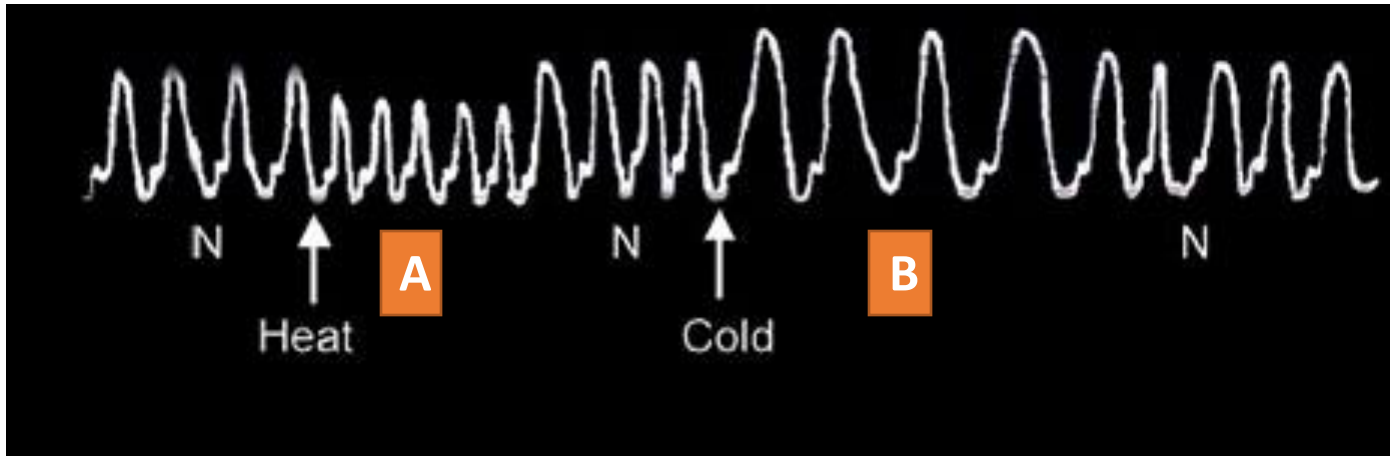
AMPHIBIAN GRAPHS OSPE 2



1

Effects of warmth/heat & cold on Sinus venosus.

a. Identify the graph(1)

b. Explain the effects 'A' and 'B' (4)

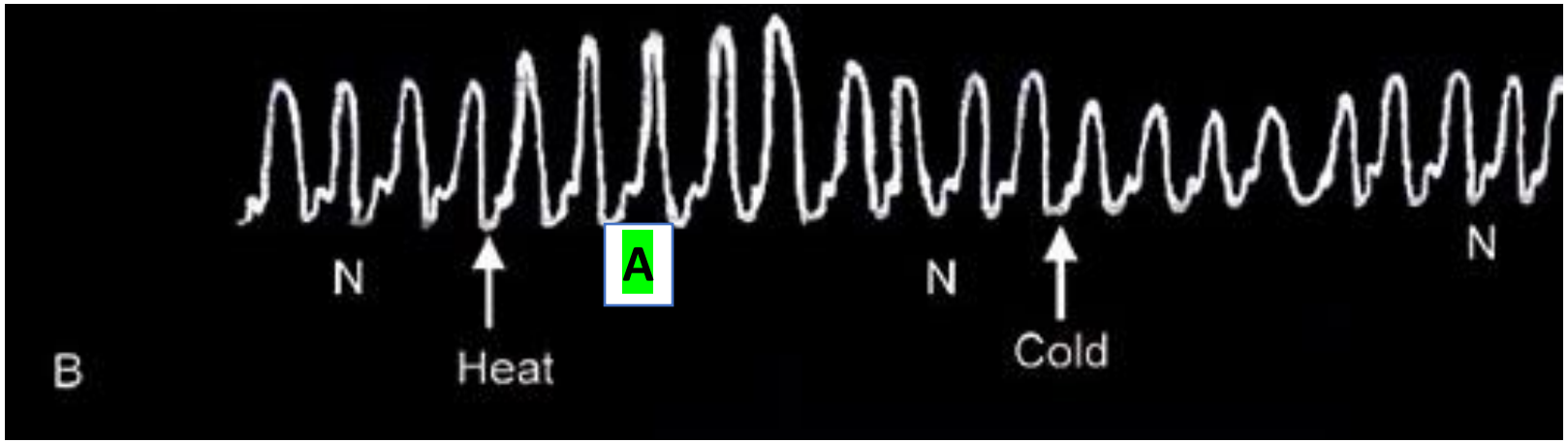
- Effect of warmth

When warmth is applied on SV (pacemaker), the metabolic rate ↑ ⇒ ↑ impulse production
So HR ↑ (1° effect)
As HR ↑ markedly, filling time ↓ and EDV ↓
So initial length of muscle fibre ↓ ⇒ amplitude ↓ (2° effect)
in accordance with Starling's Law as force of contraction ↓

