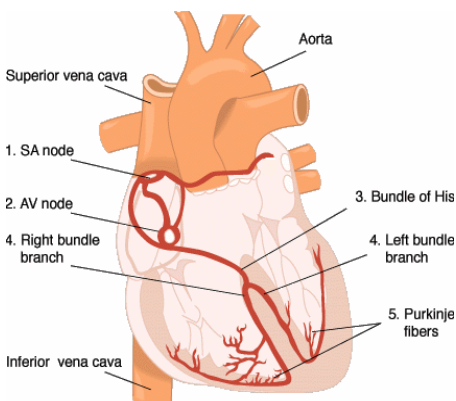


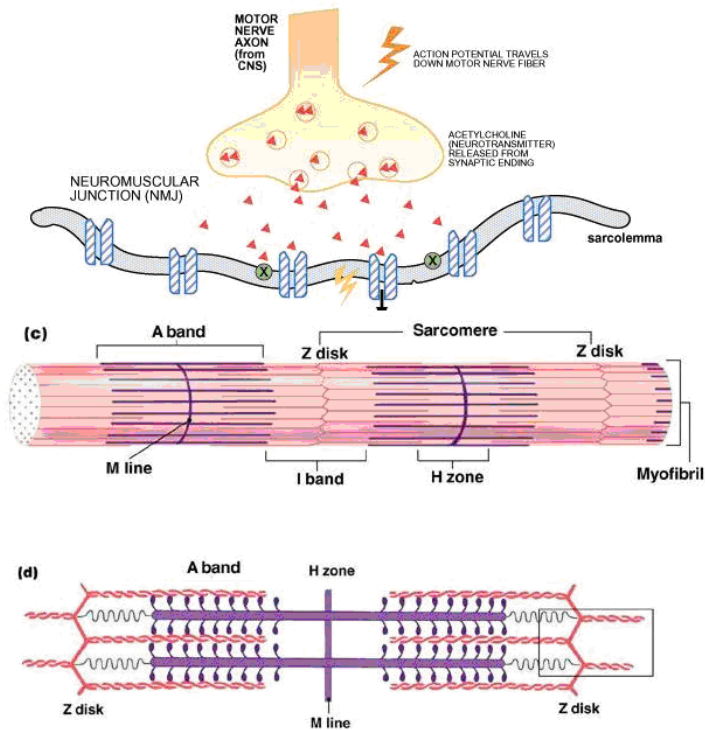
1. The information recorded on the EKG represents \_\_\_\_\_ activity of \_\_\_\_\_ of the heart muscles.
2. Myocardial contraction is caused by depolarization of the \_\_\_\_\_.
3. The recovery phase that follows depolarization is known as \_\_\_\_\_.
4. The \_\_\_\_\_ is the \_\_\_\_\_'s dominant pacemaker and its pacing activity is called a Sinus \_\_\_\_\_.
5. \_\_\_\_\_ contraction is recorded as a P wave on the EKG.
6. The AV node is the only electrical conduction pathway between the atria and the \_\_\_\_\_.
7. Depolarization conducts slowly through the AV node then rapidly through the \_\_\_\_\_ bundle to the right and left \_\_\_\_\_ branches.
8. Contraction of the \_\_\_\_\_ produces the QRS complex on the EKG.
9. The QRS complex is followed by the ST segment which represents the initial phase of \_\_\_\_\_ repolarization.
10. The \_\_\_\_\_ wave represents the rapid phase of repolarization.

