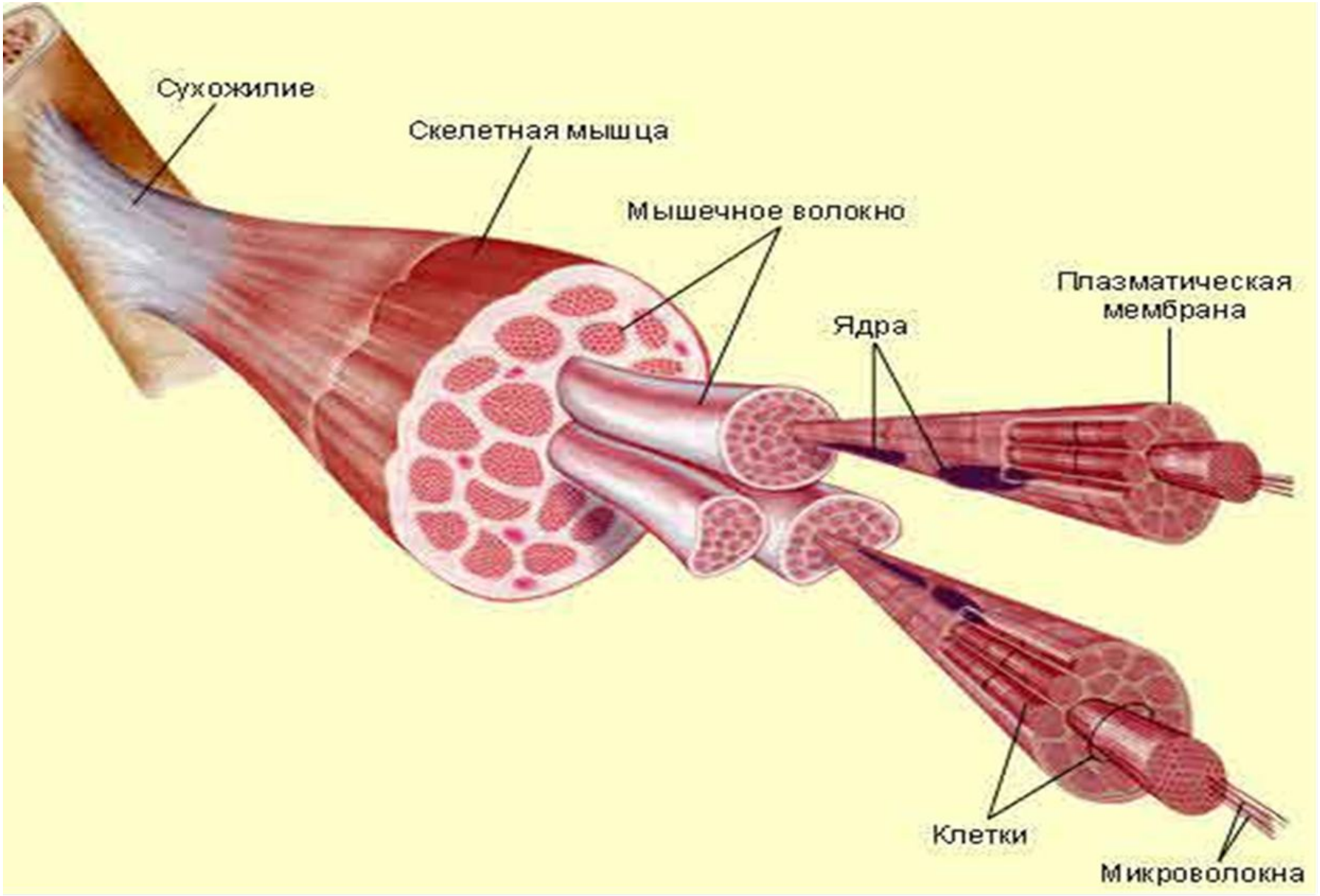
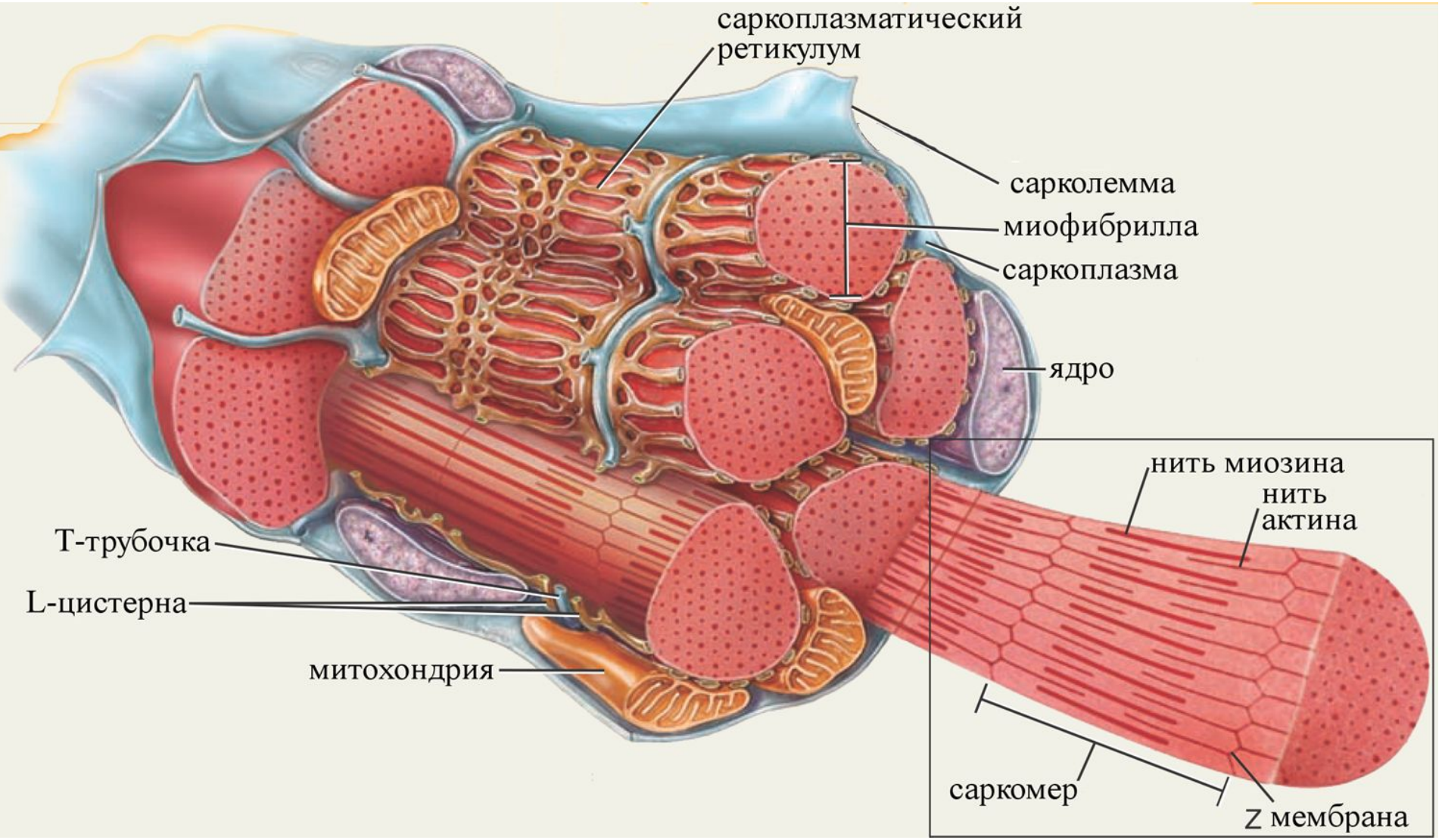
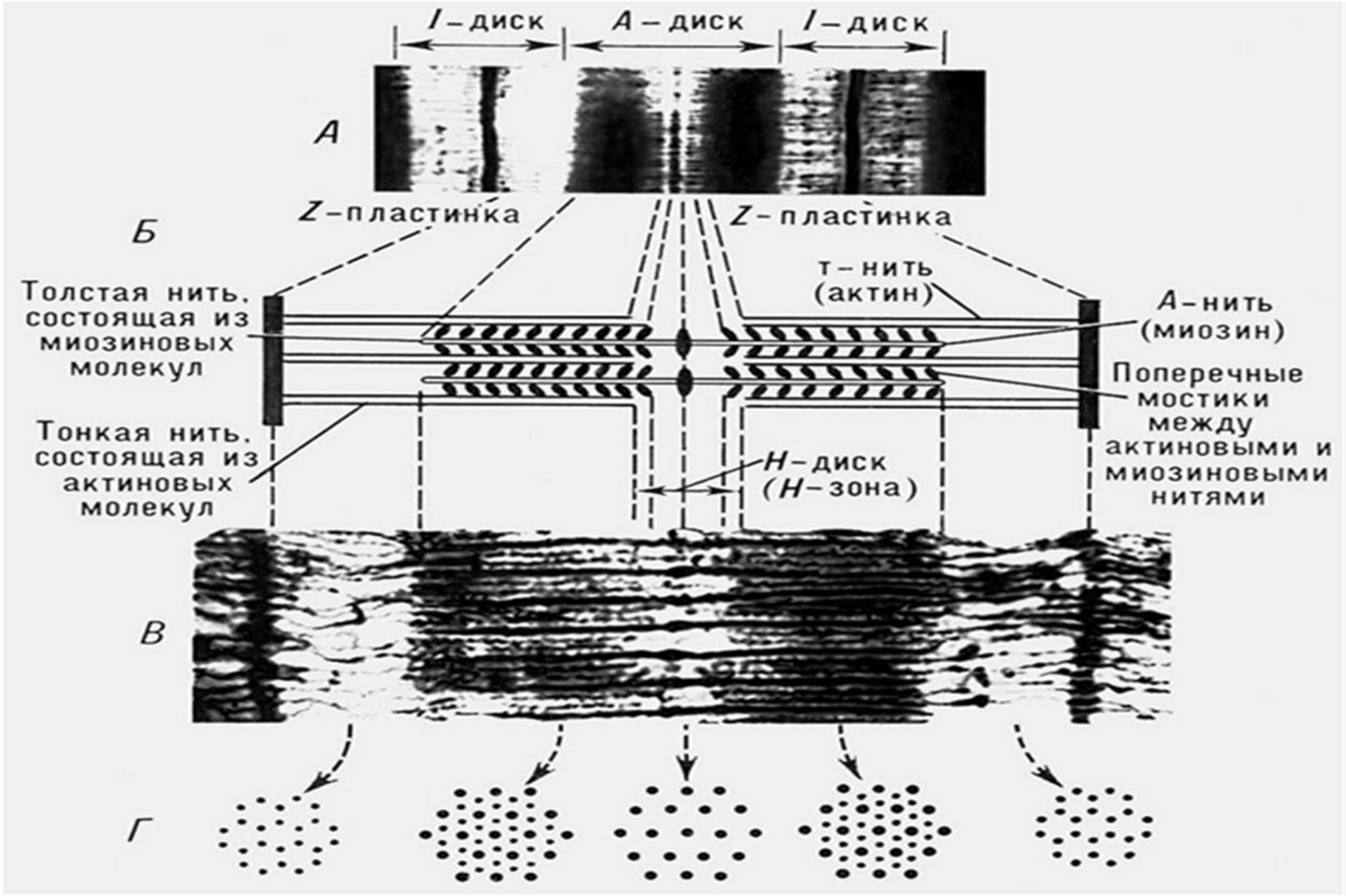
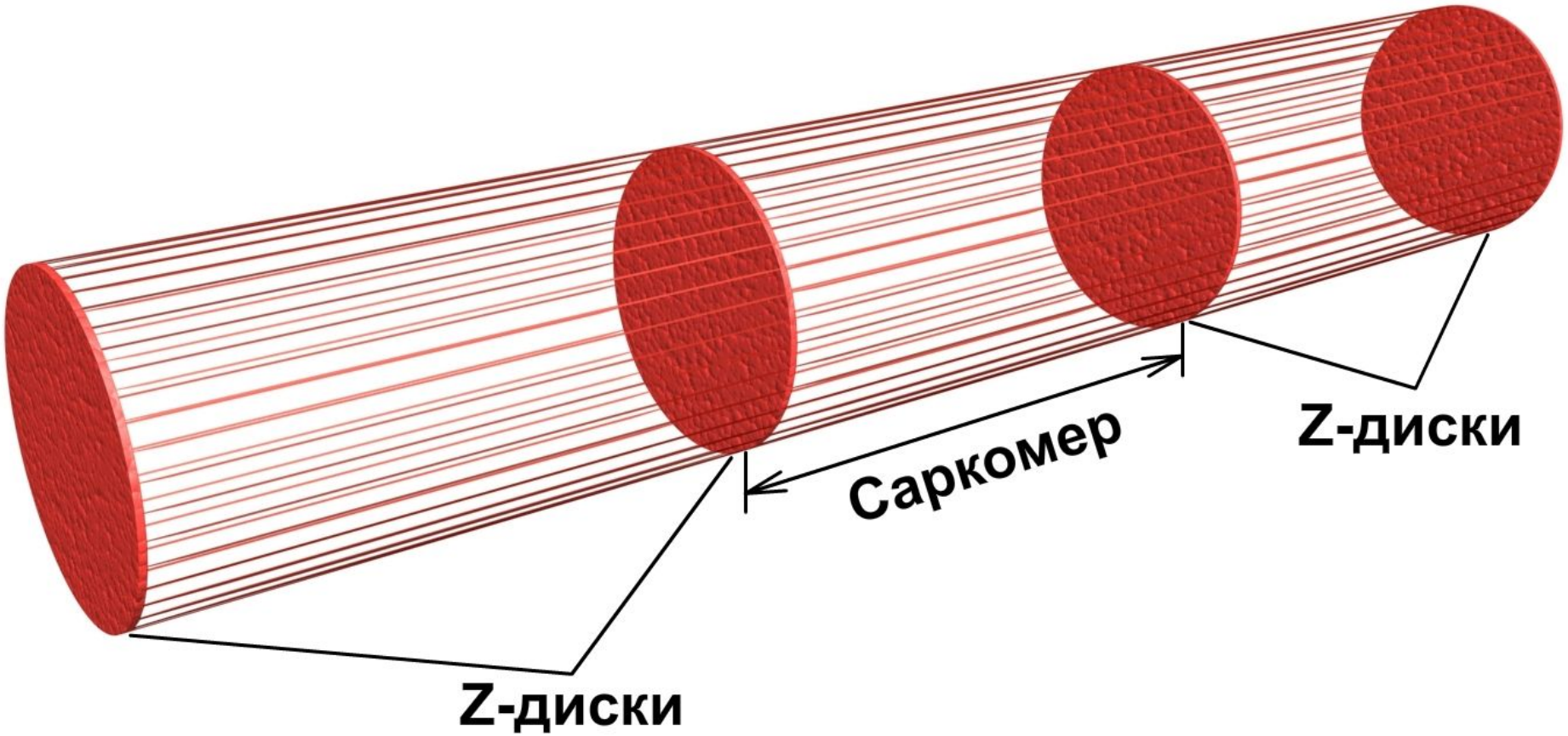
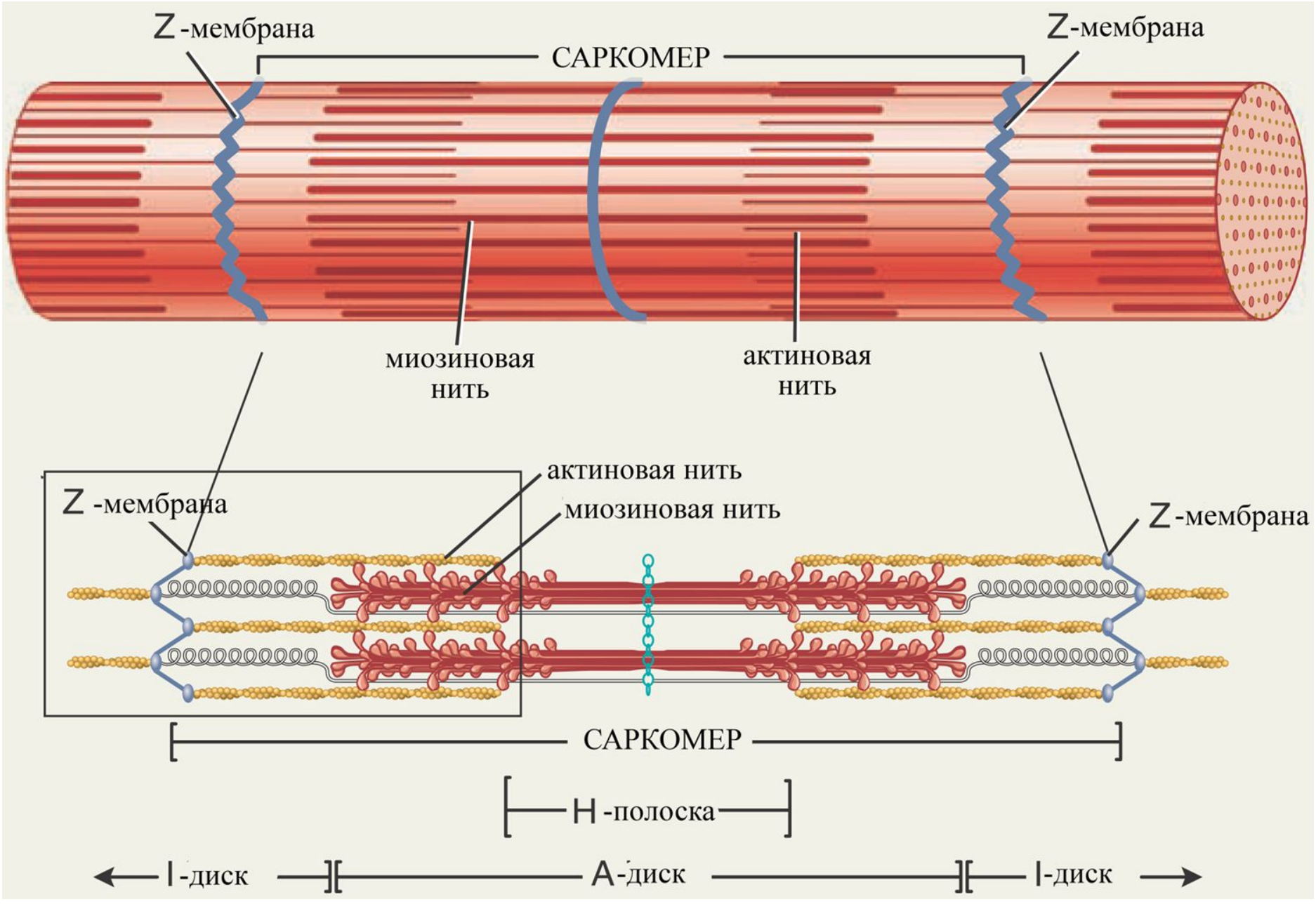
Физиология мышц. Механизм мышечного сокращения

