Vasoconstrictors

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Vaso Constrictors

Added – to counteract vasodilation effect of injectable L.A

- Decreases rate of absorption
- Reduces the risk of overdose reaction
- Increases duration of action
- Reduces bleeding at the site

Based on chemical stc → (Catechol nucleus)

Catecholamines

Epinephrine

Nor epinephrine

Dopamine

Non catecholamines

Amphetamine

Meta amphetamine

Receptors

α1 -exitatory post synaptic

α2 -inhibitory post synaptic.

β -vasodilatation and broncho dilatation. On heart it causes-increase in heart rate and strength of contractions.

β1- heart and small intestine

β2- bronchi and vascular bed.

Effect on CVS

- Increased- Heart rate, Stroke volume , cardiac out put, oxygen consumption. – BETA
  - There is also increased irritability of myocardium – premature ventricular contracture, tachycardias.
- Increase in both systolic and diastolic BP.

[high BP carotid and aortic baro receptors are stimulated- decrease in heart rate and out put.]

[intra venous administration causes- fall in BP- rather than rise in BP]

Effect - other

Respiratory system-broncho dilatation – BETA2 action.

[epinephrine – acute asthma.]

CNS- stimulant in toxic dose.-

commonly noted systemic effects- Jitteriness, Apprehension, Mild Excitation.

Blood sugar- increases- Epinephrine- increased glycogenolysis (liver and muscle)

Pupillary dilatation-epinephrine.

EPINEPHRINE :-- Adrenaline α1& β receptors

Systolic & Diastolic pressure, Heart rate

Oxygen consumption ,Stroke volume-increases.

↑ CNS stimulation , Bronchodilator, α1 – vasoconstriction,β 2 – vasodilation ,↑oxygen consumption ,↑ blood sugar level ,0.2 mg – healthy ,0.04mg – CVS impaired

- Range of dilutions available are 1:10,000-1:1,00,000. How ever now recently its been shown that dilution upto 1:2,00,000 is effective enough and with least of the side effects.
- blood pressure heart rate and cardiac out put increases – with these minimal doses observed.

- epinephrine reactions.:--

  apprehension , tachycardia, sweating and pounding in chest-

Patient with cardio vascular or thyroid problem

Epinephrine causes increase in heart rate- 25 – 70 times more

Small doses systolic increases and diastolic decreases. –greater sensitivity to β2 . only in large volumes It causes α receptor activation and increase in diastolic BP by constriction of vessels- skeletal muscle

- Nor epinephrine- lacks β2 action and causes intense peripheral vaso constriction and increased B.P.

Overall it causes increase in systolic & diastolic B.P. decreased heart rate. Increased stroke volume and increased peripheral resistance. Almost no effect on bronchioles. CNS stimulant & increases oxygen consumption.

Felypressin:-- Direct stimulation of vasculature,more action on venous system.

No direct effect on Myocardium ,Non-arrythmagenic,High doses – impaired coronary flow Vasoconstriction – coronary blood vessels, Anti-diuretic action, Oxytocin like action – uterus , Max dose. 0.04mg

In very high doses impair blood flow to coronary artery.

Note: during G.A procedures -with gaseous anesthesia - epinephrine is known to cause cardiac dysarrythmias- ventricular fibrillation.bradycardia is also one of the most common complication.

Thank you…

Allergy

- Defn: It is immunologically mediated inflammation.

components. :

allergen, antigen presenting cell, t helper cell , IgE cells , b cells , fc receptors on mast cells.

Types- local & systemic.

Philip&robin coombe- classified it as

- Type I –Ig E – anaphylactic /immediate . Eg angioedema.
- Type II – IgG -anti body dependant cytotoxic reaction.eg- transfusion reaction.
- Type III- IgG –immune complex deposited in tissue –complement dependant. Immune complex disorder. Eg serum sickness

- Type IV –antigen sensitized t-cell release lymphokines.-cell mediated/delayed.eg tissue graft.
- TypeV-antibody react with a surface component –stimulatory type.- tsh auto anti bodies in graves disease.
- TypeVI-excessive c_3 activation- eg – gram negative septicemia .innate type.

- PK test- (pransinitz-kustner) – serum of allergic individual into normal individual.-after 24 hrs re inject serum – sub Cutaneous
- PCA test- serum at different concentrations used. – intravenously.
- Shultz dale test- invitro test smooth miscle from the susceptible animal collected and exposed to antigen. Contraction means + .

- Respiratory system during allergy- vaso dilatation- increased perfusion- interstitial spaces.- laryngeal edema.– bronchospasm & asphyxia.
- CVS- there is reduced vaso motor tone and increased venous capacitance. – vaso dilatation can cause cardio vascular collapse .

- GIT- cramping pain , nausea vomiting &diarrhea.
- Conjunctivitis and rhinitis are also common

Basic

- Management - terminate dental procedure.position patient .
- BLS.
- Definitive care- observe- administer oral histamine blockers. Admn histamine every 4-6 hours. Call for medical help.
- Epinephrine50 mg IM/ chlorphenhydramine10 mg IM .CVS & Rs symptoms- epinephrine- 0.1 mg IV

Management.

