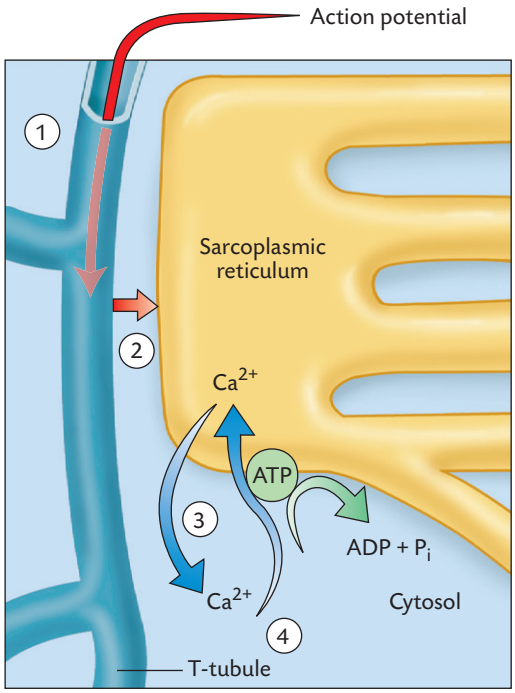

Figure 8.12 Control of Ca^{2+} concentration in the cytosol. The action potential spreads in the T-tubule system (1). Depolarization of T-tubules opens Ca^{2+} channels in the sarcoplasmic reticulum (2), which releases Ca^{2+} (3). ATP-dependent Ca^{2+} pumps in the membrane of the sarcoplasmic reticulum pump Ca^{2+} back into the organelle (4).

Pumping of Ca^{2+} back into the sarcoplasmic reticulum relaxes the muscle

continuously pump Ca^{2+} from the cytosol and into the sarcoplasmic reticulum. The Ca^{2+} concentration in the cytosol is therefore determined by the balance between the Ca^{2+} released to the cytosol and the Ca^{2+} pumped back into the sarcoplasmic reticulum. When the action potential is terminated, the release of Ca^{2+} from the sarcoplasmic reticulum ceases. Consequently, the cytosolic Ca^{2+} concentration falls. After termination of an action potential, it takes 10–100 ms before the Ca^{2+} concentration in the cytosol returns to its original low level and the muscle fiber is again completely relaxed.

The sequence of events that occurs between the generation of a nerve impulse in a motor neuron and the contraction of muscle fibers in the motor unit can be summarized as follows:

1 The nerve impulse is conducted along a motor nerve fiber to the nerve terminals that form synapses with the muscle fibers.
2 Depolarization of the nerve terminals opens voltage-gated Ca^{2+} channels, resulting in Ca^{2+} influx.
3 The increase in cytosolic Ca^{2+} concentrationin a nerve terminal leads to exocytosis of vesicles and release of acetylcholine into the synaptic cleft.
4 Acetylcholine diffuses across the synaptic cleft.
5 Acetylcholine binds to receptor sites on postsynaptic ion channels, which then open, and Na^+ diffuses into the muscle cell.
6 The inward Na^+ current depolarizes the membrane near the synapse to the threshold level, eliciting an action potential that spreads over the entire muscle fiber.
7 The action potential is conducted into the T-tubules, which results in Ca^{2+} channels in the sarcoplasmic reticulum opening.
8 Ca^{2+} flows out into the cytosol and binds to troponin.
9 Tropomyosin molecules change position, exposing the binding sites for the myosin heads on the actin filaments.
10 Myosin heads bind to the actin filaments.
11 The myosin heads bend, and the actin filaments slide relative to the myosin filaments.
12 ATP binds to the myosin heads, and the bonds between the myosin heads and actin are broken.
13 ATP is hydrolyzed and the energy released is transferred to the myosin heads, which straighten.
14 The steps from 10 to 13 are repeated for as long as Ca^{2+} is bound to troponin, and the muscle fiber contracts.
15 Ion pumps in the sarcoplasmic reticulum pump Ca^{2+} from the cytosol and back into the sarcoplasmic reticulum.
16 Ca^{2+} dissociates from troponin.
17 Tropomyosin again blocks the binding sites on actin, preventing the myosin heads f r o m binding.
18 The muscle fiber relaxes.

Skeletal muscle fibers are optimized for high contraction speed

The structure of a skeletal muscle fiber is optimized regarding contraction speed. All the overlapping regions of actin and myosin filaments are situated at exactly the same distance from the Z-discs, and the T-tubules and associated sarcoplasmic reticulum surround all the myofibrils at these regions on either side of the Z-discs. The calcium ions initiating the contraction are thus released from sites that have the shortest possible diffusion distance to where cross-bridges will be formed. Furthermore, the rapid conduction of action potentials along the whole surface of the fiber and into its central parts through the system of T-tubules results in simultaneous shortening of all the sarcomeres. However, the high contraction speed comes at

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17 Define concentric contraction and eccentric contraction.

18 Describe the mechanism of filament sliding during muscle contraction.

19 How is contraction of a skeletal muscle fiber elicited?

20 Explain how the structure of a skeletal muscle fiber ensures rapid contraction.