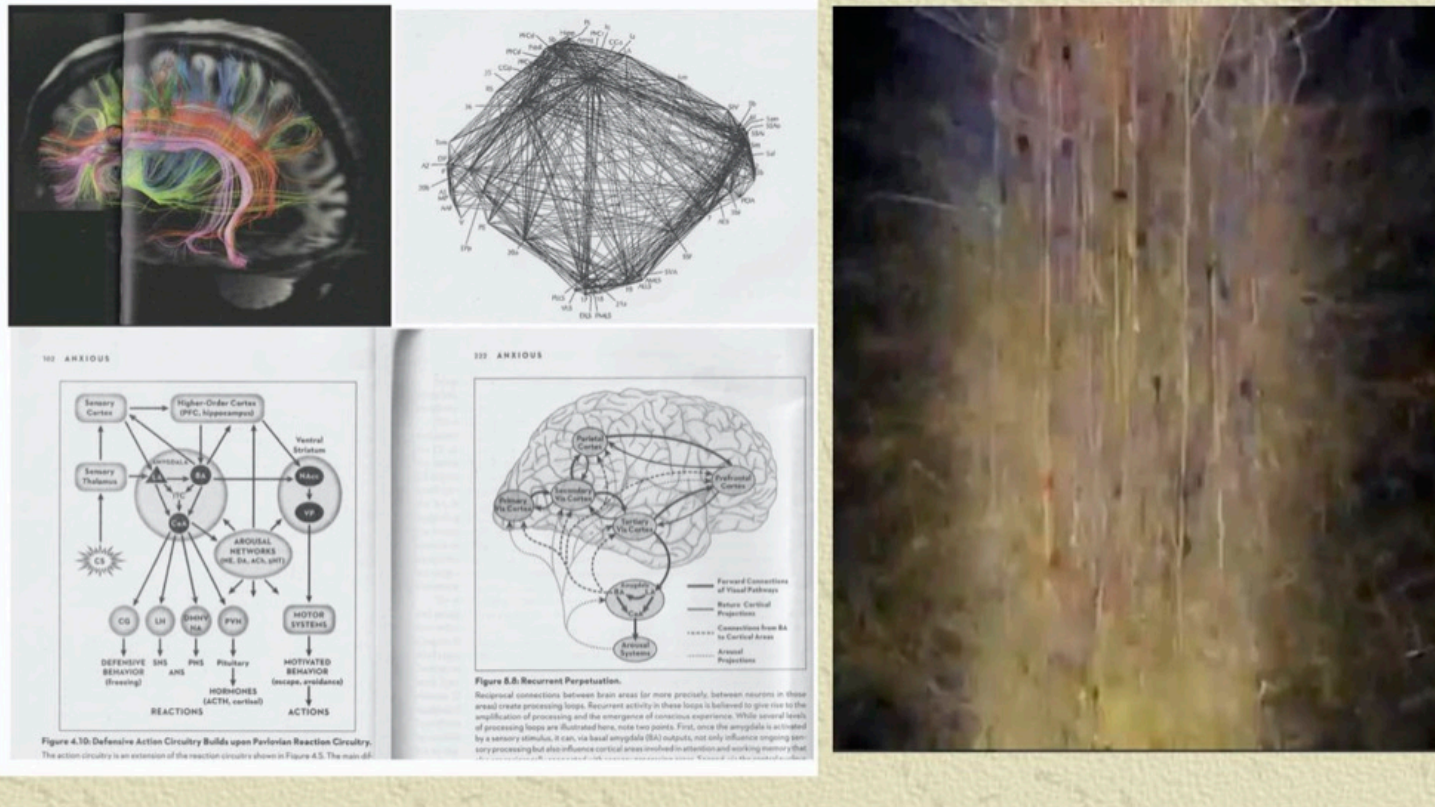


# 脳内神経系統図



原典: 左上: from 『脳と心のしくみ』 by 池谷裕二、 pages 14 - 15

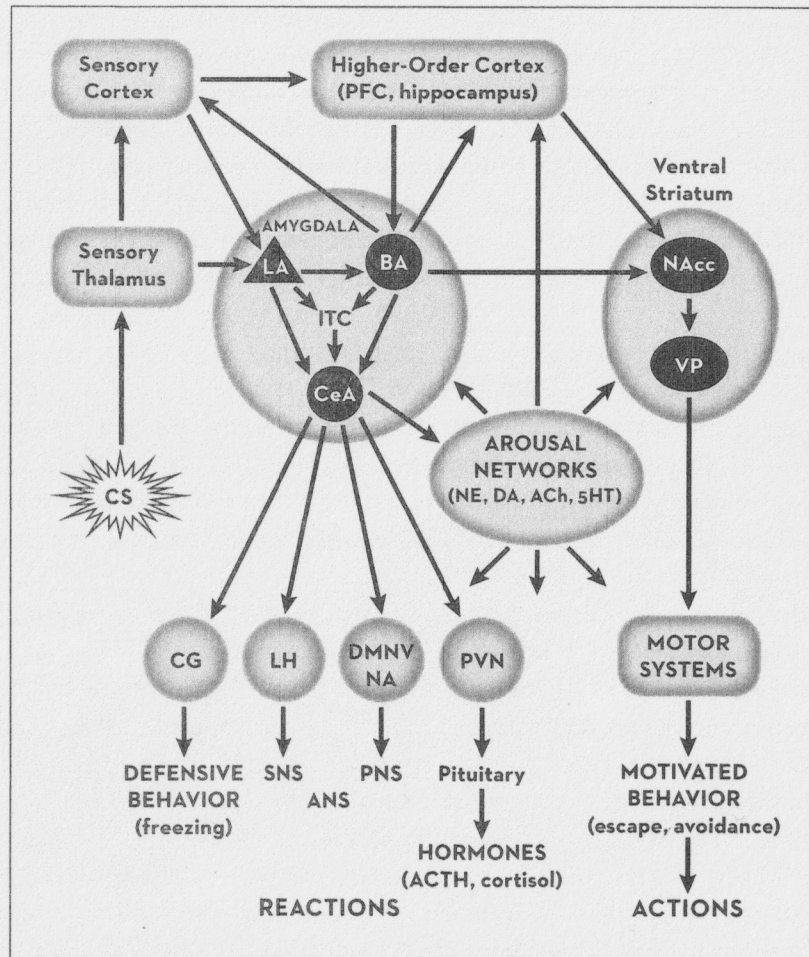
中上: from "A Universe of Consciousness" by Gerald Edelman & Giulio Tononi, Page 115

