atypical antipsychotic  An antipsychotic drug that has actions other than or in addition to the dopamine D₂ receptor antagonism that characterizes the typical antipsychotics.

antidepressant  A drug that relieves the symptoms of depression.

monoamine oxidase (MAO)  An enzyme that breaks down monoamine transmitters, thereby inactivating them.

tricyclic antidepressant  An antidepressant that acts by increasing the synaptic accumulation of serotonin and norepinephrine.

selective serotonin reuptake inhibitor (SSRI)  An antidepressant drug that blocks the reuptake of transmitter at serotonergic synapses.

depressant  A drug that reduces the excitability of neurons.

barbiturate  An early anxiolytic drug and sleep aid that has depressant activity in the nervous system.

anxiolytic  A drug that is used to combat anxiety.

benzodiazepine  Any of a class of antianxiety drug that are agonists of GABAₐ receptors in the central nervous system. One example is diazepam (Valium).

opium  An extract of the opium poppy, Papaver somniferum. Drugs based on opium are potent painkillers.

morphine  An opiate compound derived from the poppy flower.

drugs are so good at relieving the symptoms of schizophrenia that a dopaminergic model of the disease became dominant (see Chapter 12). More recently, atypical antipsychotics have been developed that have both dopaminergic and additional, nondopaminergic actions, especially the blockade of certain serotonin receptors. These drugs may be helpful in relieving symptoms that are resistant to the typical antipsychotics.

ANTIDEPRESSANTS  Disturbances of mood called affective disorders are among the most common of all psychiatric complaints (World Health Organization, 2001). In contrast to the antipsychotic drugs, which reduce synaptic activity by blocking receptors, effective antidepressant drugs act to increase synaptic transmission. Some of the earliest antidepressants were the monoamine oxidase (MAO) inhibitors, which, as their name suggests, block the enzyme responsible for breaking down monoamine transmitters like dopamine, serotonin, and norepinephrine. This action allows transmitter molecules to accumulate in the synapses (see Figure 4.7, step 8), with an associated improvement in mood. Later generations of antidepressants also increase synaptic transmitter availability, but they focus on specific transmitters: the tricyclic antidepressants block the reuptake of serotonin and norepinephrine, while selective serotonin reuptake inhibitors (SSRIs) like fluoxetine (Prozac) and citalopram (Celexa) are so named because they act specifically at serotonergic synapses.

ANXIOLYTICS  Severe anxiety, in the form of panic attacks, phobias (specific irrational fears), and generalized anxiety, can spiral out of control and become disabling; many millions of people suffer from anxiety disorders (see Chapter 12). Anything that reduces or depresses the excitability of neurons tends to counter these states, which explains some of the historical popularity of depressants like alcohol and opium. Unfortunately, these substances are burdened with strong potential for intoxication and addiction, so they are not suitable for therapeutic use. Barbiturate drugs, such as phenobarbital, were originally developed to reduce anxiety, promote sleep, and avoid epileptic seizures. They are still used occasionally for those purposes, but they are also addictive and easy to overdose on, often fatally, as illustrated in Figure 4.6B.

Over the last 50 years, the most heavily prescribed anxiolytics (anxiety drugs) have been the benzodiazepines; compared with the barbiturates, benzodiazepines are both more specific and safer (as illustrated for Ativan [lorazepam] in Figure 4.6B), although they still carry some risk of addiction. Members of this class of drug, such as Valium (diazepam) and Ativan, bind to specific sites on GABAₐ receptors and enhance the activity of GABA (Walters et al., 2000). Because GABA receptors are inhibitory, benzodiazepines help GABA to produce larger inhibitory postsynaptic potentials than GABA would produce alone. The net effect is a reduction in the excitability of neurons. The hunt for new anxiolytic agents—both exogenous and endogenous—is an area of intense research efforts. Hormones that interact with GABA receptors, as well as drugs that subtly alter serotonergic neurotransmission, are examples of these novel anxiolytics. Antidepressant drugs are often effective anxiolytics too.

OPIATES  Opium, extracted from poppy flower seedpods, has been used by humans since at least the Stone Age. Morphine, the major active substance in opium, is a very effective analgesic (painkiller) that has brought relief from severe pain to many millions of people (see Chapter 5). Unfortunately, because it produces powerful feelings of euphoria, morphine also has a strong potential for addiction, as do close relatives like heroin (diacetylmorphine) and opiate painkillers like OxyContin (oxycodone) and Vicodin (hydrocodone and acetaminophen).

Opiates like morphine, heroin, and codeine bind to specific receptors—opioid receptors—that are concentrated in various regions of the brain. The profusion of opioid receptors in an area called the periaqueductal gray (FIGURE 4.9) is especially important, because it is here that opiates exert their painkilling effects (see Chapter 5).