 2000; Arnsten et al. 1994; Cai and Arnsten 1997; Muller et al. 1998.
73. Nieuwenhuys 1985.
74. Sawaguchi et al. 1988; Sawaguchi et al. 1990. These effects may well be related to the fact that infusion of dopamine enhances the responses of cells to cue stimuli that signal reward, and also enhances the activity of cells during a delay period between the cue and the reward.
75. Lindvall et al. 1978; Berger et al. 1976; Lewis et al. 1987.
76. Arnsten 1998; Robbins 2000.
77. Yang and Seamans 1996.
78. Thierry et al. 1993.
79. Arnsten 1998.
80. Woolf 1925.
81. Baars 1997; Johnson-Laird 1993; Kihlstrom 1987; Marcel and Bisiach 1988; Norman and Shallice 1980; Shallice 1988; Kosslyn and Koenig 1992.
82. Lashley 1950.
83. For example, see: Stuss 1991; Luria 1969; Ackerly and Benton 1947.
84. Luria 1969.
85. Ackerly and Benton 1947.
86. Stuss 1991.
87. Damasio 1999; Panksepp 1998.
88. Milner 1982; Shimamura 1995.
89. Buckner and Koutstaal 1998; Wagner 1999; Cabeza and Nyberg 2000; Lepage et al. 2000; Schacter et al. 1998.
90. Wagner 1999.
91. Crick and Koch 1990, 1995.
92. He et al. 1996; Tootell et al. 1995; Damasio 1995.

93. Tootell et al. 1995.
94. For more discussion of the implications of the Tootell study for the Crick/Koch hypothesis, see Damasio 1995.
95. Milner 1974; von der Malsburg 1995.
96. von der Malsburg 1995; Roskies 1999; Treisman 1996.
97. Engel and Singer 2001; Crick and Koch 1990; Tononi and Edelman 1998; Damasio 1990; Llinas and Ribary 1994; Grossberg 1999.
98. Engel and Singer 2001.
99. Shadlen and Movshon 1999.
100. Smith and Jonides 1999; Owen et al. 1999.
101. Some have argued that there is a rudimentary lateral prefrontal cortex in the rat, but this is debatable. See: Kolb and Tees 1990; Preuss 1995; Preuss 1995.
102. Gray 1987.
103. Aston-Jones et al. 1999.
104. Dillard 1974.
105. Gallup 1991; Kennan et al. 2000.
106. Delfour and Marten 2001; Reiss and Marino 2001. Also see the journal *Consciousness and Cognition*, volume 4, issue 2, 1995, for a discussion of this work and its implications.
107. Hauser et al. 1995.
108. Weiskrantz 1996; Kihlstrom 1987; Erdelyi 1985; LeDoux 1996; Wilson et al. 2000; Wilson (in press); Bargh 1990; Bargh and Chartrand 1999; Greenwald and Banaji 1995; Zajonc 1984; Loftus and Klinger 1992; Bowers 1984; Bowers and Meichenbaum 1984; Öhman 2000; Debner and Jacoby 1994; de Gelder et al. 1999.
109. Gazzaniga 1985; Gazzaniga 1992; Gazzaniga 1998.
110. Gazzaniga and LeDoux 1978.
111. Gazzaniga 1985; Gazzaniga 1992; Gazzaniga 1998.

CHAPTER EIGHT THE EMOTIONAL BRAIN REVISITED

1. James 1890.
2. Bard 1928; Cannon 1929; Herrick 1933; Papez 1937; Kluver and Bucy 1937; Hess and Brugger 1943; MacLean 1949; MacLean 1952; MacLean 1970; MacLean 1990.
3. The time I'm referring to is between the early 1960s and the early 1980s. Researchers like Mort Mishkin, Edmund Rolls, Jeffrey Gray, Jaak Panksepp, John Flynn, and Alan Siegel each did work on emotions and the brain for some of this period. For summaries, see: Flynn 1967; Mishkin and Aggleton 1981; Gray 1982; Rolls 1986; Panksepp 1982; Siegel and Edinger 1981.
4. See Neisser 1967; Gardner 1987.
5. Bard 1928; Cannon 1929; Herrick 1933; Papez 1937; MacLean 1949; MacLean 1952.
6. For example, during the behaviorist time, emotional and other mental states were reinterpreted in terms of verbal behavior. What had previously been called "feelings" by the introspectionists became verbal descriptions of one's emotional response tendencies. During the cognitive period, emotions were also recast, but this time in terms of consciously accessible and verbally describable thought processes called appraisals. Unconscious emotions have long been emphasized in the Freudian tradi-

tion, but verbal descriptions of mental states also play a key role in the psychoanalytic process, which seeks to bring repressed emotions to the forefront of consciousness, where they can be talked about.