- Effect of cold

Cold ↓ metabolism ⇒ ↓ impulse production ⇒ ↓ HR (1° effect)
So filling time ↑ & EDV ↑ ⇒ force of contraction ↑ (Starling's Law)
⇒ amplitude ↑ (2° effect)

2



Effect of warmth/heat on ventricles

a. **Identify the graph (1)**
b. **Explain the effect 'A' (2)**
c. **Mention factors affecting heart rate (2)**

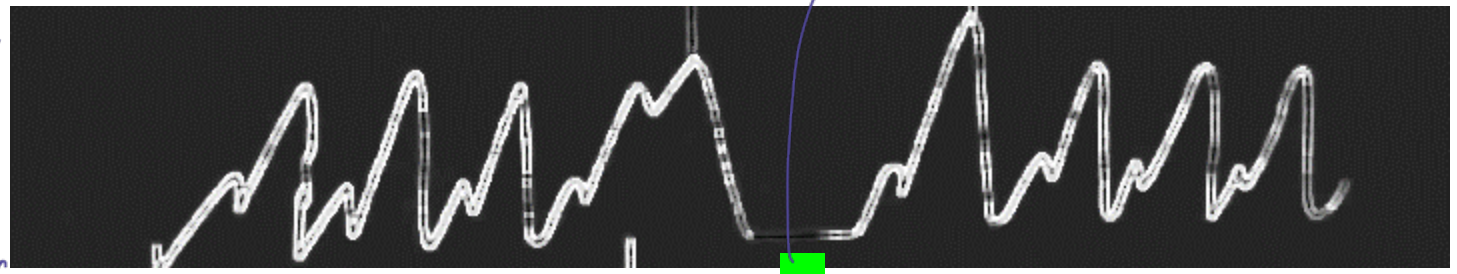
b. When warmth is applied to ventricles, force of contraction ↑ and ⇒ amplitude ↑ as metabolism of ventricles ↑ on application of heat

C. Body temperature, age, emotions, body positions.

2. In cardiac muscle, refractory period is long. ARP extends throughout systole and early ⅓rd of diastole. ⇒ No response of stimulus applied during this period. Stimulus applied during latter part of diastole produces extra systole. It is followed by the compensatory pause. Following the extra systole next normal impulse from SV reach the ventricle during ARP of extrasystole ⇒ impulse fails to produce a heart beat and the ventricle has to wait for next normal impulse from SV to contract. During the Comp. pause, the ventricle gets enough time to get filled so next beat is of ↑ amplitude.

3

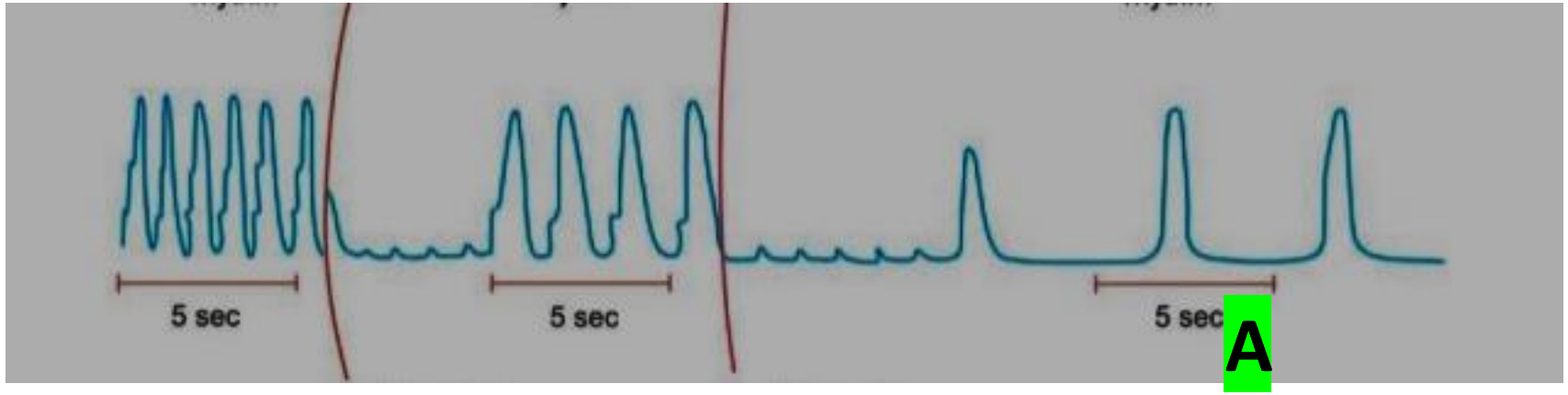
→ Compensatory pause



Demonstration of extrasystole in normal

1. Identify the graph (1) cardiogram of frog
2. What is the reason for the part marked 'A' (3)
3. Which is the pace maker of frog's heart? (1) Sinus Venosus

4



1. Identify the graph (1)
2. What does the part marked 'A" denote? (2)
3. Can this experiment be done in mammalian heart. Why? (2)

Stannius Ligature

Quiescent period (due to overide suppression)

3. • Coronary flow will get obstructed leading to myocardial death
• Chambers of heart are clearly separated.
• 1st Ligature cannot be applied due to SA Node pos^{n} – posterior wall of RA.
• 2nd Ligature cannot be applied at the AV junction as it occludes coronary circulation

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Vagal Inhibition

Vagal Escape

→ Causes of Vagal Inhibition

- With weak stimulus, the heart may beat faster more forcefully due to excitation of symp fibres in vagal trunk. When strength ↑, HR ↓ and is weaker & finally heart stops in diastole.
- With continued stimulation (do not ↑ I) the heart begins to beat inspite of the stimulation because it escapes from the inhibition and begins to beat again at a slower rate – idioventricular rhythm.