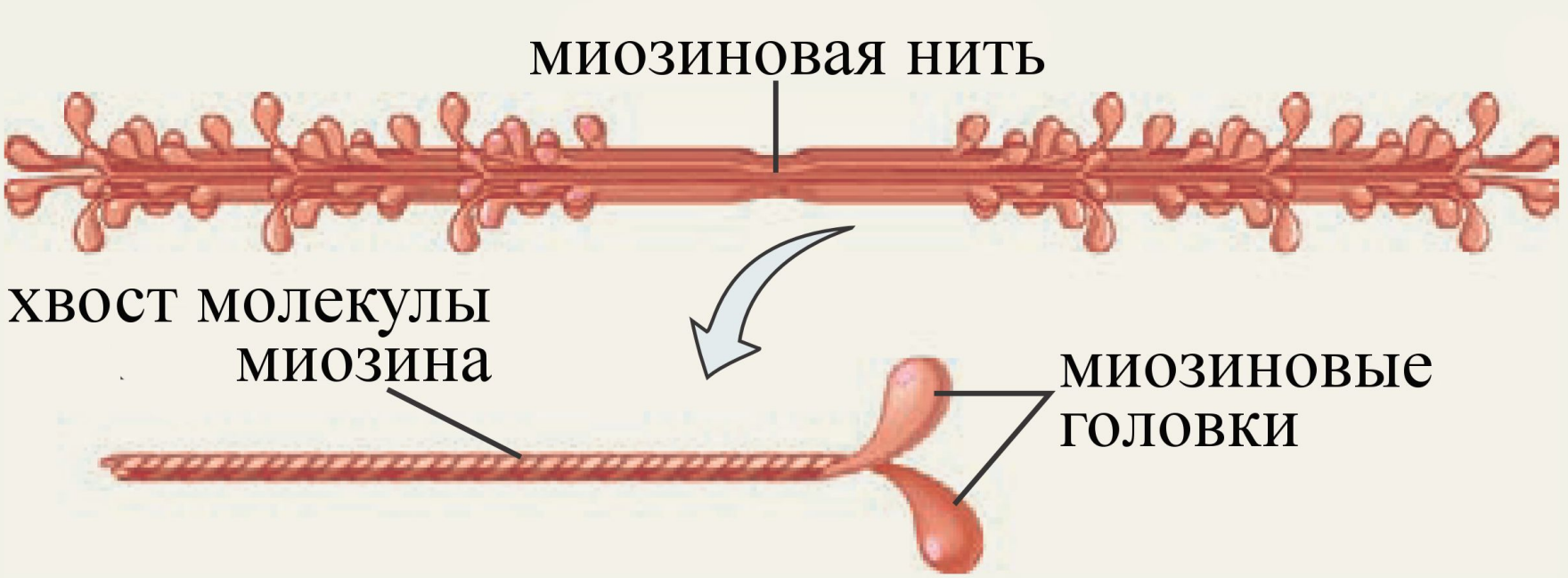

Сухожилие
Скелетная мышца
Мышечное волокно
Ядра
Плазматическая мембрана
Клетки
Микроволокна


саркоплазматический ретикулум
сарколемма
миофибрилла
саркоплазма
ядро
нить миозина
нить актина
Т-трубочка
L-цистерна
митохондрия
саркомер
Z мембрана

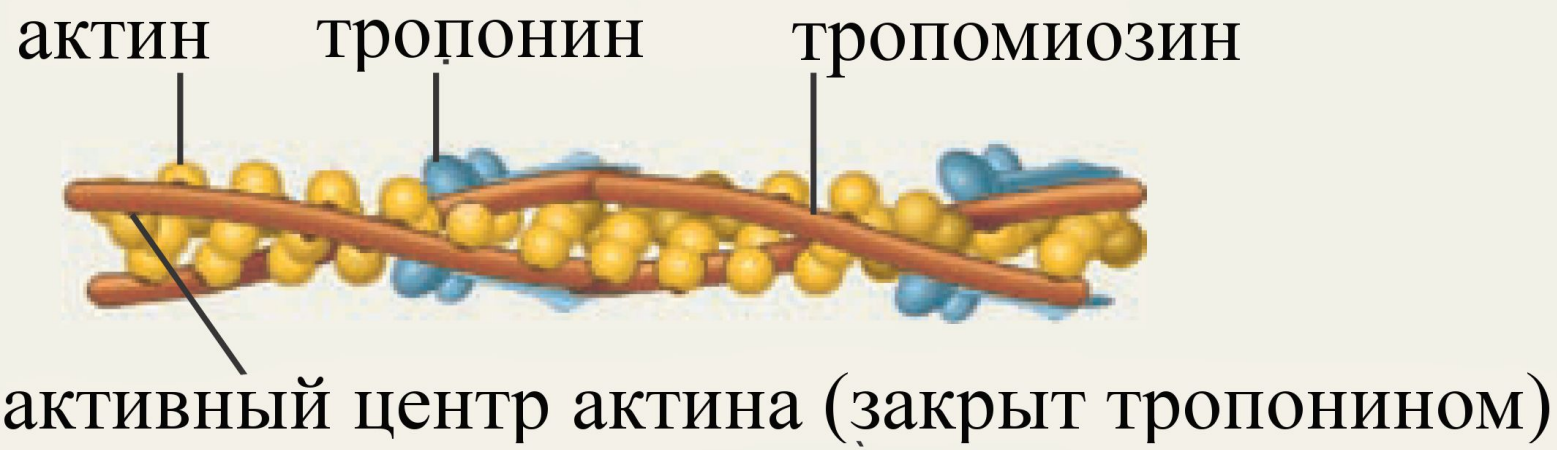

I – диск
A – диск
I – диск
А
Б
Z – пластинка
Z – пластинка
т – нить (актин)
А – нить (миозин)
Толстая нить, состоящая из миозиновых молекул
Поперечные мостики между актиновыми и миозиновыми нитями
Тонкая нить, состоящая из актиновых молекул
Н – диск (Н – зона)
В
Г


Саркомер
Z-диски
Z-диски

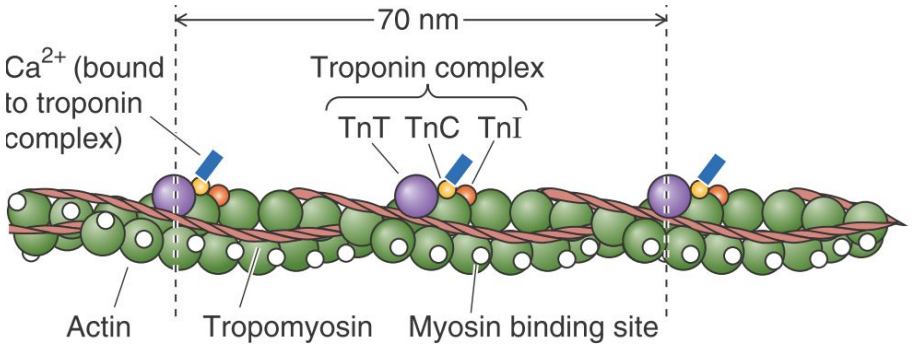

Z -мембрана
САРКОМЕР
Z-мембрана
миозиновая нить
актиновая нить
актиновая нить
миозиновая нить
Z -мембрана
Z-мембрана
САРКОМЕР
H -полоска
I -диск
A-диск
I-диск