左下: from "Anxious" by Joseph LeDoux, page 102

中下: from "Anxious" by Joseph LeDoux, page 218

右: from the TED lecture "A brain in a supercomputer" presented by Henry Markram

<https://www.youtube.com/watch?v=LS3wMC2BpxU&t=678s>

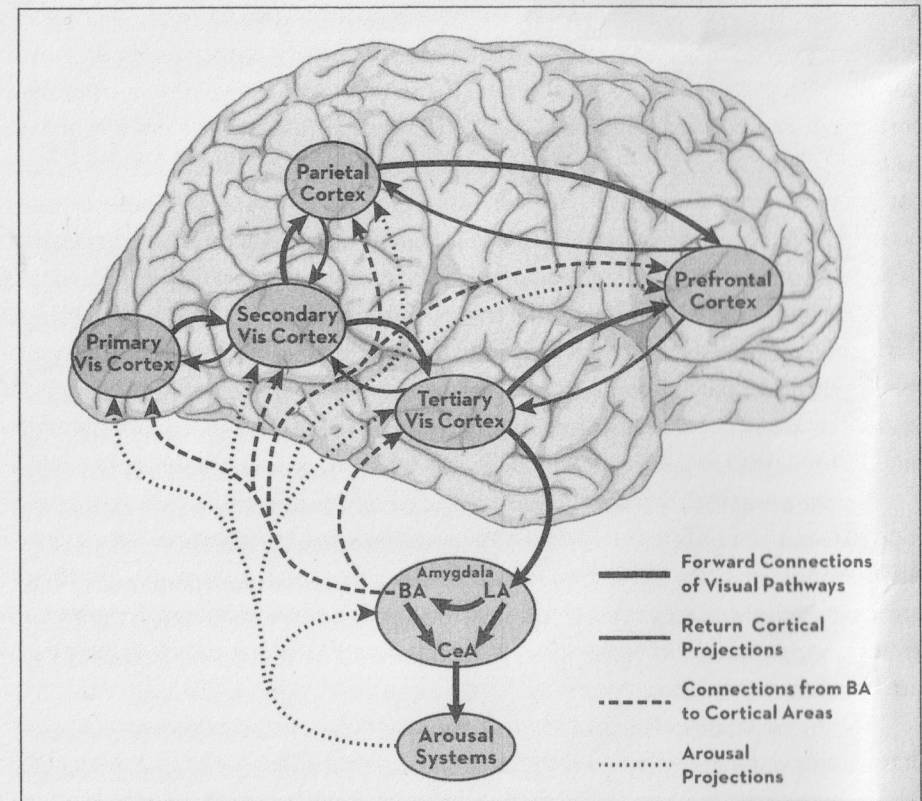


**Figure 4.10: Defensive Action Circuitry Builds upon Pavlovian Reaction Circuitry.**

The action circuitry is an extension of the reaction circuitry shown in Figure 4.5. The main difference is the connection from the basal amygdala (BA) to the NAcc of the ventral striatum, which allows the emission of actions under motivational influences signaled by information from the BA. For other abbreviations, see Figure 4.5.

freezing response is eliminated.<sup>81</sup> Thus, the CeA, although not necessary for avoidance, has a regulatory role in avoidance learning.

The redirection of information flow that allows avoidance to proceed is controlled by interactions between the amygdala and PFC<sub>VM</sub>.<sup>82</sup> A key output target of the BA is the *ventral striatum*, especially the *nucleus accumbens* (NAcc) and specifically its shell subdivision; damage to or functional inactivation of this



**Figure 8.8: Recurrent Perpetuation.**

Reciprocal connections between brain areas (or more precisely, between neurons in those areas) create processing loops. Recurrent activity in these loops is believed to give rise to the amplification of processing and the emergence of conscious experience. While several levels of processing loops are illustrated here, note two points. First, once the amygdala is activated by a sensory stimulus, it can, via basal amygdala (BA) outputs, not only influence ongoing sensory processing but also influence cortical areas involved in attention and working memory that also are reciprocally connected with sensory processing areas. Second, via the central nucleus (CeA), in addition to initiating defensive responses and supporting