→ Causes of Vagal Escape

- Idioventricular rhythm: due to inhb. of pacemaker, ventricles contract at their own rhythm
- Stimulation of symp. NS in vagus trunk.

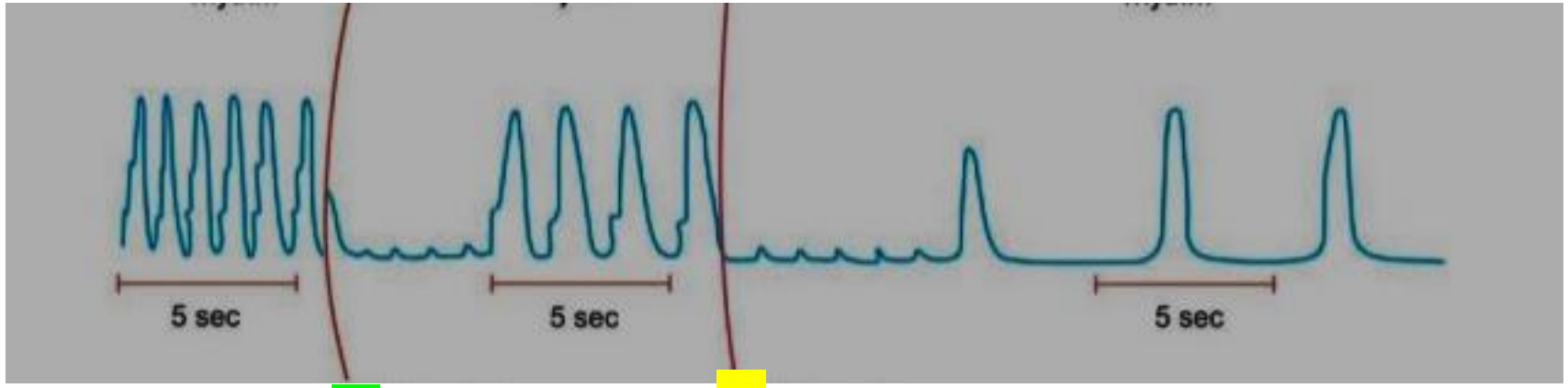
Effect of Vagal Stimulation of frog's heart

1. Identify the graph (1)

2. Explain the parts marked A and B giving reasons (4)

- Exhaustion of stored Ach in nerve ↑ destr. of Ach in heart tissue.
- Theory of tachyphylaxis: ↓ response of tissue to repeated stim. ⇒ Ach not produce same response the same time.
- Accum. of blood & stretch of vent. muscle

6



Stannius Ligature

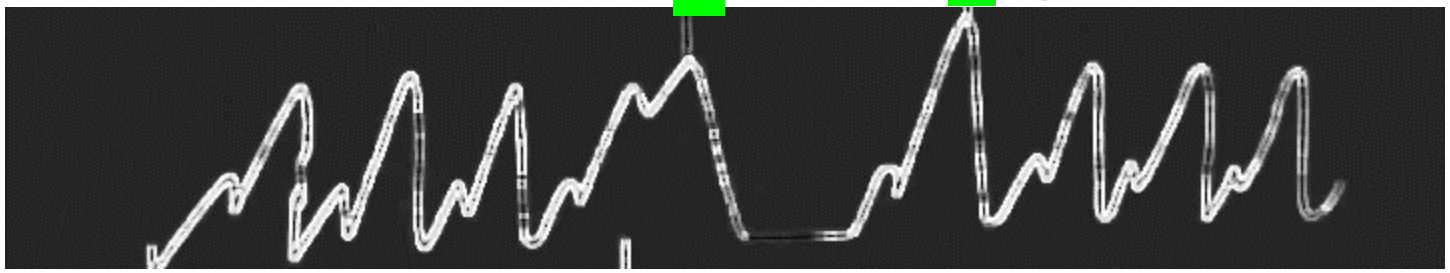
1. Identify the graph (1)
2. What does the points marked 'A" and 'B" denote? (2) 1st & 2nd Stannius Ligature resp.
3. What does this experiment prove? (2) To prove Sinus Venosus is pace-maker of frog's heart and different parts of the heart have different rates of impulse production

Extrasystole

Post extrasystolic potentiation

A

B



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1. Identify the part marked “A” (1)
2. Can this occur in human heart?(1)
3. What is the reason for the higher amplitude of ‘B’? (3)

Under pathological conditions

Similar to answer 2 of Q3. (last part)