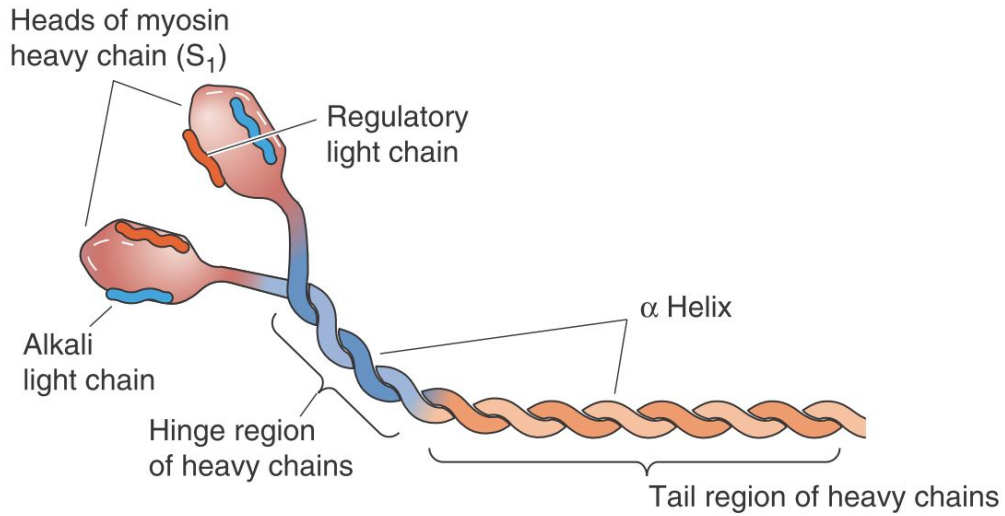
миозиновая нить и молекула миозина



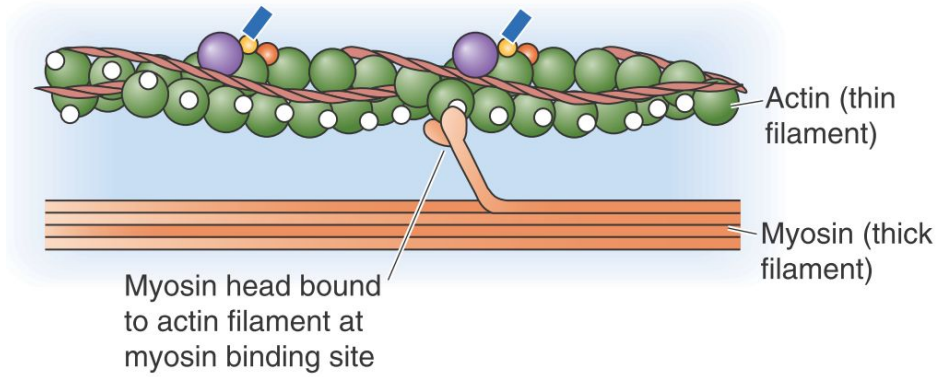
участок актиновой нити

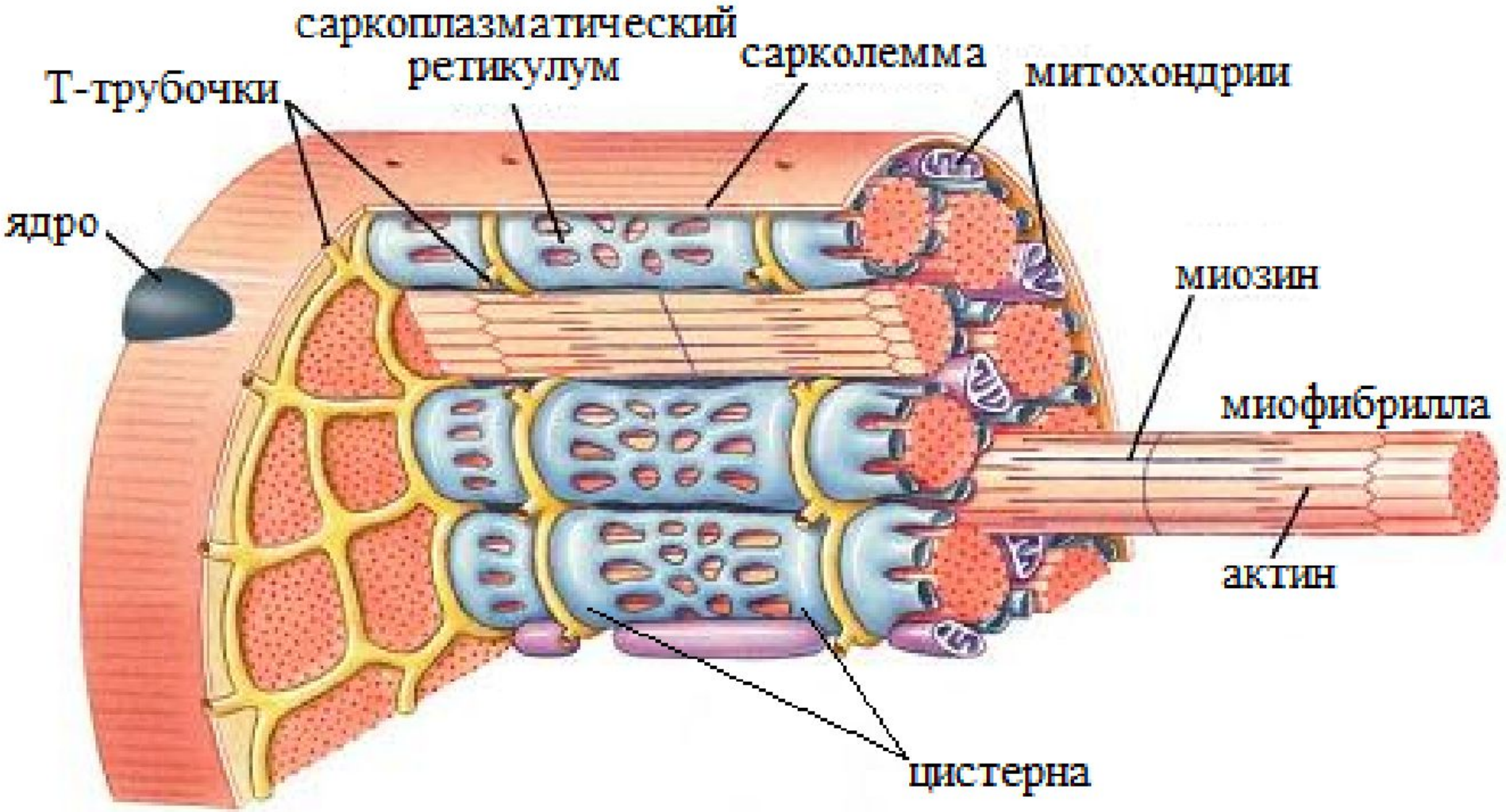


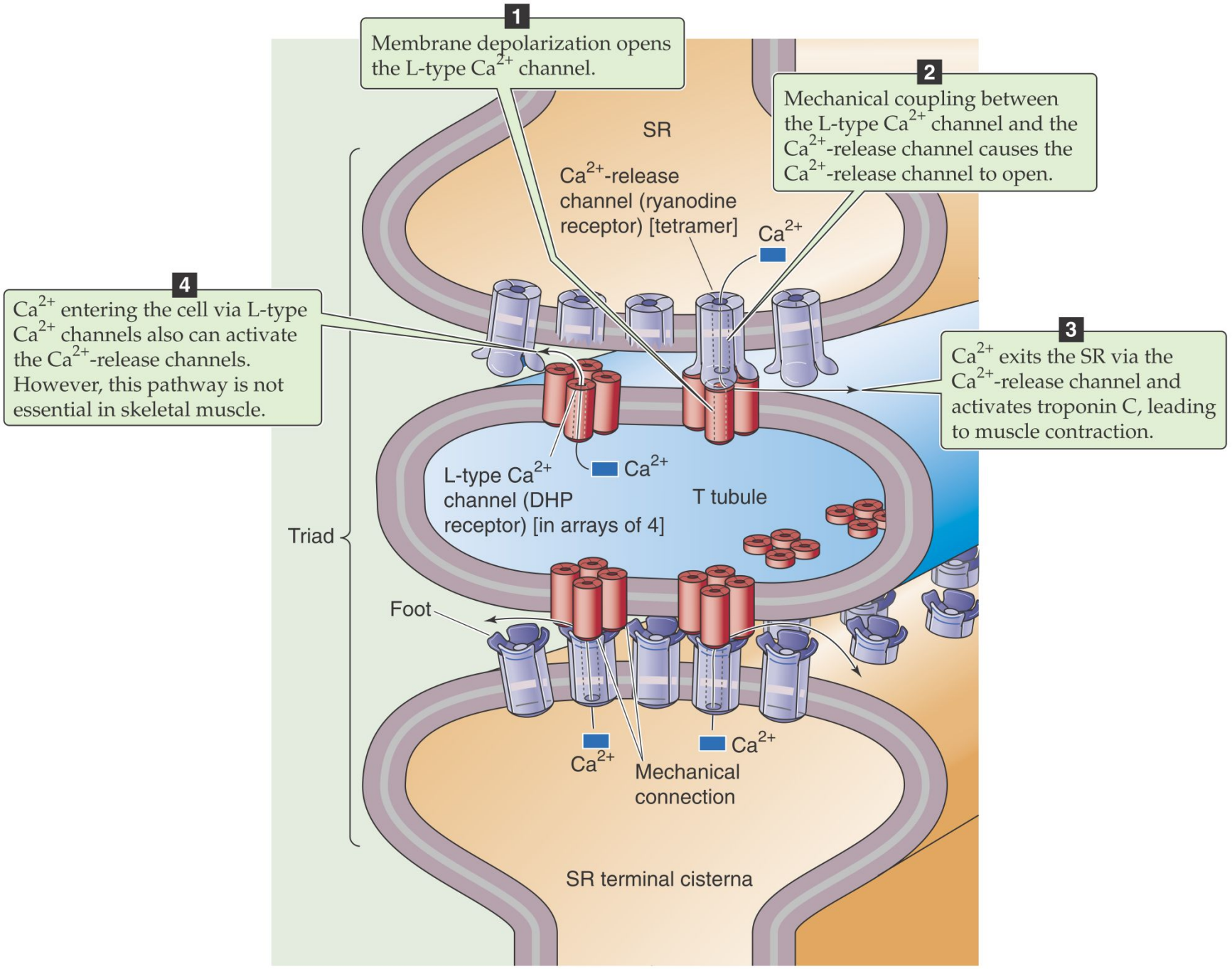
B MYOSIN MOLECULE

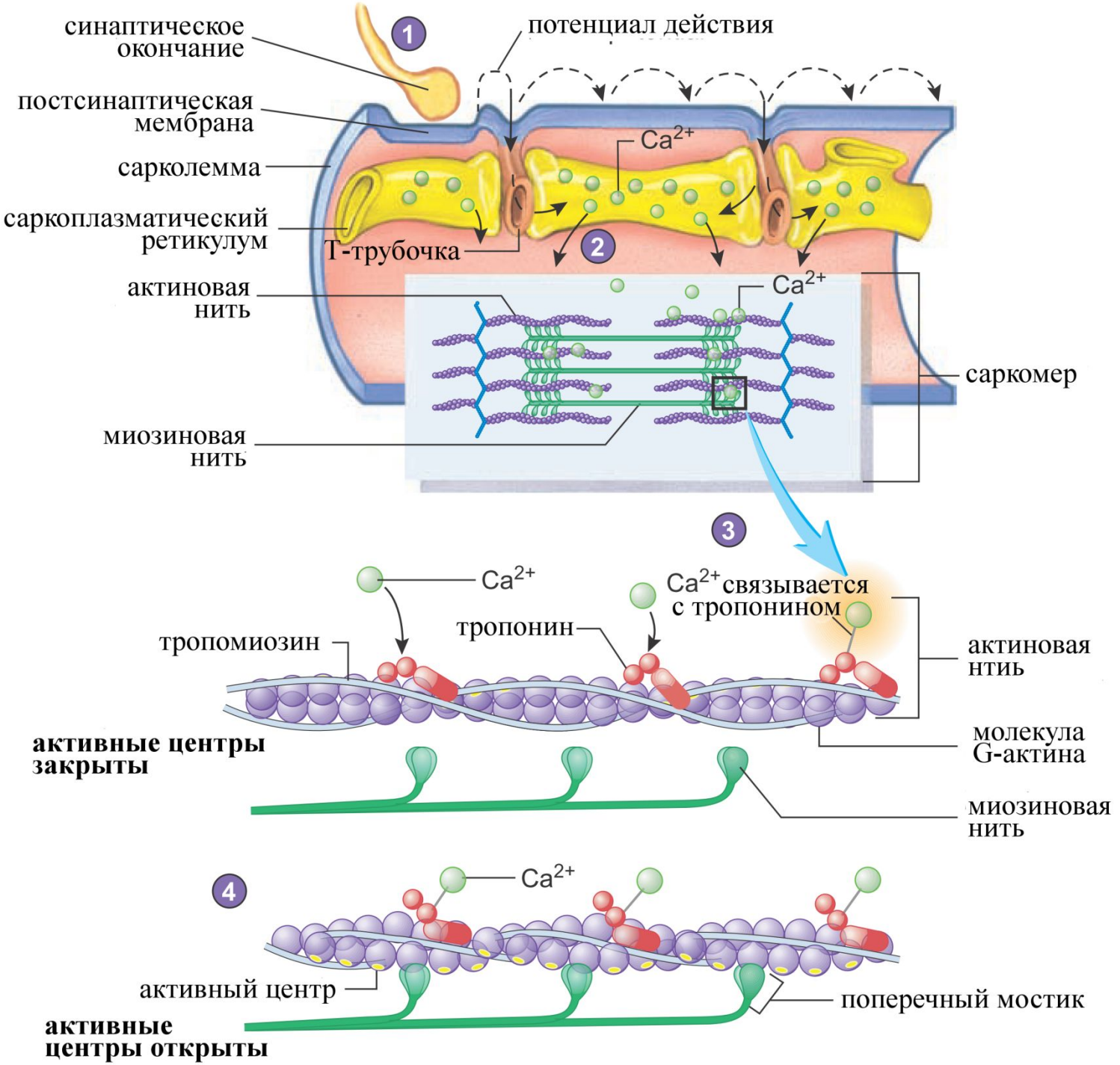


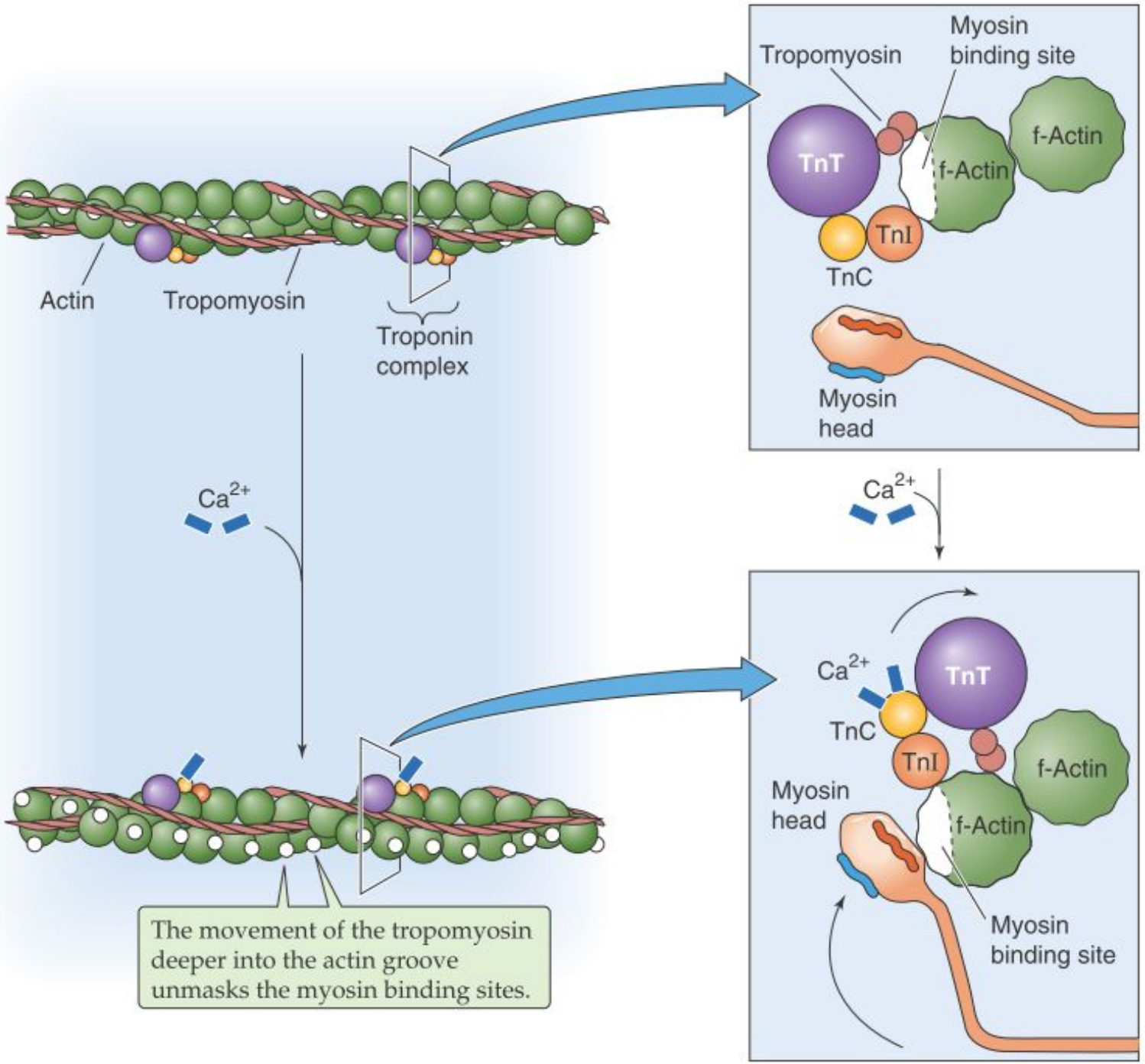
C INTERACTION OF THIN AND THICK FILAMENTS

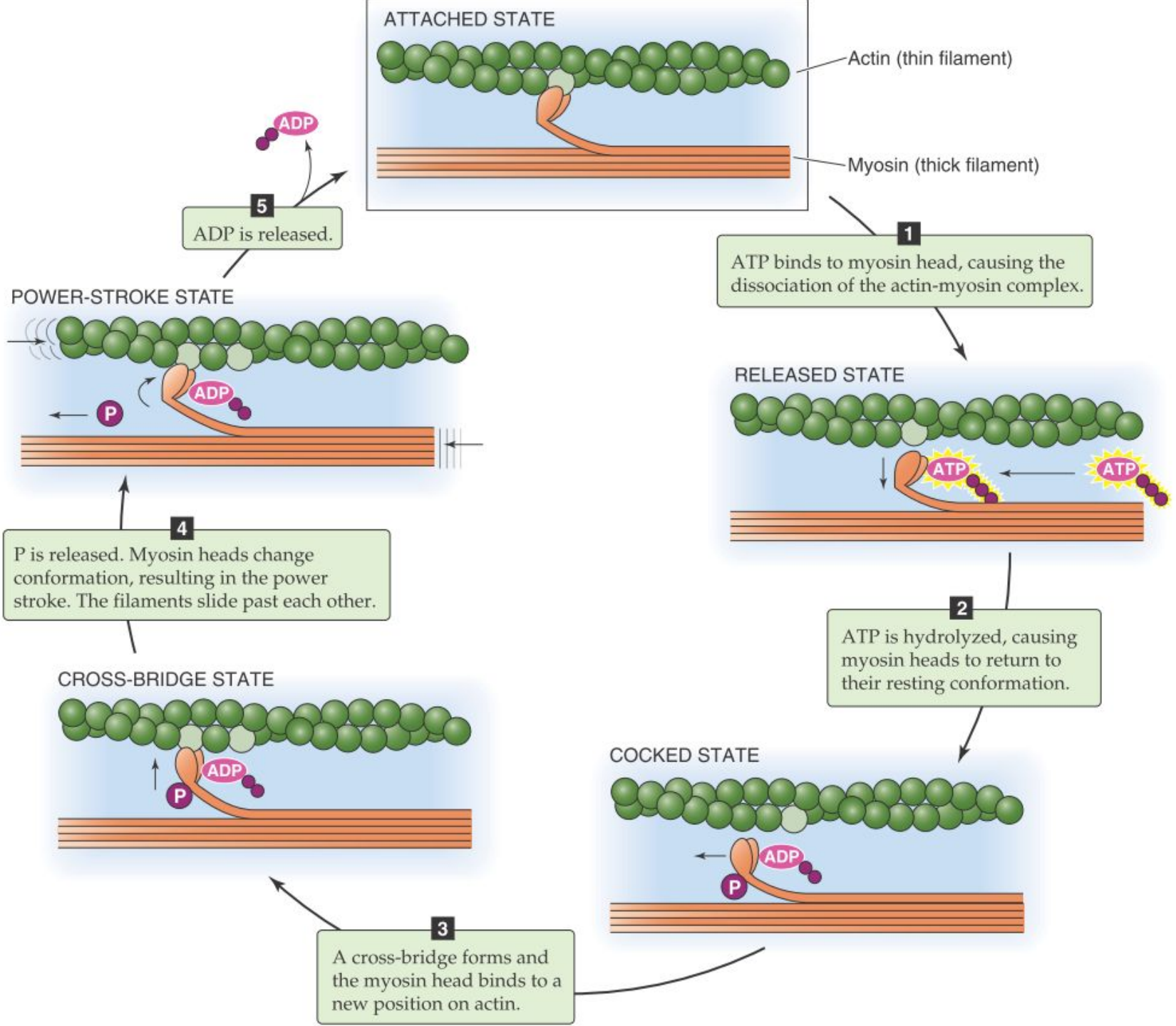


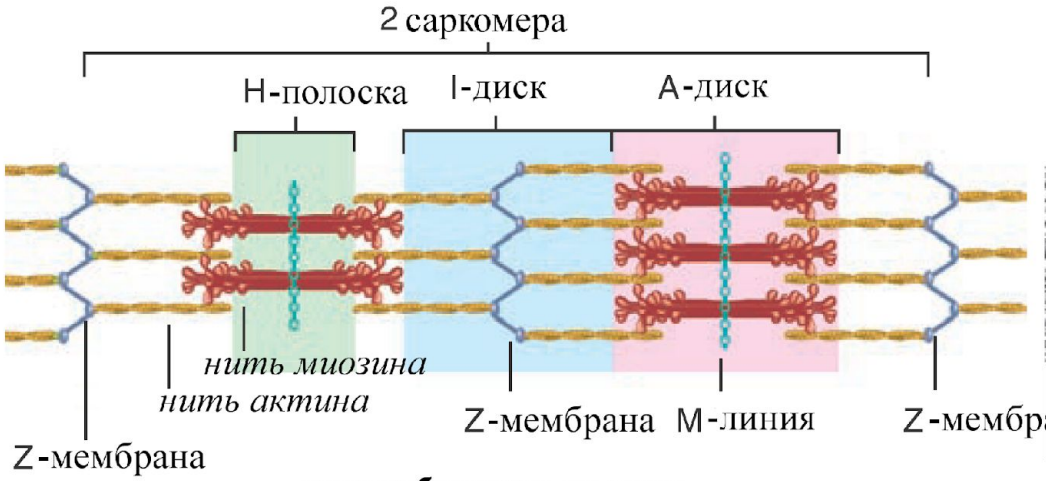

саркоплазматический ретикулум
сарколемма
митохондрии
Т-трубочки
ядро
миозин
миофибрилла
актин
цистерна