- **Laryngeal edema – type of angio neurotic oedema- life threating.**
  - **Edema upper air way – laryngeal edema**
  - **Lower air way affect broncioles- small.**

- **Management**
  - **`skin reactions-**
    - **Delayed – non life threatening - oral histramine blockers- 50 mg diphenhidramine**
    - **Immediate reaction—with conjunctivites rhinitis- vigerous managemennt.**
    - **0.3 mg epinephrine. IM**
    - **50 mg diphenhydramine Im**
    - **medical help summoned**

- **Observe patient for minimum of 60 min**
- **Oral histamine blockers for 5 days.**
- **Respiratory reaction –**
  - **patient in comfortable position.**
  - **administer - oxygen**
  - **Admn epinephrine- bronchodilator**
  - **Observe for 60 min , advise anti histamines to prevent relapse.**

- **Laryngeal edema-**
  - **Patient position ,oxygen, broncho dilator, oral anti histamines.**
  - **If condition not improving cricothyrotomy - achieve patent air way if necessary give artificial ventilation**

Monitoring of vital signs –

position the patient. administer oxygen, BLS,

additional drugs like corticosteroids are known to be helpful.

respiratory distress due to bronchial obstruction-

broncho dilator- nebulisation –oxygen- intubate-

oxygen saturation less than 80.

SYNCOPE

DEFINITION:

"A sudden transient loss of consciousness without prodromal symptoms followed with resumption of consciousness within seconds to minutes"

Possible causes:

→Vasodepressor syncope

→ Drug administration

→ Orthostatic hypotension

→ hypoglycemic reaction

→Acute allergic reaction.

Patho physiology-

- Reduced cerebral metabolism resulting from inadequate oxygen.
- Causes- Dilatation of peripheral arterioles-

  Sharp drop in cardiac output

  Constriction of cerebral vessels as Co_2 is lost through hyperventilation.

  ventricular dysarrithmias.
- Brain consumes 20% o_2 and 65% total glucose of body.

Compensatory mechanism cause increased H.R.- feeling of warmth- bradycardia – Decompensation occurs -- vagal mediated nausea- reduced cerebral blood flow- lightheadedness- if prolonged syncope.

Patho physiology- Pre Syncope.

Stress- catecholamines into circulation-change in tissue blood perfusion- less venous return and more flow tissue- pooling of blood- reduced arterial BP.

Compensatory mechn- Baro receptors constrict B.Vessels- Increase heart rate.

[Critical level- 30 ml blood / 100gmbrain tissue per minute]

- Reduced cerebral metabolism-

  can be due to general or local systemic causes like-

  - Hyperventilation
  - Hypoglycemia
  - Acute drug allergy.

- Direct or reflex affect on part of CNS-
  - loss of concious ness due to changes in the brain it self.
- Psychic mechanism affecting levels of consciousness.
  - Emotional disturbances
  - Vaso depressor syncope and
  - Hyper ventilation