**Fun Fact #1:** Fibrillation is caused by rapid discharges from numerous irritable automaticity foci in the atria or ventricles

**Fun Fact #2:** A premature ventricular contraction called a PVC originates suddenly in an irritable ventricular automaticity focus and produces a giant QRS complex on an EKG



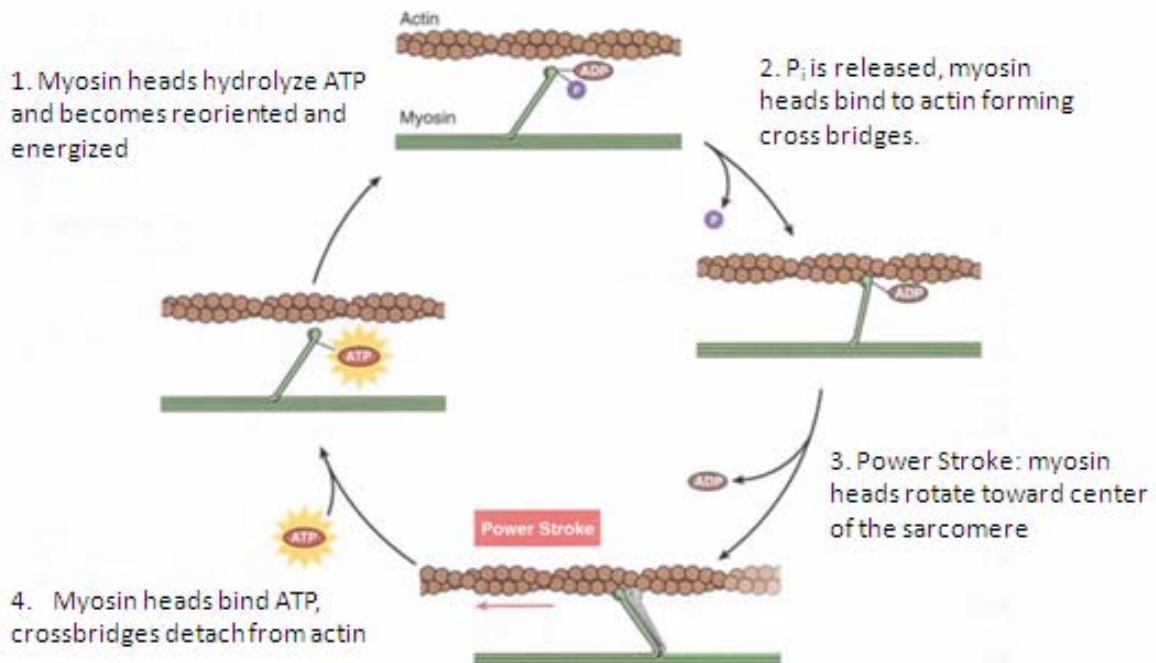
<u>WORD BANK</u>				
Myocytes	Contraction	Ventricles	Repolarization	His
T	Atrial	Heart	Electrical	Rhythm
SA node	Bundle	Ventricular	Ventricles	