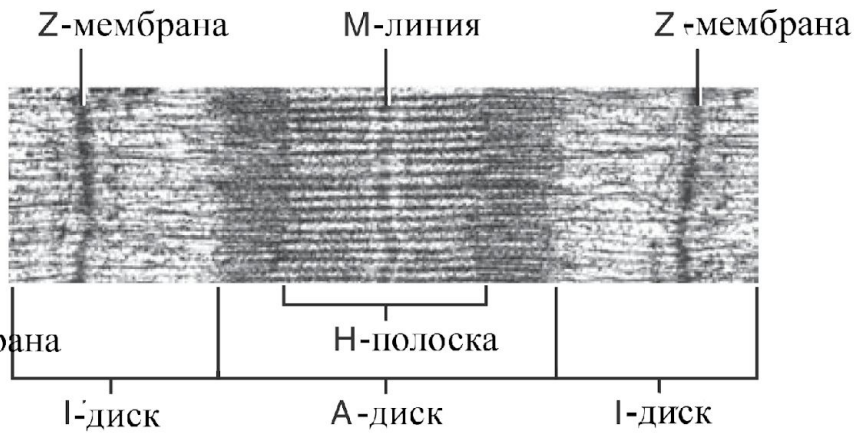

1
Membrane depolarization opens the L-type Ca^{2+} channel.
2
Mechanical coupling between the L-type Ca^{2+} channel and the Ca^{2+}-release channel causes the Ca^{2+}-release channel to open.
3
Ca^{2+} exits the SR via the Ca^{2+}-release channel and activates troponin C, leading to muscle contraction.
4
Ca^{2+} entering the cell via L-type Ca^{2+} channels also can activate the Ca^{2+}-release channels. However, this pathway is not essential in skeletal muscle.
SR
Ca^{2+}-release channel (ryanodine receptor) [tetramer]
Ca^{2+}
L-type Ca^{2+} channel (DHP receptor) [in arrays of 4]
Ca^{2+}
T tubule
Triad
Foot
Ca^{2+}
Ca^{2+}
Mechanical connection
SR terminal cisterna


синаптическое окончание
потенциал действия
постсинаптическая мембрана
сарколемма
саркоплазматический ретикулум
Т-трубочка
актиновая нить
миозиновая нить
саркомер
Ca2+
1
2
3
4
тропомиозин
тропонин
Ca2+ связывается с тропонином
актиновая нтиь
молекула G-актина
миозиновая нить
активные центры закрыты
активный центр
поперечный мостик
активные центры открыты

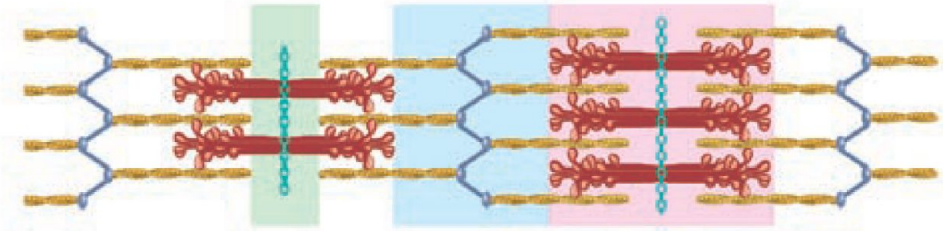

Actin
Tropomyosin
Troponin complex
Tropomyosin
Myosin binding site
TnT
f-Actin
f-Actin
TnI
TnC
Myosin head
Ca2+
Ca2+
Ca2+
TnT
TnC
TnI
f-Actin
Myosin head
f-Actin
Myosin binding site
The movement of the tropomyosin deeper into the actin groove unmasks the myosin binding sites.

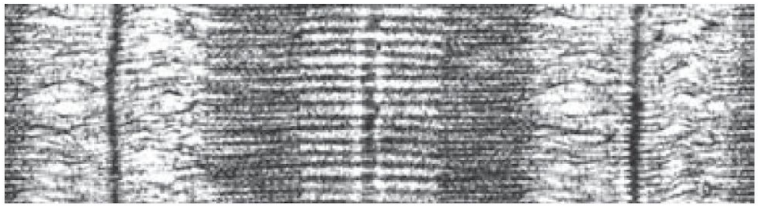

ATTACHED STATE
Actin (thin filament)
Myosin (thick filament)
1
ATP binds to myosin head, causing the dissociation of the actin-myosin complex.
RELEASED STATE
ATP
ATP
2
ATP is hydrolyzed, causing myosin heads to return to their resting conformation.
COCKED STATE
ADP
P
3
A cross-bridge forms and the myosin head binds to a new position on actin.
CROSS-BRIDGE STATE
ADP
P
4
P is released. Myosin heads change conformation, resulting in the power stroke. The filaments slide past each other.
POWER-STROKE STATE
ADP
P
5
ADP is released.
ADP



