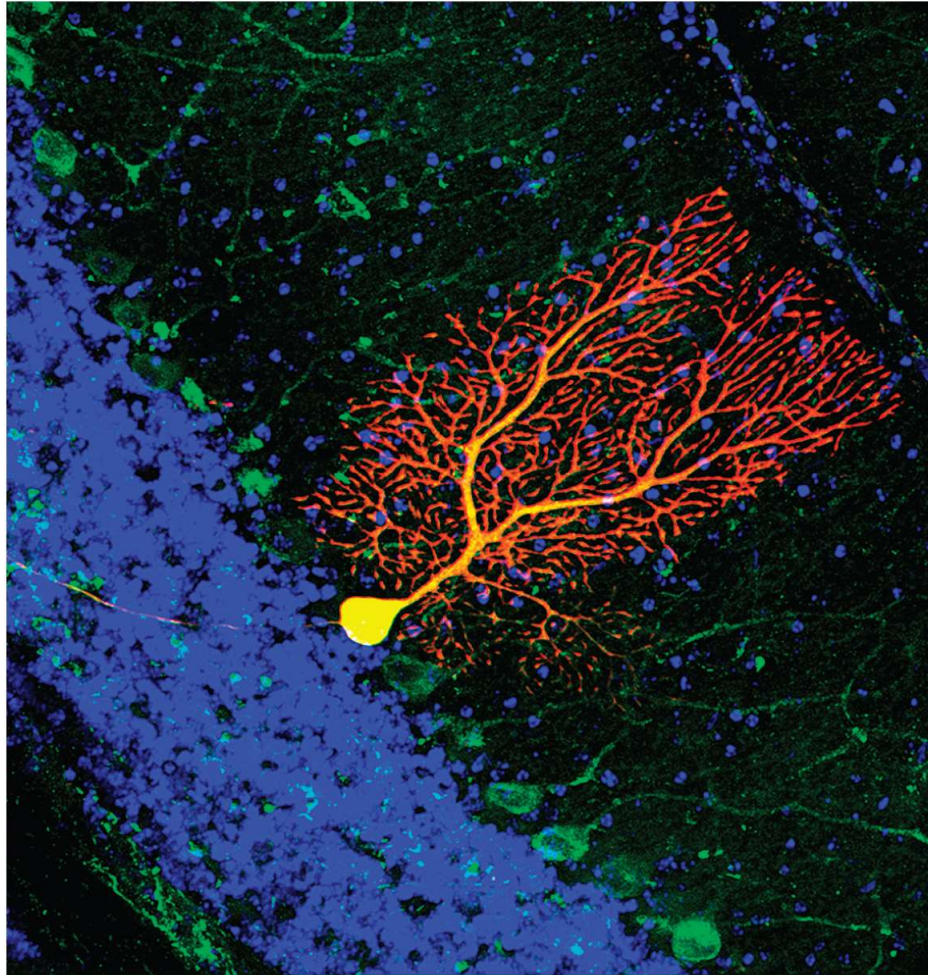


# Chapter 12

## Neural Coding



# Neural Circuits

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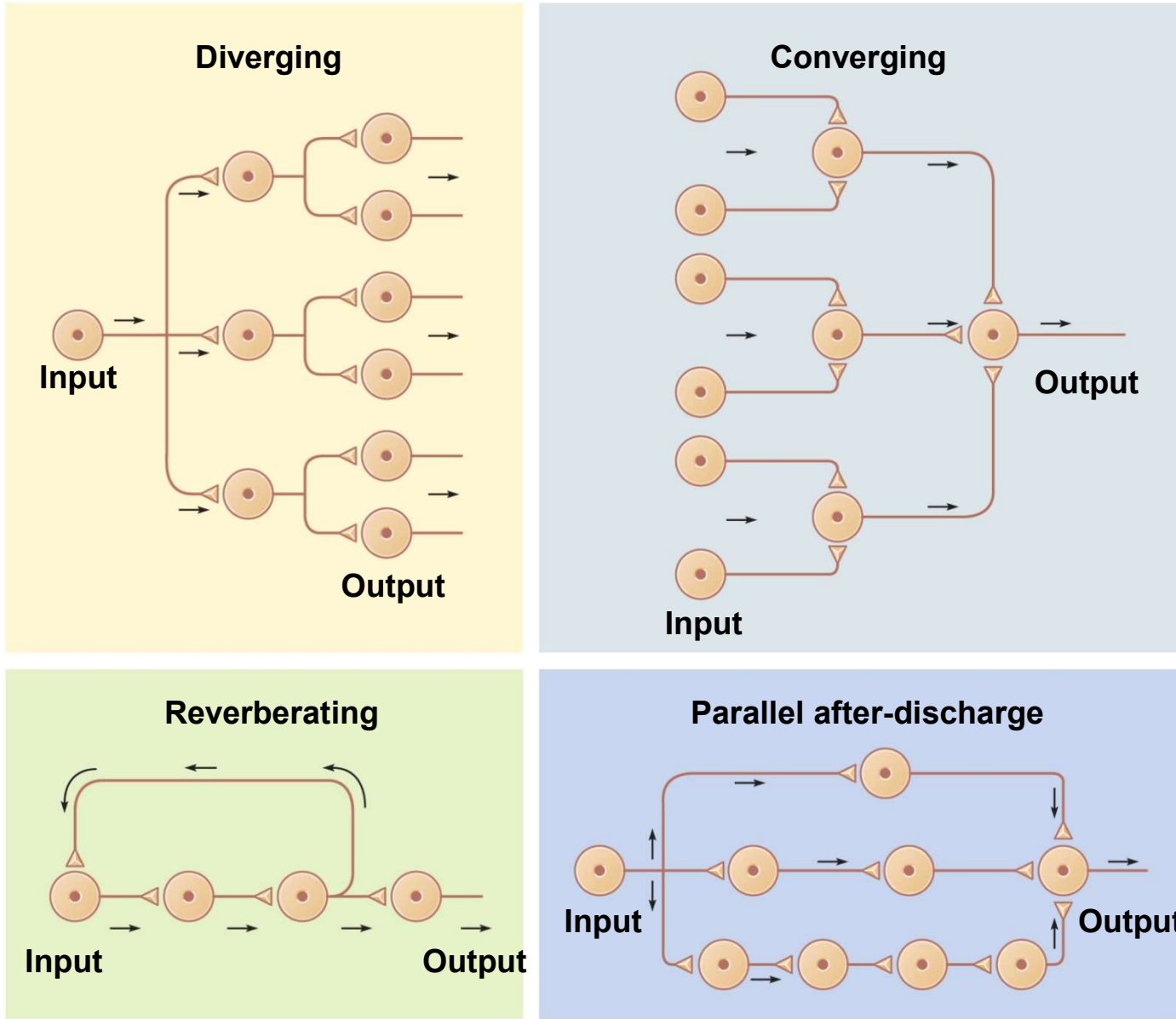


Figure 12.30

# Memory and Synaptic Plasticity

- physical basis of memory is a pathway through the brain called a **memory trace** or **engram**
  - along this pathway, new synapses were created or existing synapses modified to make transmission easier
  - **synaptic plasticity** – the ability of synapses to change
  - **synaptic potentiation** - the process of making transmission easier
- kinds of memory
  - **immediate, short-** and **long-term** memory
  - correlate with different modes of synaptic potentiation

# Immediate Memory

- **immediate memory** – the ability to hold something in your thoughts for just a few seconds
  - essential for reading ability
- feel for the flow of events (sense of the present)
- our memory of what just happened “echoes” in our minds for a few seconds
  - reverberating circuits

# Short-Term or Working Memory

- **short-term memory (STM)** - lasts from a few seconds to several hours
  - quickly forgotten if distracted
  - calling a phone number we just looked up
  - reverberating circuits
- facilitation causes memory to last longer
  - **tetanic stimulation** – rapid arrival of repetitive signals at a synapse // causes  $\text{Ca}^{2+}$  accumulation and postsynaptic cell more likely to fire
  - **post-tetanic potentiation** - to jog a memory
    - $\text{Ca}^{2+}$  level in synaptic knob stays elevated
    - little stimulation needed to recover memory

# Long-Term Memory

- types of long-term memory
  - **declarative** - retention of events that you can put into words
  - **procedural** - retention of motor skills
- physical remodeling of synapses // new branching of axons or dendrites
- molecular changes - **long-term potentiation**
  - changes in receptors and other features increases transmission across “experienced” synapses
  - effect is longer-lasting

# Molecular Changes and Long-Term Memory

- molecular changes are called **long-term potentiation**
- method described
  - receptors on synaptic knobs are usually blocked by  $Mg^{+2}$  ions
  - when bind glutamate and receive tetanic stimuli, they repel  $Mg^{+2}$  and admit  $Ca^{+2}$  into the dendrite –  $Ca^{+2}$  acts as second messenger
    - more synaptic knob receptors are produced
    - synthesizes proteins involved in synapse remodeling
    - releases nitric oxide that triggers more neurotransmitter release at presynaptic neuron