physiological changes in the body (not shown here), arousal systems within the brain are activated and release their chemicals and modulate processing in all of the areas mentioned above, but also release in the amygdala and modulate its processing. Thus, so long as the threat persists, a massive system of feed-back and feed-forward amplification of processing occurs via multiple layers or recurrent connectivity, keeping the organism engaged and energized to cope with the threat.

exerts bottom-up influences on sensory processing and attention; once attention is captured, top-down executive attention biases sensory processing. People with anxiety disorders have this in the extreme.<sup>76</sup> Through arousal and reentrant pro-

CONSCIOUS EXPERIENCE REQUIRES STRONG AND RAPID REENRANT INTERACTIONS

Perhaps the most direct indications of how important rapid reentrant neural interactions are for generating a unified conscious experience are disconnection syndromes in neurology and dissociation disorders in psychiatry. These are syndromes in which one or more areas of the brain are anatomically or functionally disconnected from the rest of the brain owing to some pathological process, traumatic event, or surgery while the areas themselves are relatively undamaged.

Disconnection Syndromes<sup>1</sup>

The most dramatic and certainly the best studied form of disconnection is the split-brain syndrome, in which persons with intractable epilepsy undergo surgery that severs the large number of reciprocal connections between the two halves of the brain (see figure 6.1; these connections are made via the corpus callosum and, in a few cases, the anterior commissure).

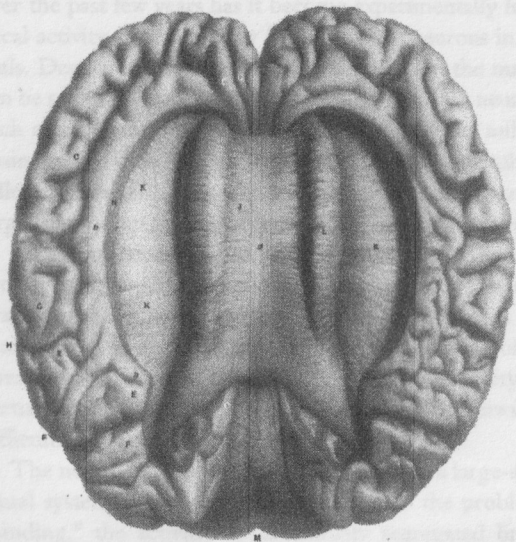


FIGURE 6.1 The corpus callosum, approximately 200 million nerve fibers reciprocally linking the two cerebral hemispheres. The brain was dissected from above; the fibers run horizontally, and their faint striations can be seen.