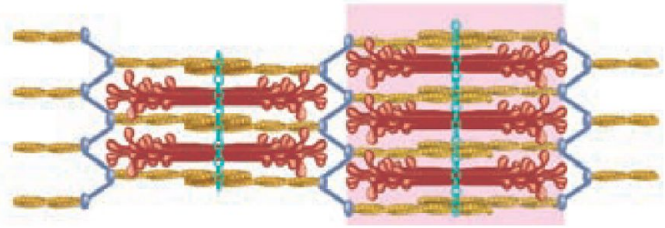


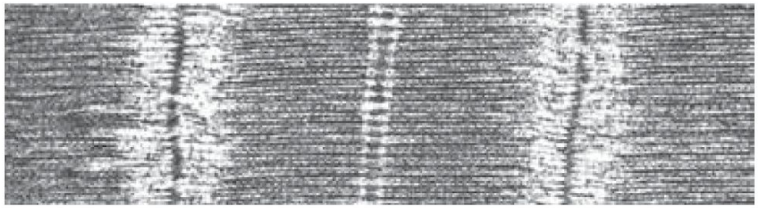
расслабленная мышца



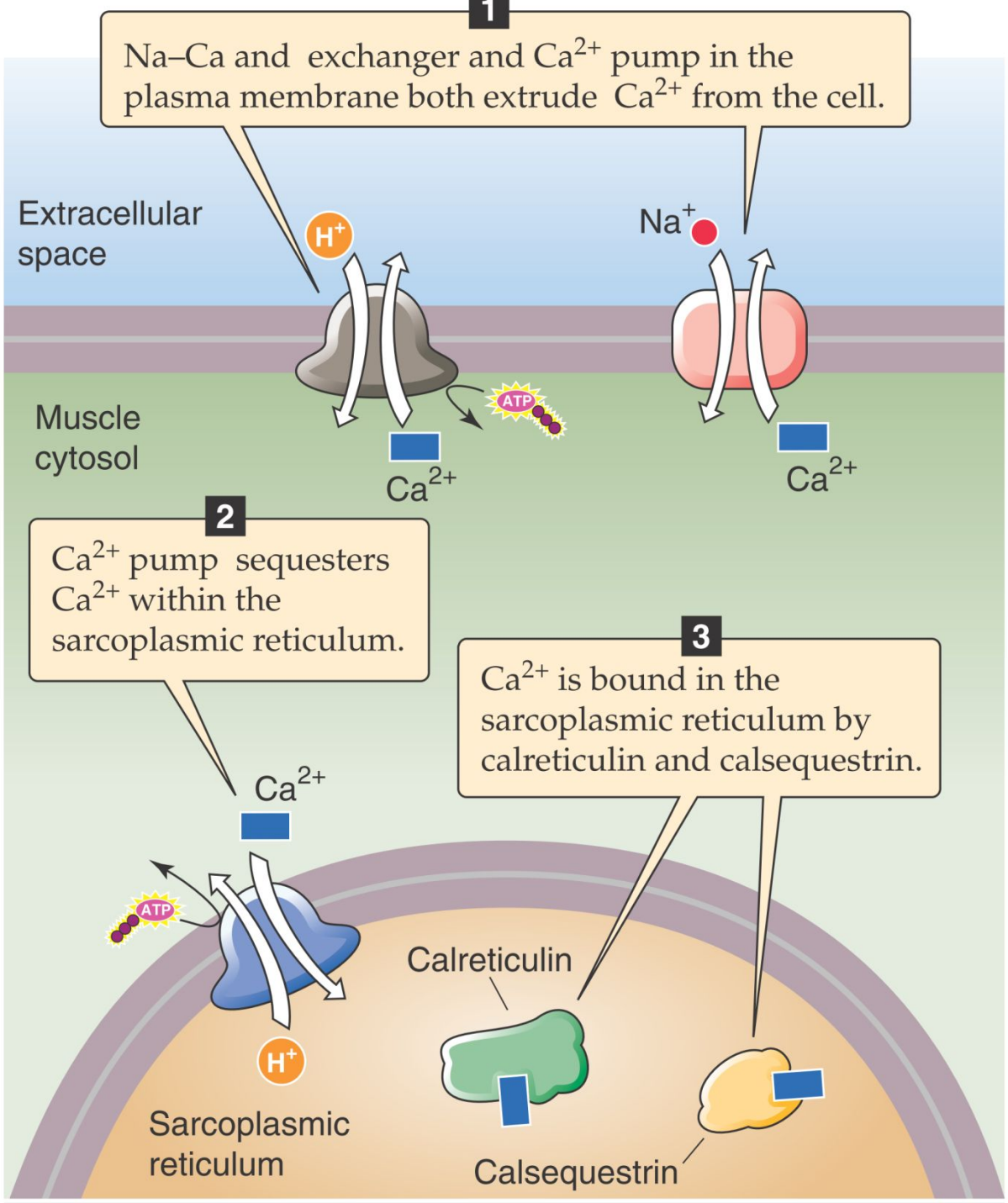


частично сокращённая мышца

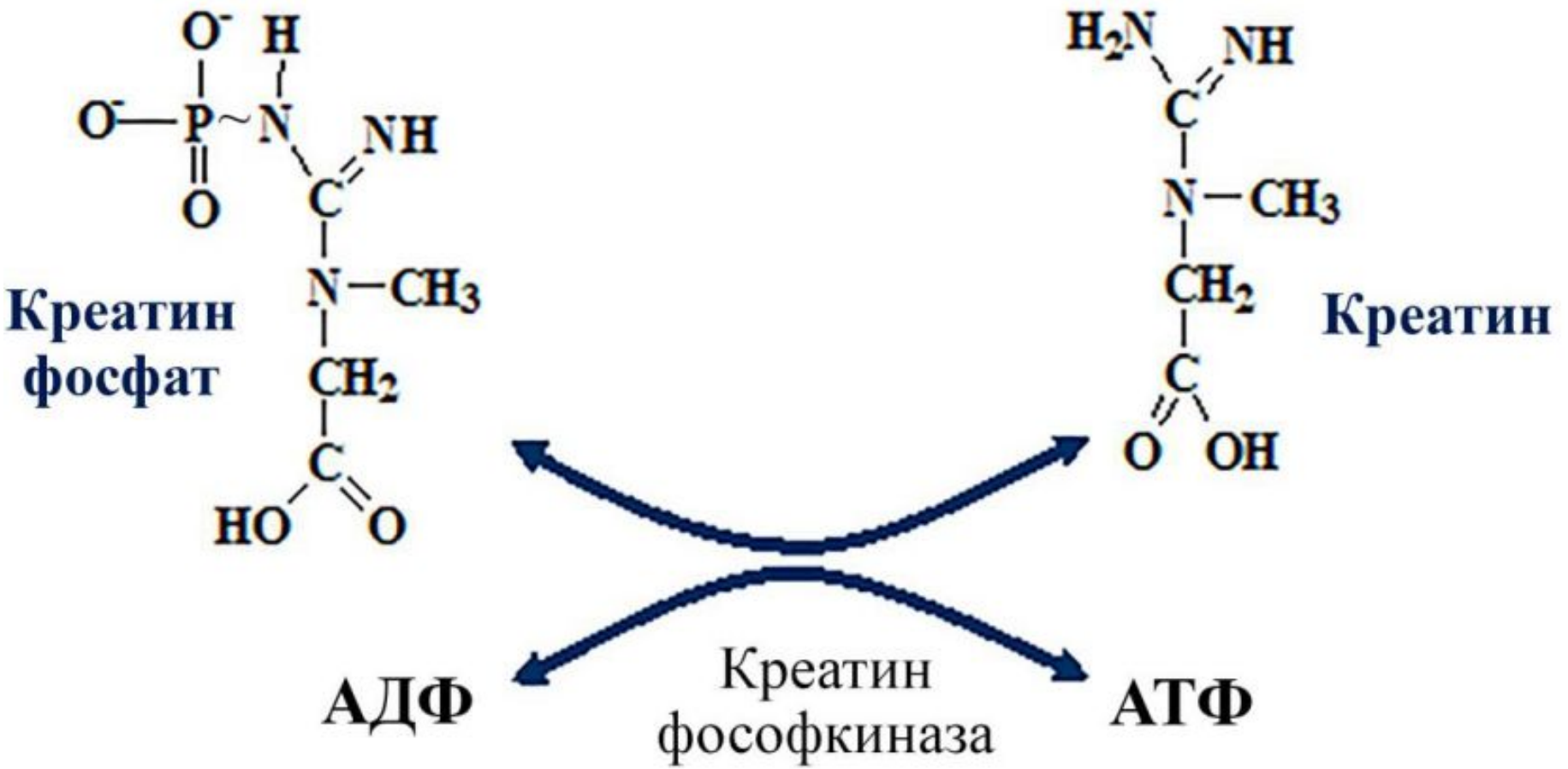


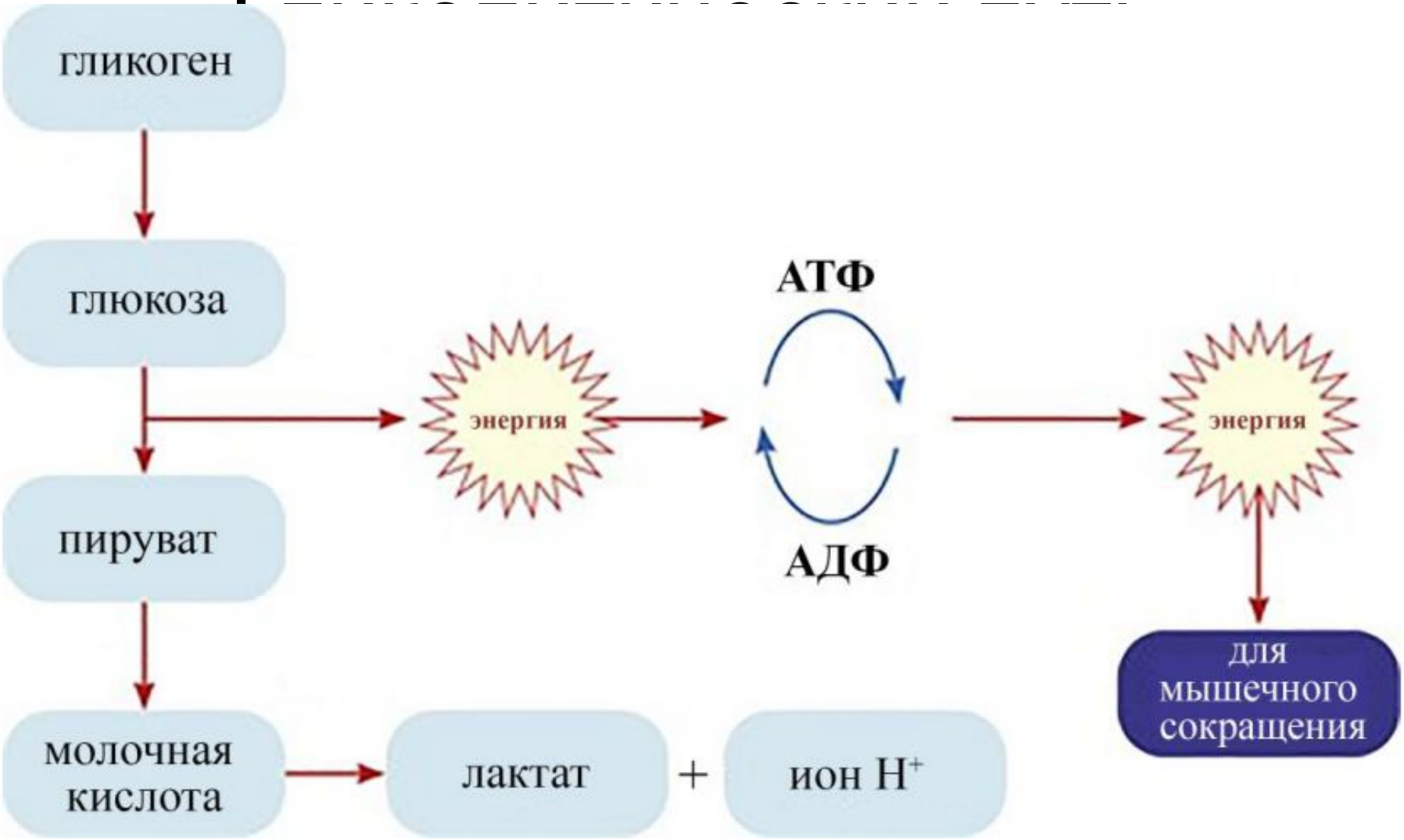


максимально сокращённая мышца

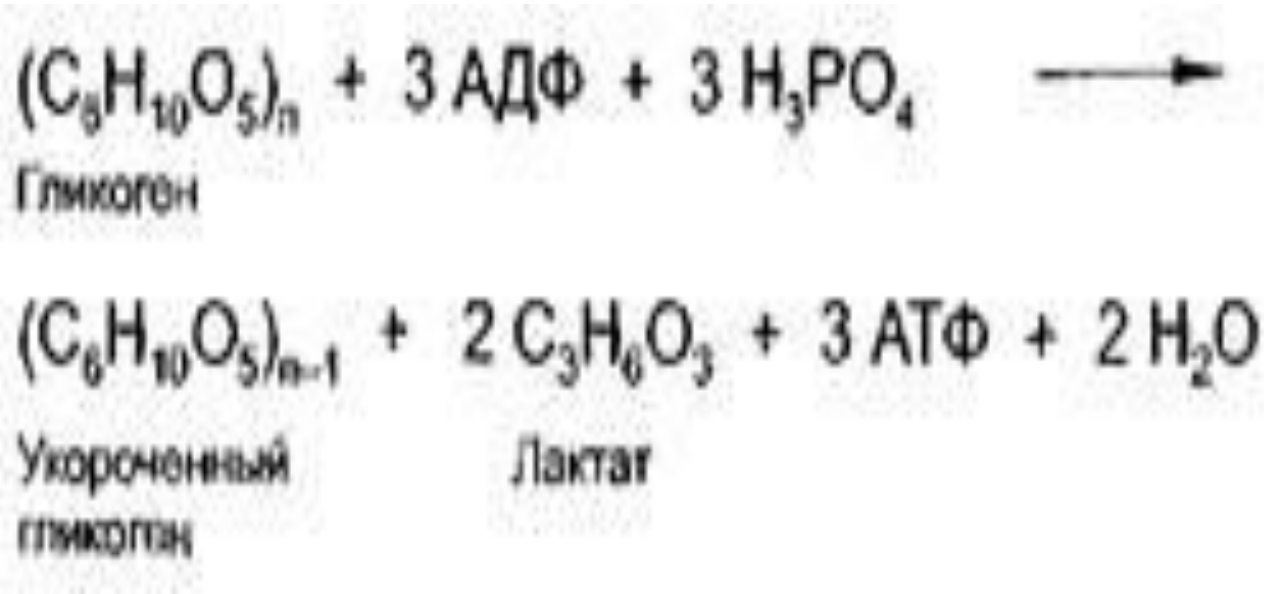

1
Na–Ca and exchanger and Ca^{2+} pump in the plasma membrane both extrude Ca^{2+} from the cell.
Extracellular space
H^+
Na^+
ATP
Muscle cytosol
Ca^{2+}
Ca^{2+}
2
Ca^{2+} pump sequesters Ca^{2+} within the sarcoplasmic reticulum.
3
Ca^{2+} is bound in the sarcoplasmic reticulum by calreticulin and calsequestrin.
Ca^{2+}
ATP
Calreticulin
H^+
Sarcoplasmic reticulum
Calsequestrin

Креатинфосфатный путь

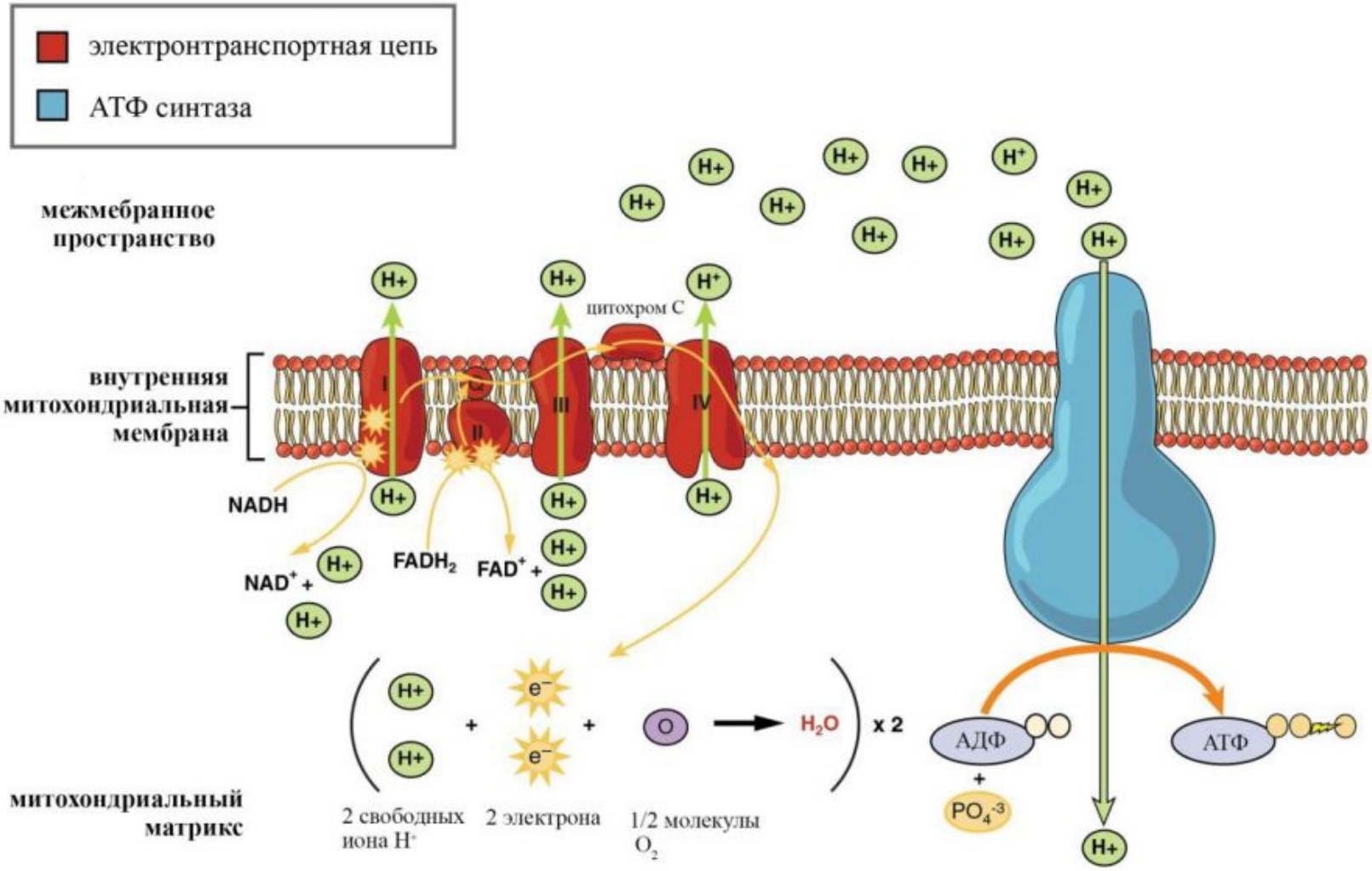


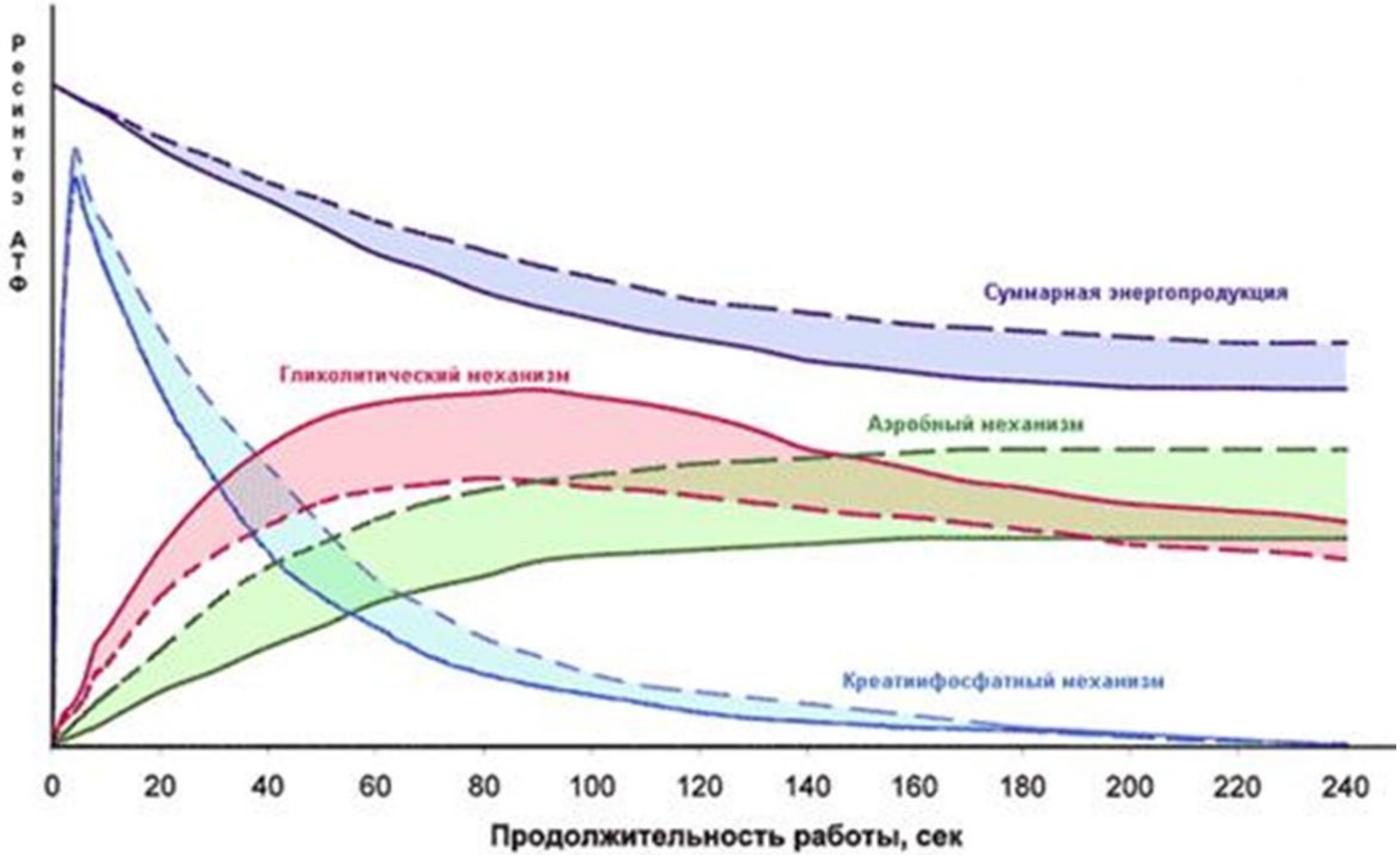

гликоген
глюкоза
пируват
молочная кислота
лактат
+
ион H^+
энергия
АТФ
АДФ
энергия
для мышечного сокращения