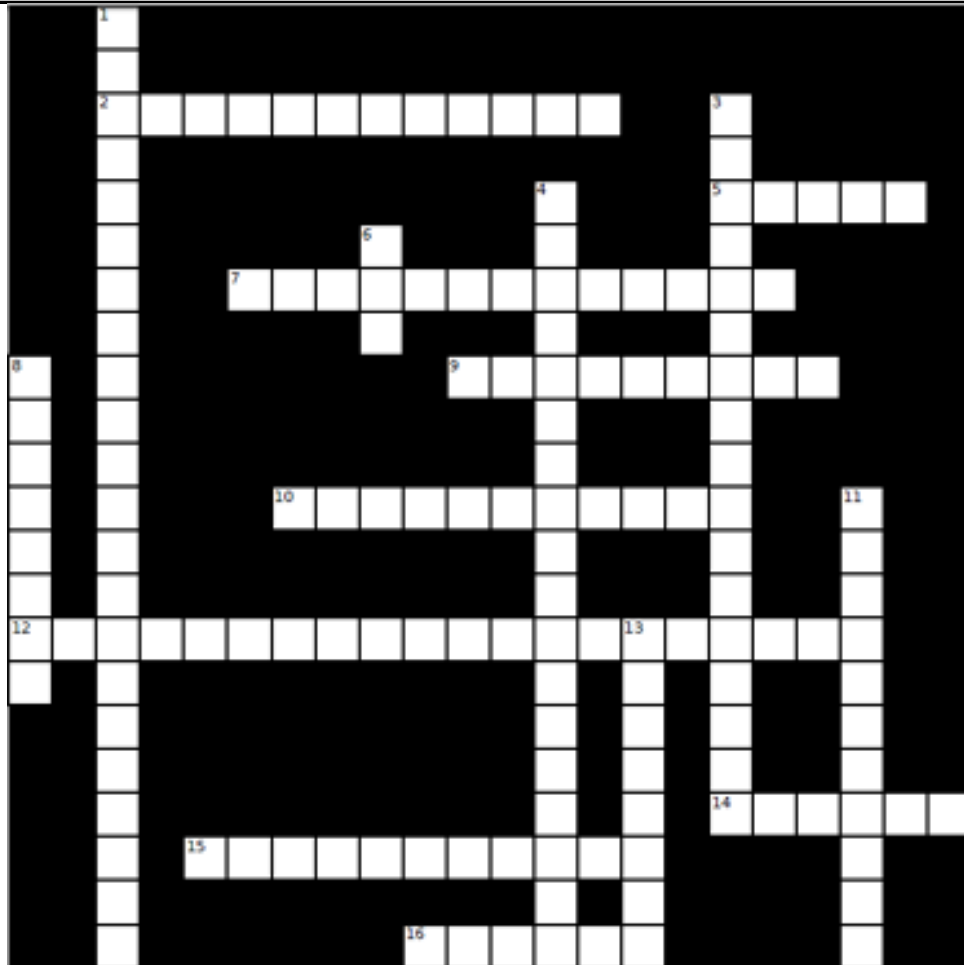
1. An action potential is propagated through the motor neuron
2. Voltage-gated  $\text{Ca}^{2+}$  channels open
3. Vesicles fuse and release of the acetylcholine (ACh) into the synaptic cleft
4. ACh diffuses and binds to its receptor on the muscle membrane
5.  $\text{Na}^+/\text{K}^+$  channels open,  $\text{Na}^+$  enters the muscle causing a depolarizing potential that generates an action potential in the muscle
6. The action potential propagates along the muscle
7. ACh in the synaptic cleft is degraded by acetylcholinesterase (AChE)

The motor neuron terminal contacts muscle fibers at the motor end plate forming the neuromuscular junction

### Excitation-Contraction Coupling



	Smooth Muscle	Skeletal Muscle	Cardiac Muscle
<b>Striations</b>	No	Yes	Yes
<b>Nucleus</b>	One central nucleus	Multiple nuclei located peripherally	One central nucleus
<b>Regulation of Contraction</b>	Involuntary	Voluntary	Involuntary
<b>Type of Contraction</b>	Relatively slow, graded	All-or-nothing	All-or-nothing
<b>Type of Innervation</b>	Autonomic efferent	Somatic efferent	Autonomic efferent
<b>Capacity for effective Regeneration</b>	Considerable	Limited	None
<b>Response to Increased Demands</b>	Hypertrophy, Hyperplasia	Hypertrophy	Hypertrophy



- Across
- Condition which results from lack of ATP generation causing inability of actin and myosin to dissociate
  - Each thick myosin filament is surrounded by 6 thin \_\_\_\_\_ filaments
  - The ability of muscle tissue to contract forcefully when stimulated by an action potential
  - Functional unit of skeletal muscle
  - Increased proliferation of cells within an organ or tissue
  - Enzyme responsible for degradation of acetylcholine
  - The only muscle type not under voluntary control
  - The filamentous protein that winds around actin and lies in grooves in its surface, providing strength
  - Contraction of skeletal muscle requires ATP which binds to the globular head of this protein

- Down
- The specialized form of smooth endoplasmic reticulum in muscle
  - Autoimmune disease in which autoantibodies attach to acetylcholine receptors blocking the ability of acetylcholine to bind
  - Specialized end to end junctions found in cardiac muscle
  - Energy molecule which causes dissociation of the myosin head from actin
  - Movements of the whole body rely on the integrated functioning of bones, joints, and this type of muscle
  - Increase in the volume of an organ or tissue due to the enlargement of its component cells
  - Calcium binding to this protein alters the position of tropomyosin, exposing myosin binding site on actin