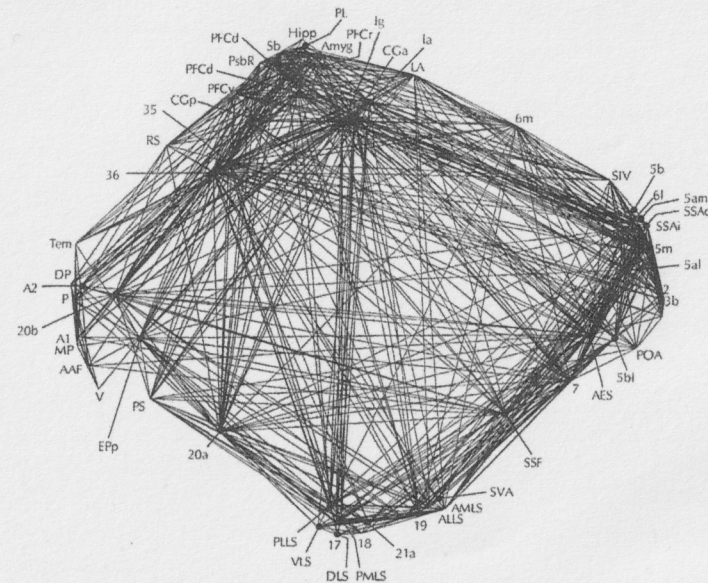


FIGURE 10.1 WHAT IS CONNECTED TO WHAT. The diagram represents 64 areas from the cerebral cortex of the cat with 1,134 connection paths between them (the abbreviations referring to the technical names of different brain areas do not need to concern us here). Most connection paths are reciprocal. Areas are depicted close to each other if they are connected and far away if they are not connected. The resulting topological organization reflects their connections, not their locations in the brain.

In the model (figure 10.2), reentry occurs among nine visual cortical areas, divided into three anatomical streams mediating responses to form, color, and motion, respectively. No superordinate area coordinates the responses of the model. Consistent with functional segregation in the visual cortex, neural units within each separate area of the model respond to different properties of the stimuli, and the firing of each has different functional consequences within the network. For example, groups of neurons in model area V1, corresponding to the primary visual cortex, respond to elementary features of objects, such as the orientation of edges in a particular position of the visual field. Groups of neurons in higher visual areas, such as area IT, corresponding to the inferotemporal cortex, respond to classes of objects that have a certain shape irrespective of their position in the visual field.