Гликолитический путь



Тканевое дыхание

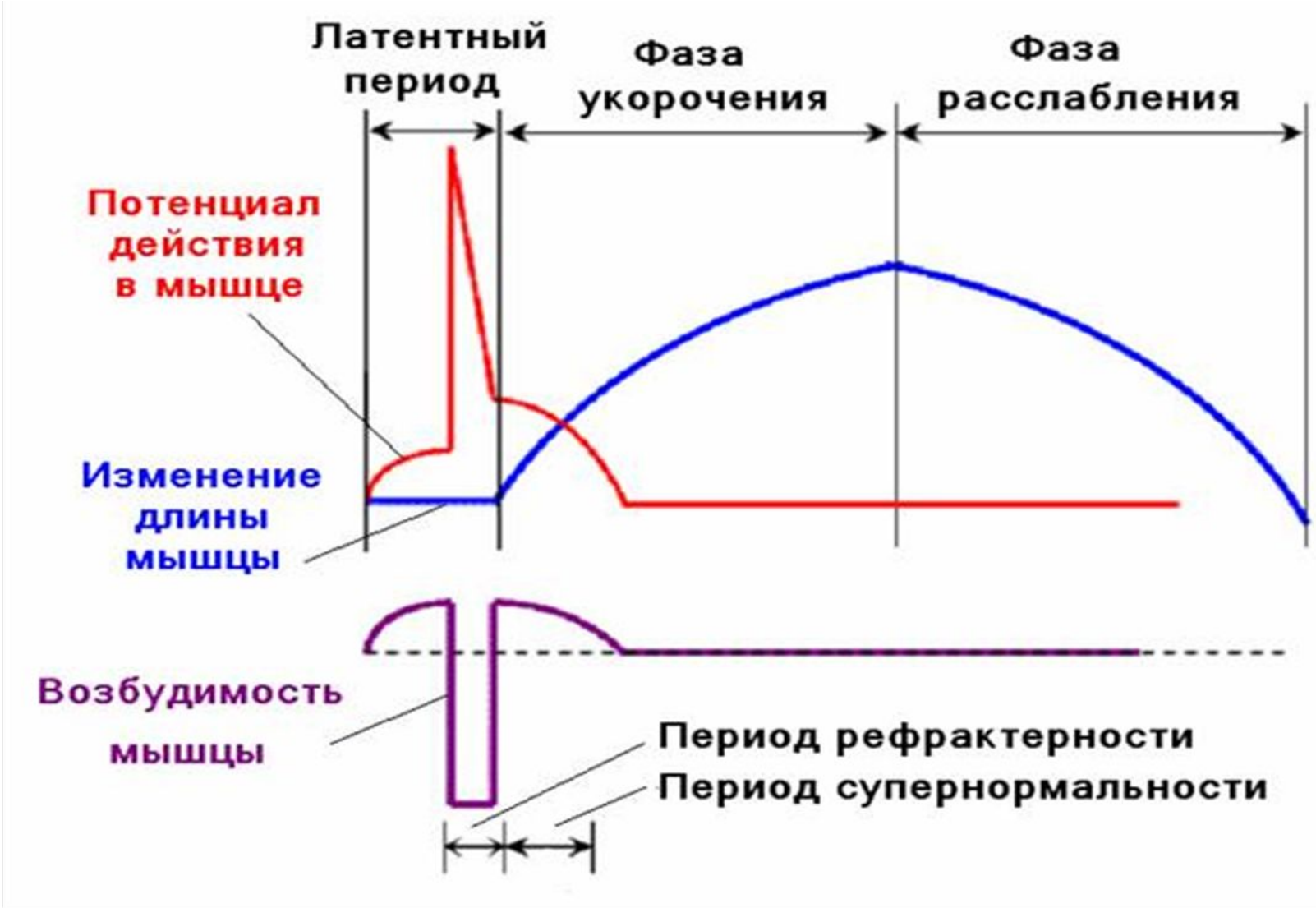


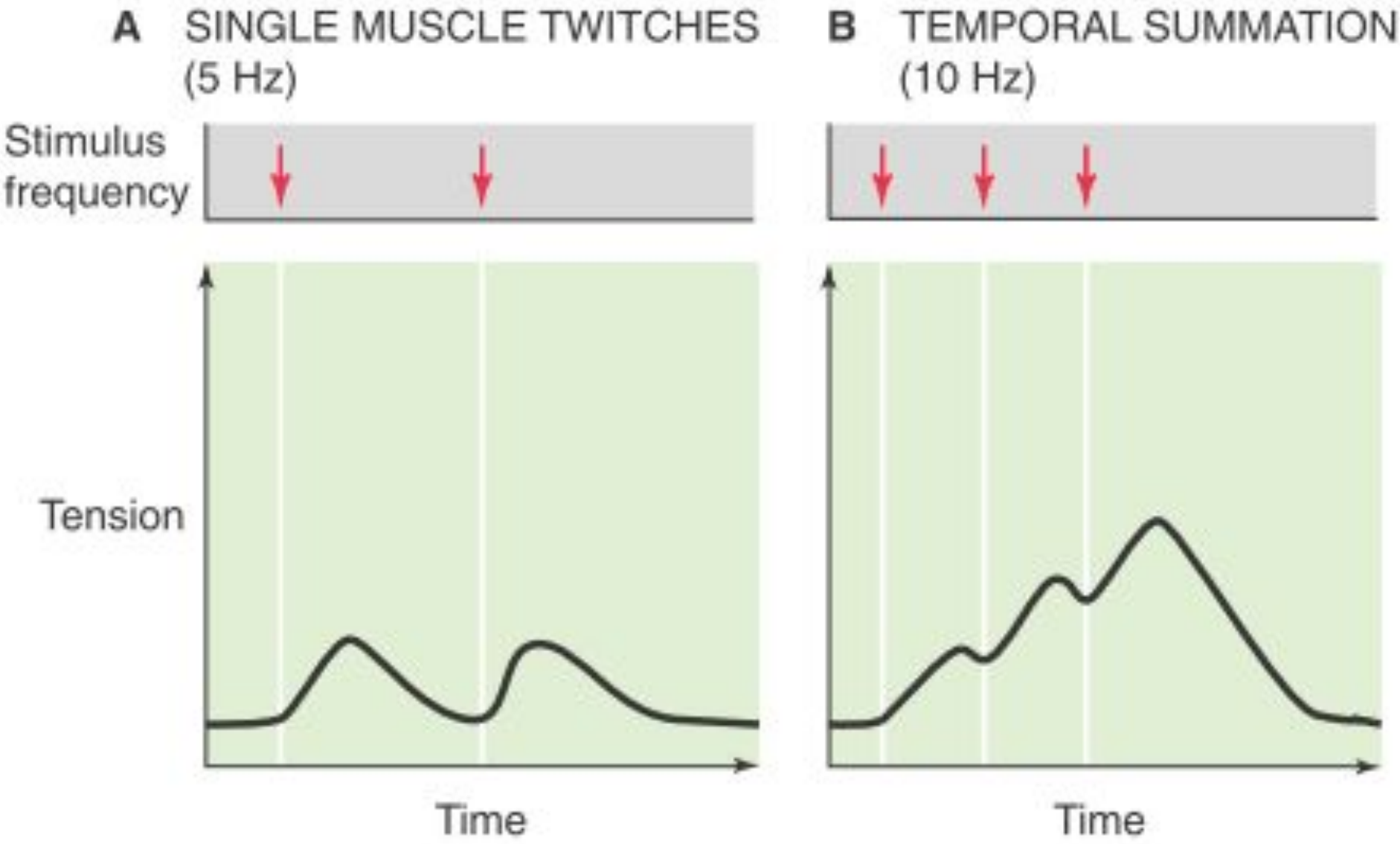

Ресинтез АТФ
Суммарная энергопродукция
Гликолитический механизм
Аэробный механизм
Креатинфосфатный механизм
0
20
40
60
80
100
120
140
160
180
200
220
240
Продолжительность работы, сек

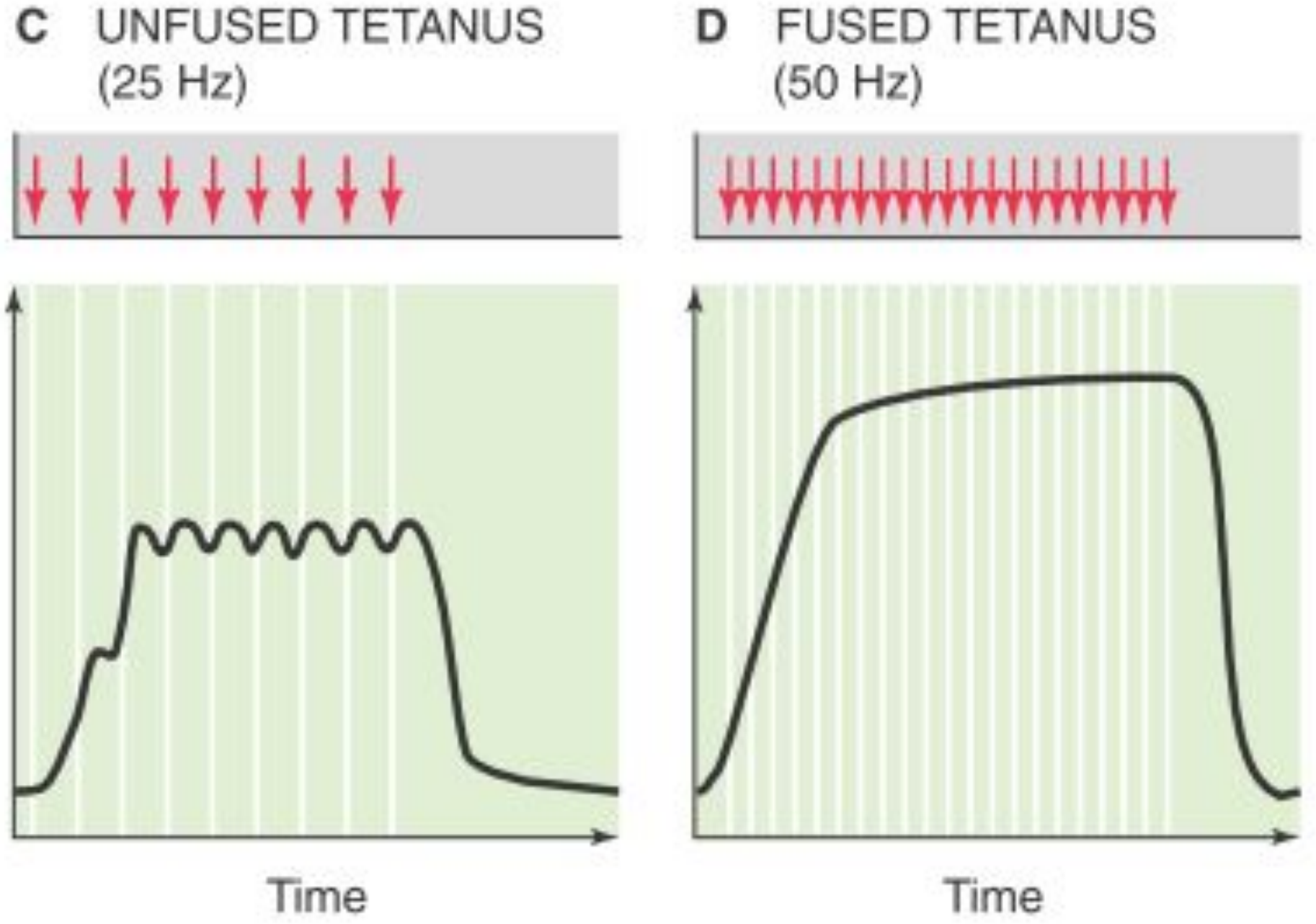

Красные
высокая митохондриальная составляющая
Промежут-ые
средняя МС
Белые
низкая МС

	Slow Twitch	Fast Twitch	Fast Twitch
Synonym	Type I	Type IIa	Type IIb
Fatigue	Resistant	Resistant	Fatigable
Color	Red (myoglobin)	Red (myoglobin)	White (low myoglobin)
Metabolism	Oxidative	Oxidative	Glycolytic
Mitochondria	High	Higher	Fewer
Glycogen	Low	Abundant	High