7. Larsen and Fredrickson 1999; Stone et al. 1999; Schwarz and Strack 1999.
8. Plutchik 1980.
9. Plutchik 1980.
10. Hebb 1946.
11. Kahneman 1999.
12. Loftus 1986; Loftus and Hoffman 1989.
13. Bartlett 1932.
14. Kahneman 1999; Stone et al. 1999.
15. Larsen and Fredrickson 1999; Stone et al. 1999; Schwarz and Strack 1999.
16. Schacter and Singer 1962; Schacter 1975.
17. Panksepp 1998; Masson and McCarthy 1995.
18. Lamb et al. 1991.
19. Tinbergen 1951.
20. Neisser 1967; Gardner 1987.
21. Panksepp 1998.
22. LeDoux 1984; LeDoux 1987; LeDoux 1990.
23. Murphy and Zajonc 1993; Soares and Öhman 1993; Morris et al. 1998; Morris et al. 1999.
24. This does not, strictly speaking, imply that only humans are conscious. Instead, it means that humans are an example of a species where consciousness clearly exists. I discussed some issues about consciousness in other animals in chapter 7.
25. Behaviorists often used the term *black box* to refer to the fact that psychological processes in the mind were invisible to the experimenter. The cognitive and brain sciences have changed that.
26. Schacter and Singer 1962; Frijda 1986; Lazarus 1991; Smith and Lazarus 1990; Frijda 1993; Scherer 1988; Scherer 1993; Smith and Ellsworth 1985; Ellsworth 1991; Averill 1994; Oatley and Johnson-Laird 1987; Ortony et al. 1988.
27. MacLean 1949; MacLean 1952; MacLean 1970; MacLean 1990; Isaacson 1982.
28. Scoville and Milner 1957.
29. MacLean 1949; MacLean 1952.
30. Swanson 1983.
31. Nauta 1979.
32. Kaada 1960.
33. Isaacson 1982; Swanson 1983; Livingston and Escobar 1971.
34. Brodal 1982; Kotter and Meyer 1992; LeDoux 1987; LeDoux 1991.
35. Some researchers had been using fear conditioning to study behavior, and these were important developments in the ultimate application of this work to the brain. Included were: Estes and Skinner 1941; Mowrer and Lamoreaux 1946; Mowrer 1947; Miller 1948; Miller 1951; Brady and Hunt 1951; Solomon and Wynne 1954; Kamin 1963; Rescorla and Solomon 1967; Annau and Kamin 1961; Stebbins and Smith 1964; Brown et al. 1951; LoLordo 1967; Blanchard and Blanchard 1969; McAllister and McAllister 1971; Bolles et al. 1966; Bolles and Fanselow 1980; Bouton and Bolles 1980.

36. Several researchers used conditioned fear to study brain mechanisms of fear but did not pursue the circuits in detail, including John Harvey, Orville Smith, and Neal Schneiderman. See: Harvey et al. 1965; Marshall and Smith 1975; Schneiderman et al. 1974.
37. Blanchard and Blanchard 1972; Kapp et al. 1979; Kapp et al. 1984; Kapp et al. 1992; Hitchcock and Davis 1986; Davis et al. 1987; Davis 1992; Davis et al. 1997; Kim et al. 1993; Fanselow 1994; Maren and Fanselow 1996; Maren et al. 1996; Maren 2001; Fendt and Fanselow 1999; LeDoux 1984; LeDoux et al. 1985; Iwata et al. 1986; LeDoux et al. 1990; LeDoux 1986; LeDoux et al. 1989; LeDoux 1990; LeDoux 1992; Romanski and LeDoux 1992; LeDoux 1994; LeDoux 1995; LeDoux 1996; LeDoux 2000.
38. Cohen 1974; Cohen 1980.
39. Other approaches are available for studying fear, including various forms of avoidance conditioning, studies of behavior in an open field, reactions to a shock probe, "elevated plus maze" performance, and so on. Of these, avoidance conditioning has been used most extensively to study the neural basis of fear. This will be discussed further in chapter 9.
40. Some studies have found differences in the function of the left and right amygdala in the human brain, but the extent and significance of the differences are not understood at this point. For a recent interesting example, see Cahill et al. 2001.
41. Anagnostaras et al. 1999; Kim and Fanselow 1992; Maren and Fanselow 1996; Phillips and LeDoux 1992; Everitt and Robbins 1992.
42. Darwin 1872.
43. For example, some have suggested that hippocampal lesions make animals more active and so they freeze less not because they have not processed the context but simply because they are hyperactive. But if this were true, rats with hippocampal lesions should freeze less to a tone as well, but they do not. See: McNish et al. 1997; Maren et al. 1998.
44. Michael Davis and colleagues failed to find this effect, but they studied fear conditioning using a different procedure, which may account for the difference. See Gewirtz et al. 1997.
45. Quirk et al. 2000.
46. Garcia et al. 1999.
47. Damasio 1994; Bechara et al. 1999.
48. LaBar et al. 1995.
49. Bechara et al. 1995.
50. O'Connor et al. 1999.
51. LaBar et al. 1998; Morris et al. 1998.
52. Rolls 1999.
53. Actually, these studies don't measure neural activity but instead infer it from such measures as blood oxygenation in fMRI studies or blood flow in PET studies.
54. Morris et al. 1999.
55. Morris et al. 1996.
56. Breiter et al. 1996.
57. Whalen et al. 1998.
58. Adolphs et al. 1994; Calder et al. 1996; Young et al. 1996; Hamann et al. 1996; Scott et al. 1997.