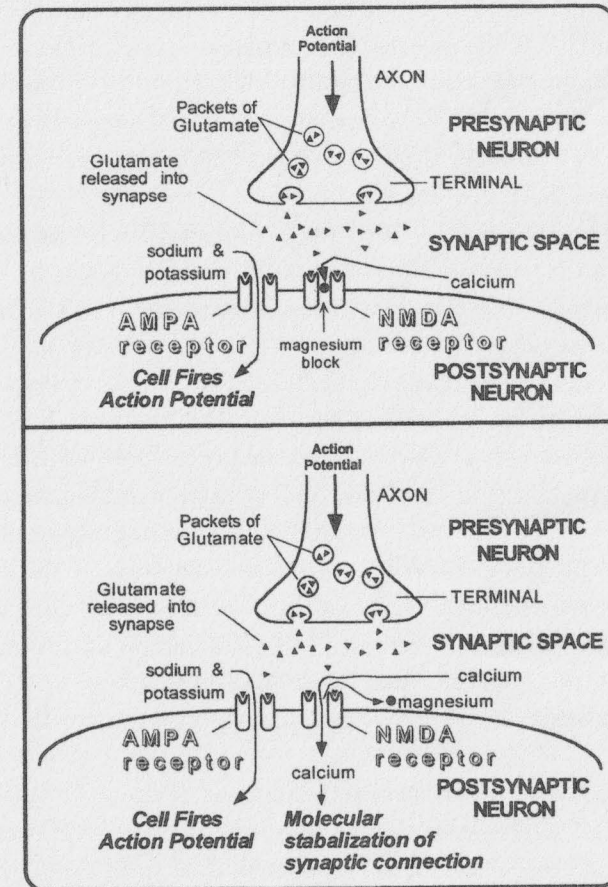
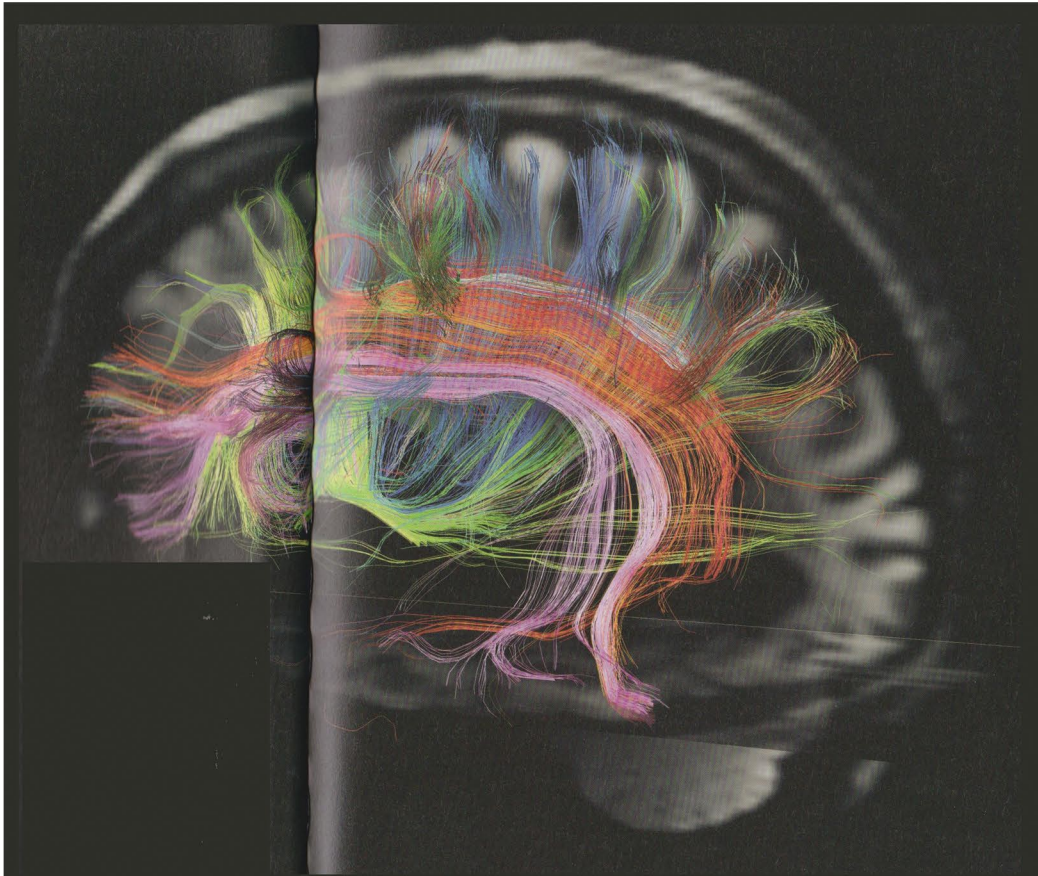


FIGURE 7-11  
Glutamate Receptors.

When an action potential comes down the axon to the terminal area, it causes packets of glutamate to be released from the terminal of the presynaptic neuron. The released glutamate diffuses into the synaptic space and binds to AMPA and NMDA receptors on the dendrites of postsynaptic neurons. When glutamate binds to AMPA receptors, sodium and potassium flow into the postsynaptic neuron and help generate an action potential (above). Although NMDA receptors are normally blocked by magnesium, the magnesium block is removed by the action of glutamate at AMPA receptors. Calcium then flows into the cell below, resulting in a host of molecular changes that then strengthen and stabilize the connection between the pre- and postsynaptic neuron. (Illustration based on figure 1 in F.A. Edwards (1992), Potentially right on both sides.