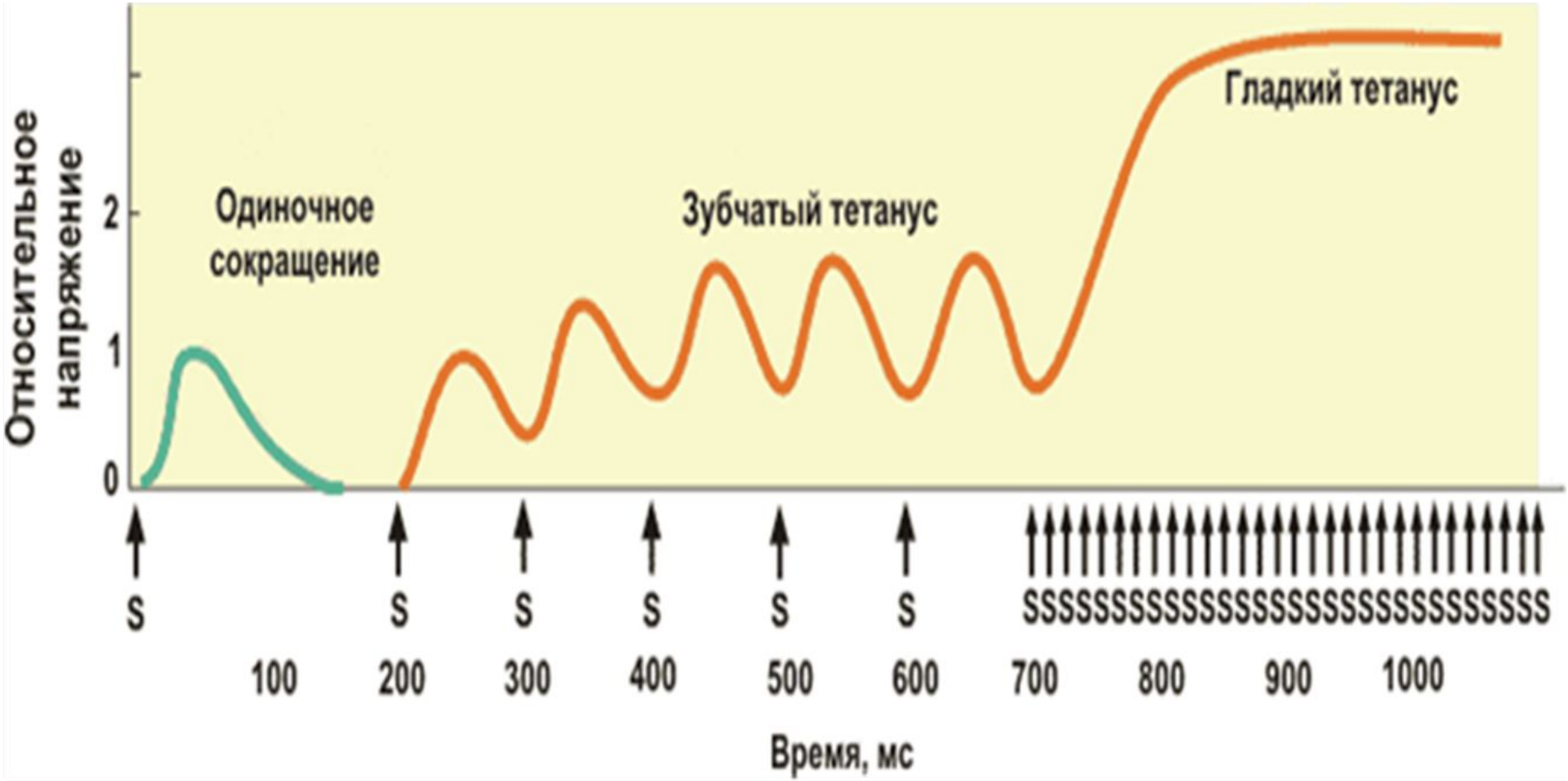
Одиночное сокращение

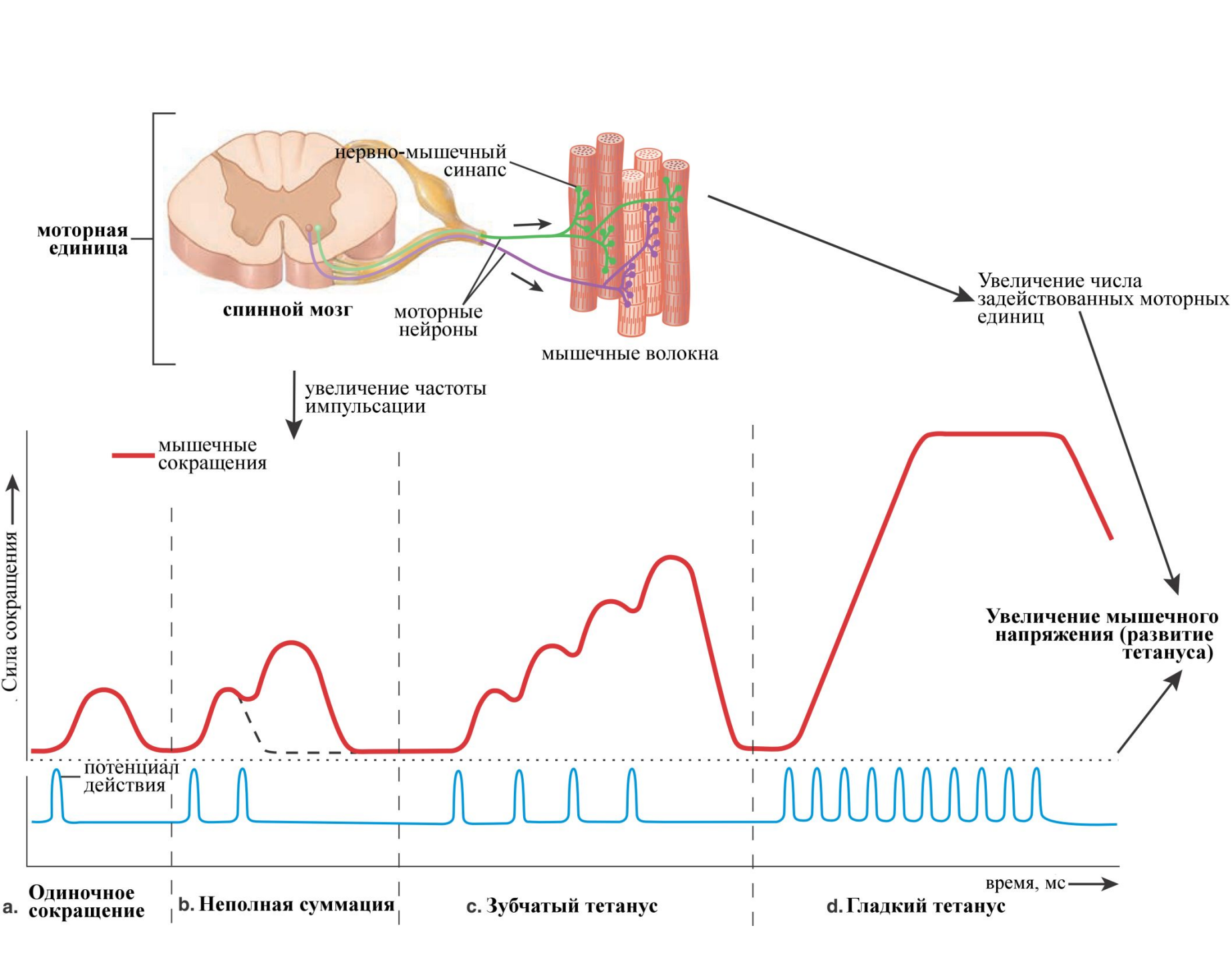


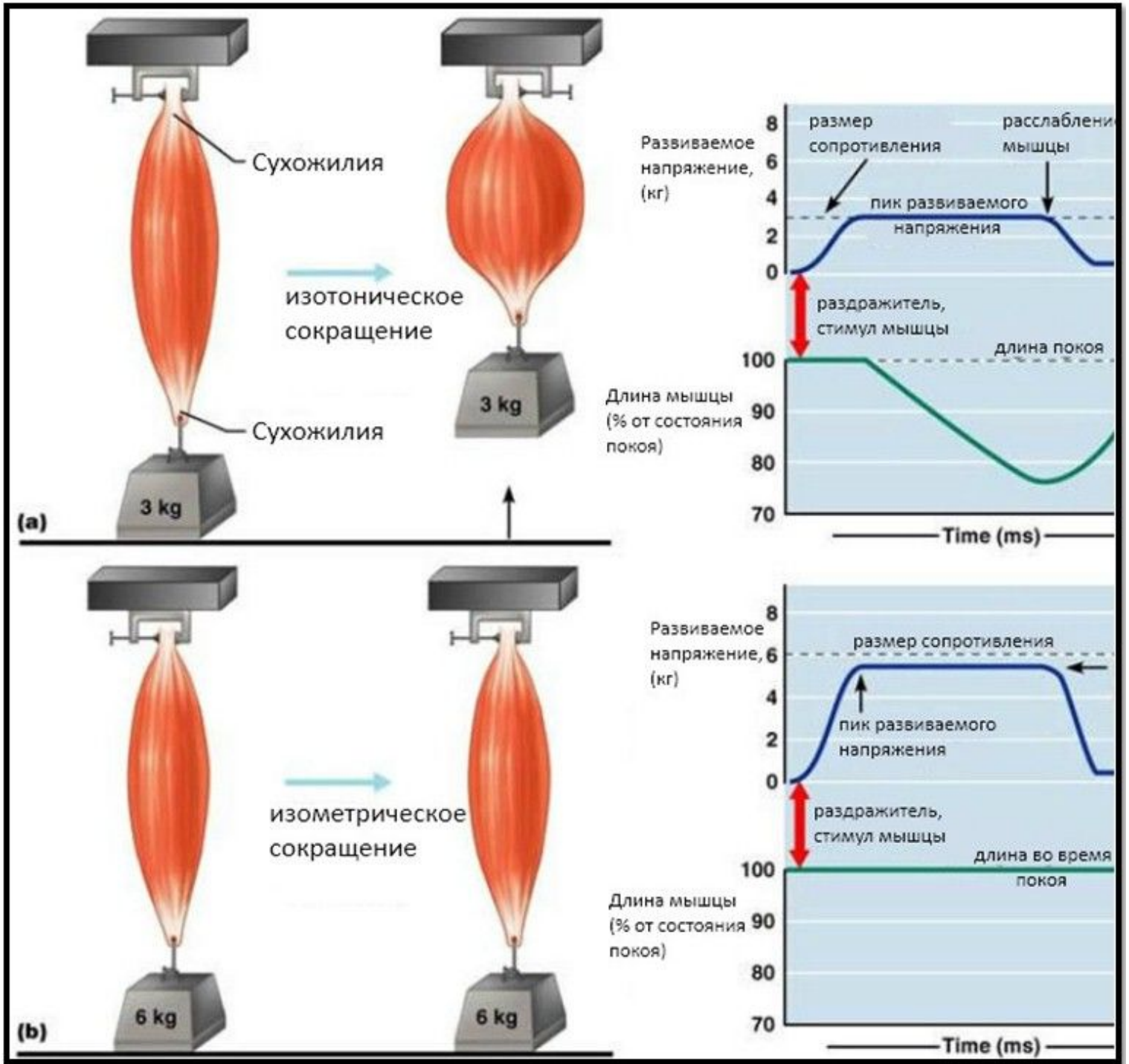

A SINGLE MUSCLE TWITCHES
(5 Hz)
B TEMPORAL SUMMATION
(10 Hz)
Stimulus frequency
Tension
Time
Time

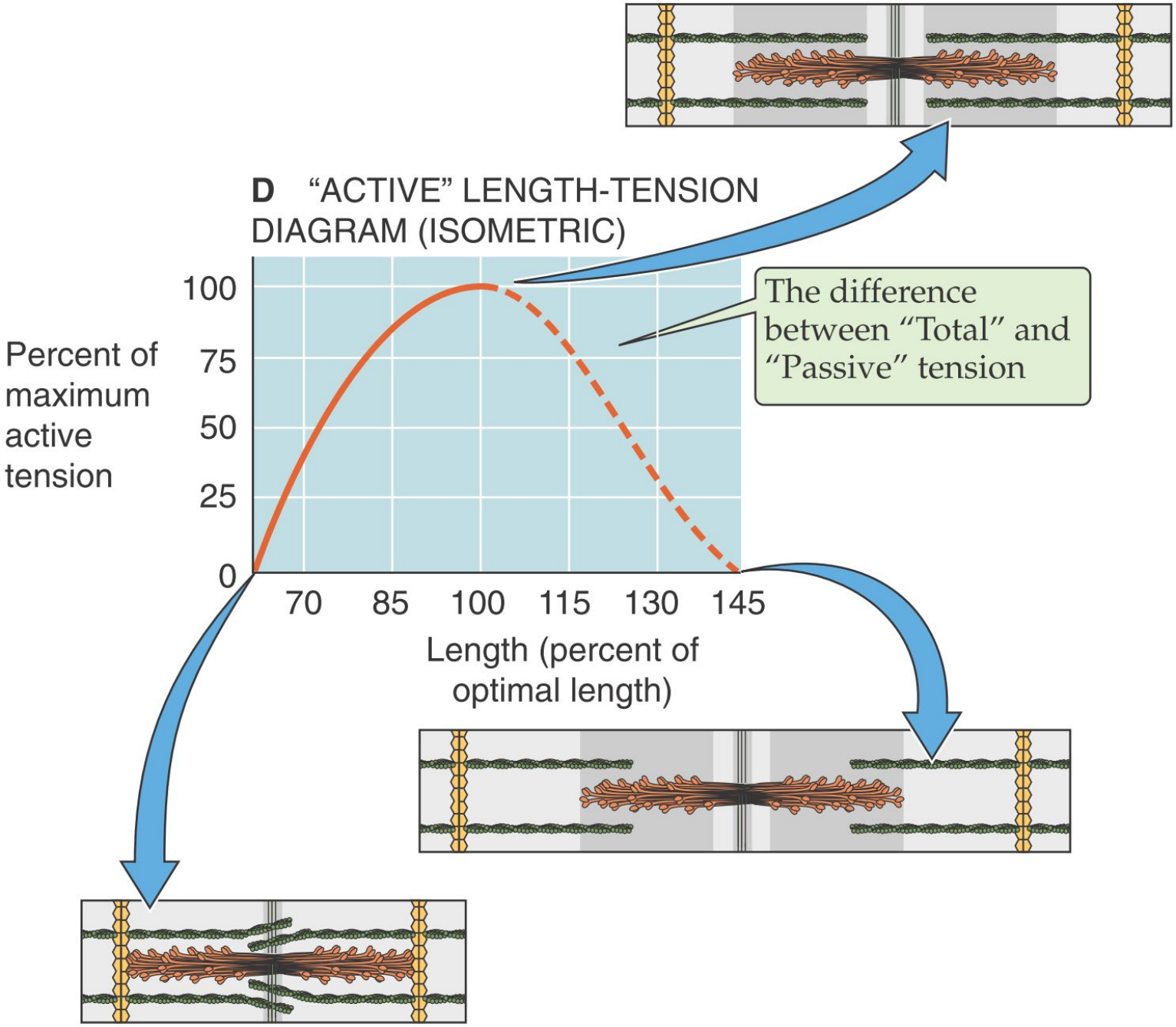

C UNFUSED TETANUS
(25 Hz)
D FUSED TETANUS
(50 Hz)
Time
Time

Тетаническое сокращение

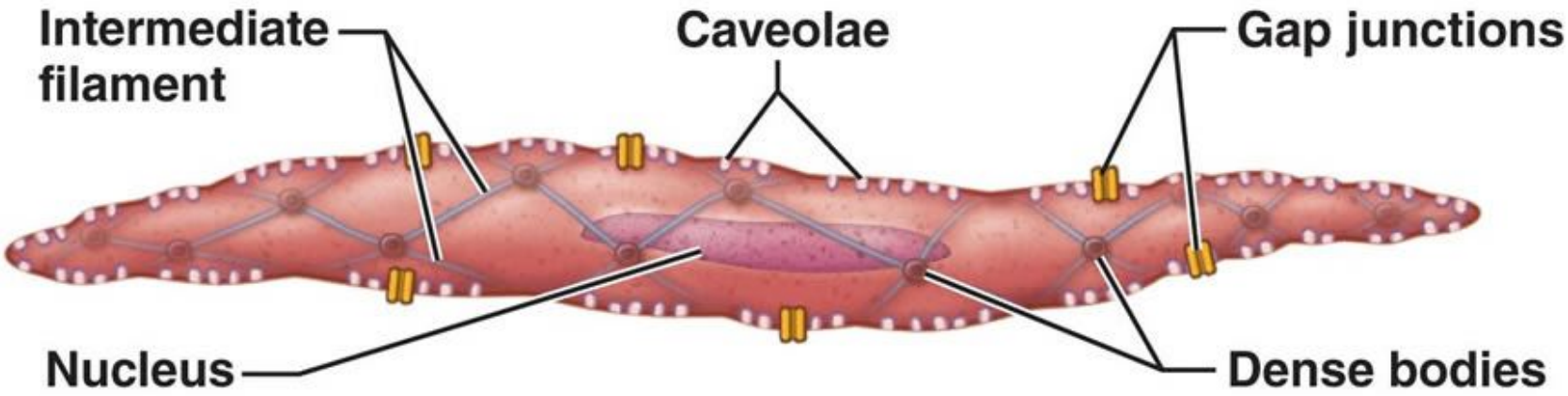



моторная единица
нервно-мышечный синапс
спинной мозг
моторные нейроны
мышечные волокна
Увеличение числа задействованных моторных единиц
увеличение частоты импульсации
мышечные сокращения
Сила сокращения
потенциал действия
Увеличение мышечного напряжения (развитие тетануса)
время, мс
a. Одиночное сокращение
b. Неполная суммация
c. Зубчатый тетанус
d. Гладкий тетанус

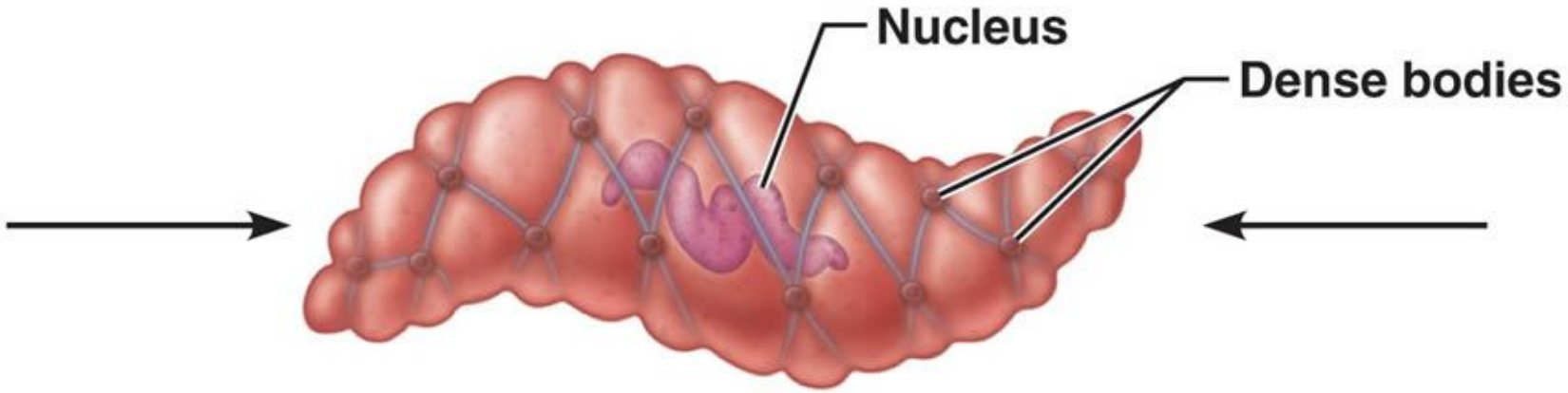

Сухожилия
изотоническое сокращение
3 kg
Сухожилия
3 kg
(a)
Развиваемое напряжение, (кг)
размер сопротивления
расслабление мышцы
пик развиваемого напряжения
раздражитель, стимул мышцы
длина покоя
Длина мышцы (% от состояния покоя)
Time (ms)
изометрическое сокращение
6 kg
6 kg
(b)
Развиваемое напряжение, (кг)
размер сопротивления
пик развиваемого напряжения
раздражитель, стимул мышцы
длина во время покоя
Длина мышцы (% от состояния покоя)
Time (ms)


D "ACTIVE" LENGTH-TENSION DIAGRAM (ISOMETRIC)
Percent of maximum active tension
100
75
50
25
0
70
85
100
115
130
145
Length (percent of optimal length)
The difference between "Total" and "Passive" tension

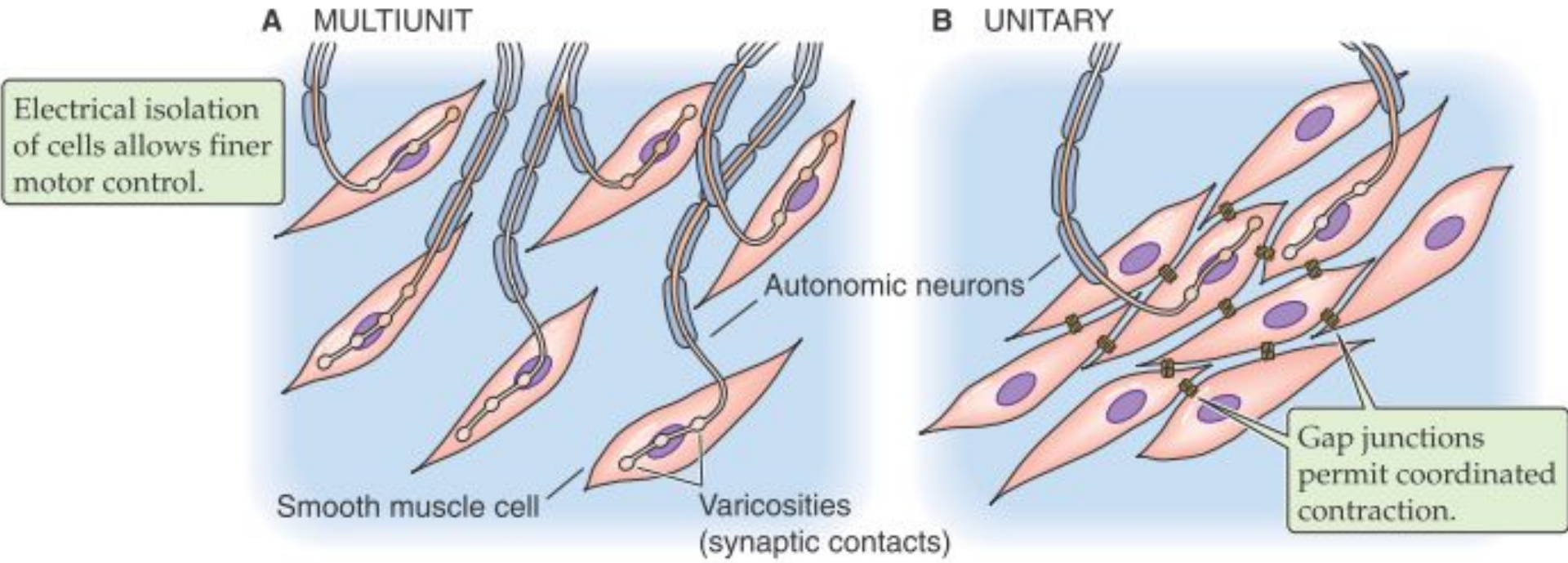
Гладкие мышцы

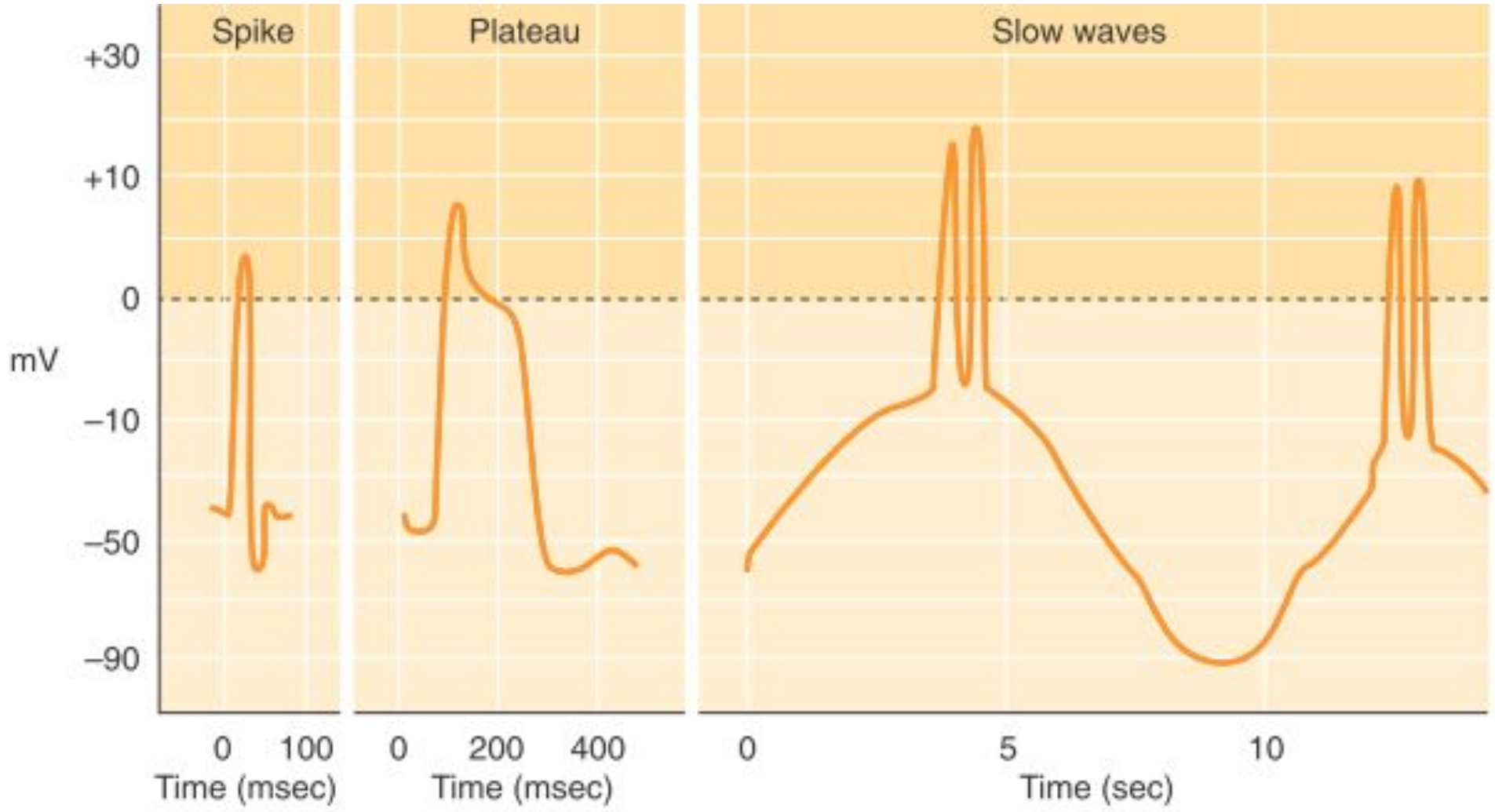


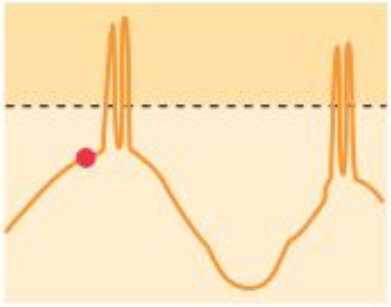
(a) Relaxed smooth muscle fiber (note that gap junctions connect adjacent fibers)



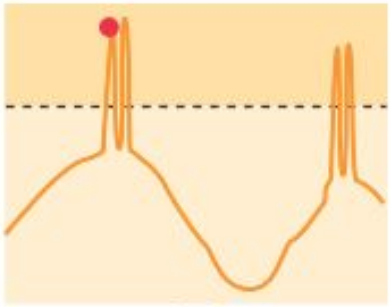
(b) Contracted smooth muscle fiber


A MULTIUNIT
B UNITARY
Electrical isolation of cells allows finer motor control.
Autonomic neurons
Smooth muscle cell
Varicosities (synaptic contacts)
Gap junctions permit coordinated contraction.


Spike
Plateau
Slow waves
+30
+10
0
mV
–10
–50
–90
0
100
Time (msec)
0
200
400
Time (msec)
0
5
10
Time (sec)



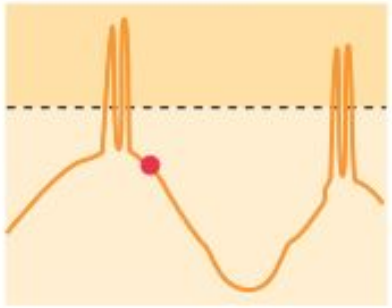
1 Voltage-gated Ca^{2+} channels open



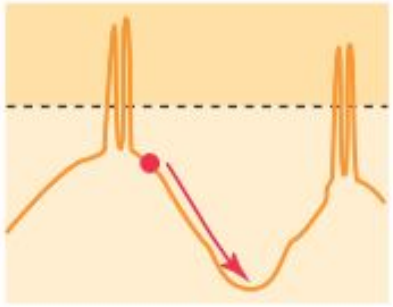
2 Action potential spike

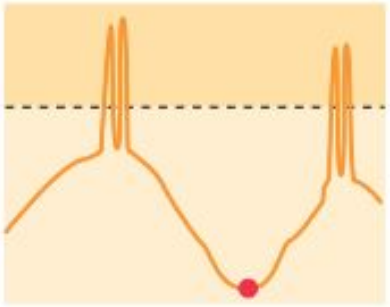
3 Ca^{2+} influx, rise in internal Ca^{2+} concentration

4 Open Ca^{2+}-dependent K^+ channels



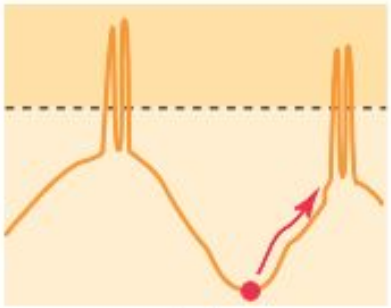
5 Slow hyperpolarization

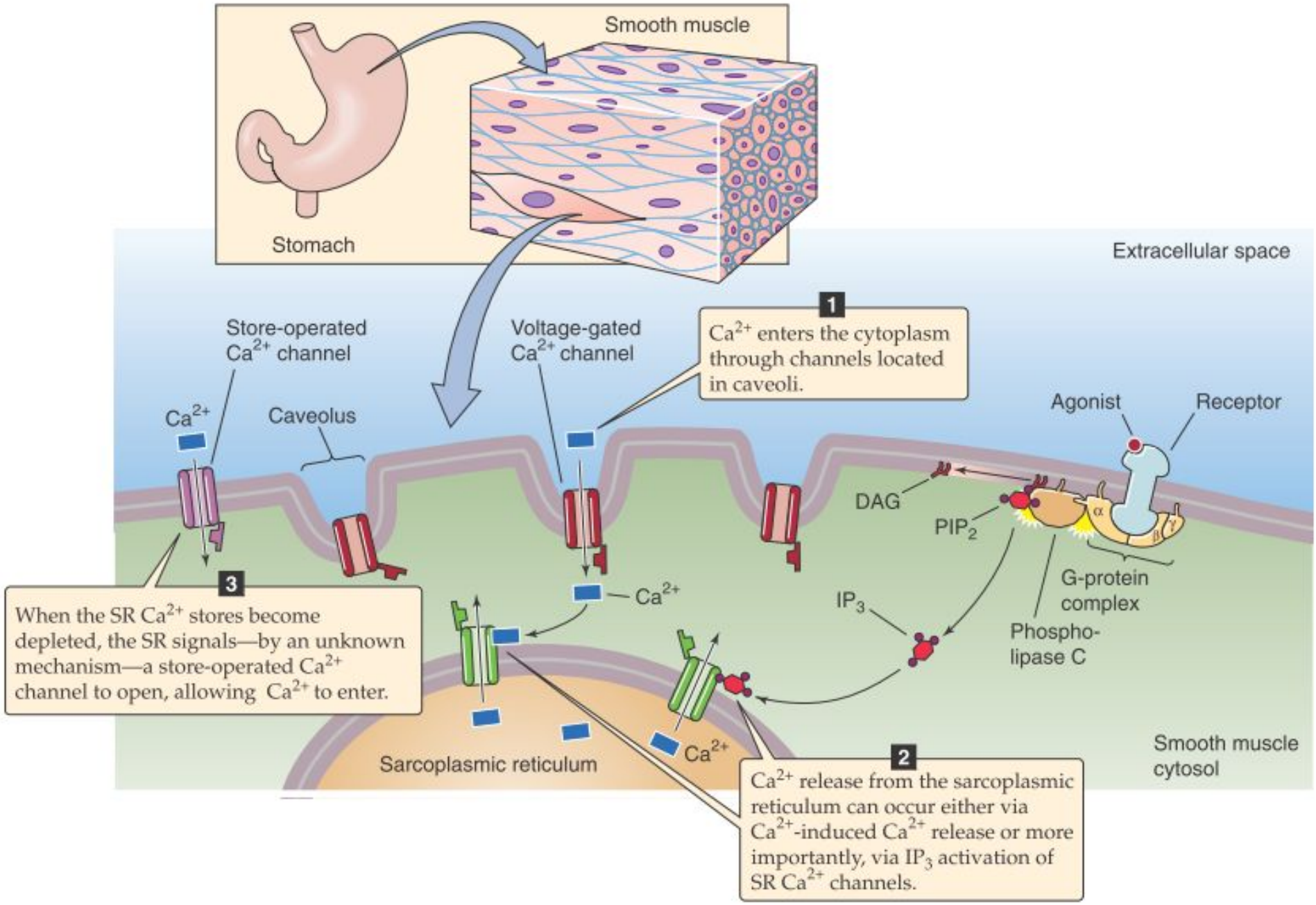


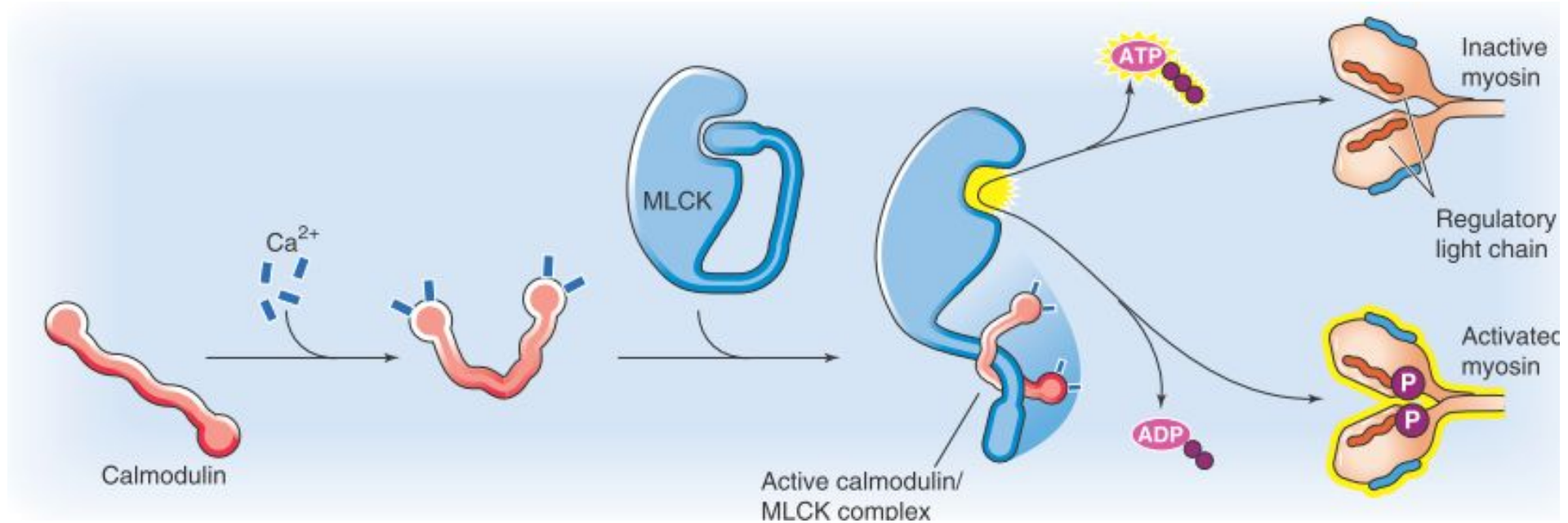


6 Voltage-gated Ca^{2+} channels close; internal Ca^{2+} concentration decreases

7 Ca^{2+}-dependent K^+ channels close




Smooth muscle
Stomach
Extracellular space
Store-operated Ca^{2+} channel
Voltage-gated Ca^{2+} channel
1
Ca^{2+} enters the cytoplasm through channels located in caveoli.
Ca^{2+}
Caveolus
Agonist
Receptor
DAG
PIP_2
IP_3
G-protein complex
Phospho-lipase C
3
When the SR Ca^{2+} stores become depleted, the SR signals—by an unknown mechanism—a store-operated Ca^{2+} channel to open, allowing Ca^{2+} to enter.
Ca^{2+}
Ca^{2+}
Sarcoplasmic reticulum
2
Ca^{2+} release from the sarcoplasmic reticulum can occur either via Ca^{2+}-induced Ca^{2+} release or more importantly, via IP_3 activation of SR Ca^{2+} channels.
Smooth muscle cytosol


Ca2+
Calmodulin
MLCK
Active calmodulin/
MLCK complex
ATP
ADP
Inactive
myosin
Regulatory
light chain
Activated
myosin
P
P

	Skeletal	Cardiac	Smooth
Mechanism of excitation	Neuromuscular transmission	Pacemaker potentials Electrotonic depolarization through gap junctions	Synaptic transmission Hormone-activated receptors Electrical coupling Pacemaker potentials
Electrical activity of muscle cell	Action potential spikes	Action potential plateaus	Action potential spikes, plateaus Graded membrane potential changes Slow waves
Ca^{2+} sensor	Troponin	Troponin	Calmodulin
Excitation-contraction coupling	L-type Ca^{2+} channel (DHP receptor) in T-tubule membrane coupling to Ca^{2+}-release channel (ryanodine receptor) in SR	Ca^{2+} entry through L-type Ca^{2+} channel (DHP receptor) triggers Ca^{2+}-induced Ca^{2+} release from SR	Ca^{2+} entry through voltage-gated Ca^{2+} channels Ca^{2+}- and IP_3-mediated Ca^{2+} release from SR Ca^{2+} entry through store-operated Ca^{2+} channels
Terminates contraction	Breakdown of ACh by acetylcholinesterase	Action potential repolarization	Myosin light chain phosphatase
Twitch duration	20-200 ms	200-400 ms	200 ms—sustained
Regulation of force	Frequency and multifiber summation	Regulation of calcium entry	Balance between MLCK phosphorylation and dephosphorylation Latch state
Metabolism	Oxidative, glycolytic	Oxidative	Oxidative