Protocol for Resuscitation

PHASE I : BASIC LIFE SUPPORT

PHASE II :ADVANCED LIFE SUPPORT

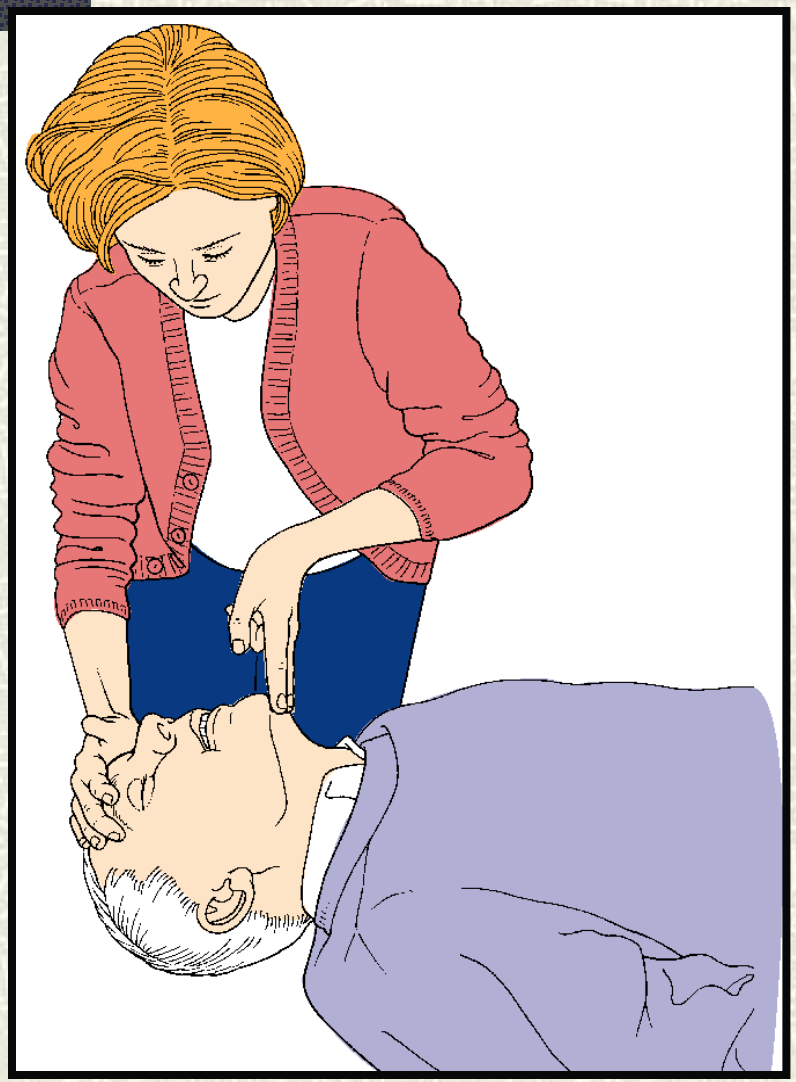
PHASE III : PROLONGED LIFE SUPPORT

Shake and Shout

If No Response.

- Shout for help
- Open their airway
- Check for breathing

Opening the airway



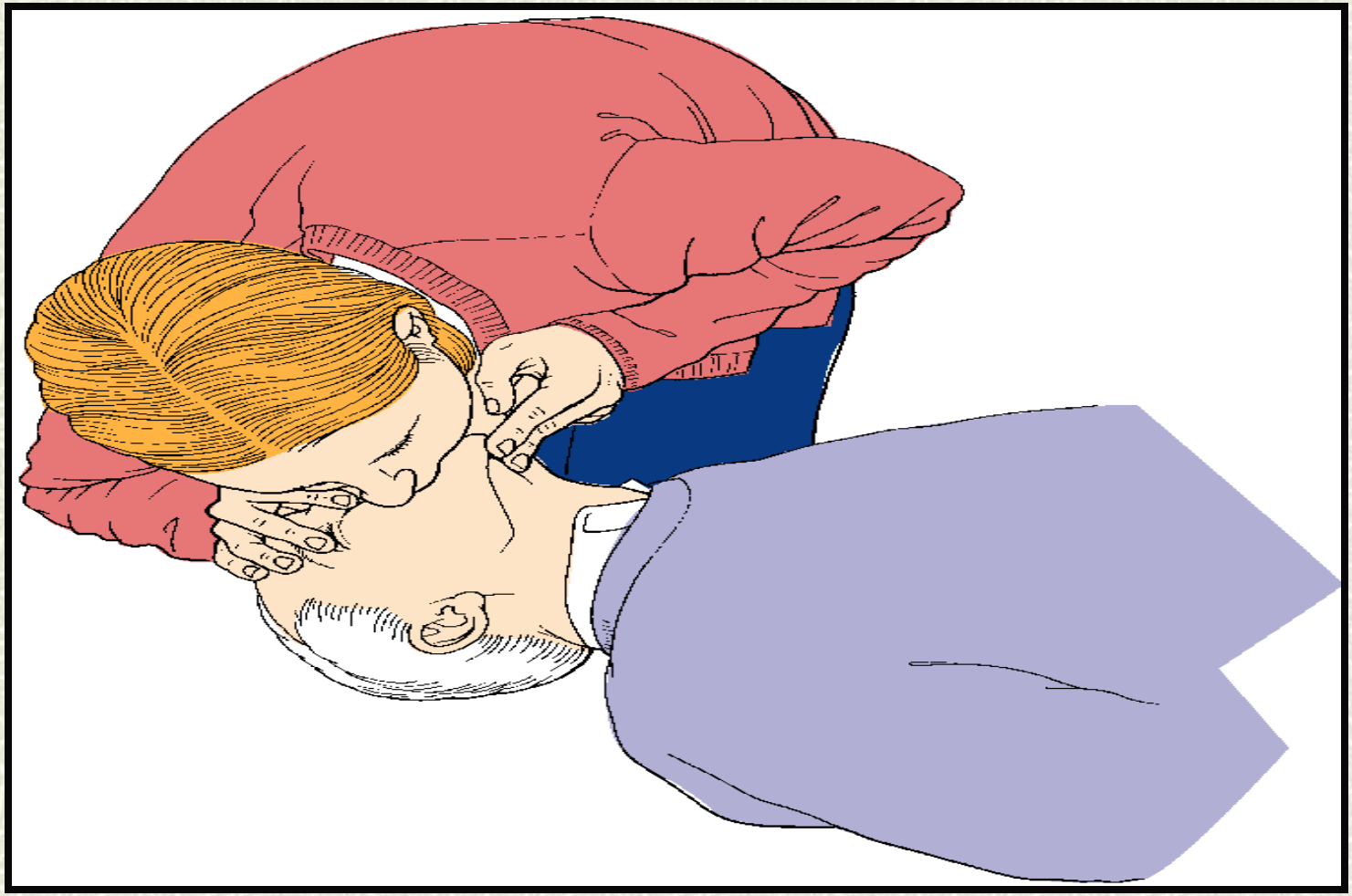
- Head tilt
- Chin lift
- If cervical spine injury suspected:
  - jaw thrust

Assess Breathing

- Look for chest movement
- Listen for breath sounds
- Feel for expired air
- Assess for 10 seconds before deciding breathing is absent

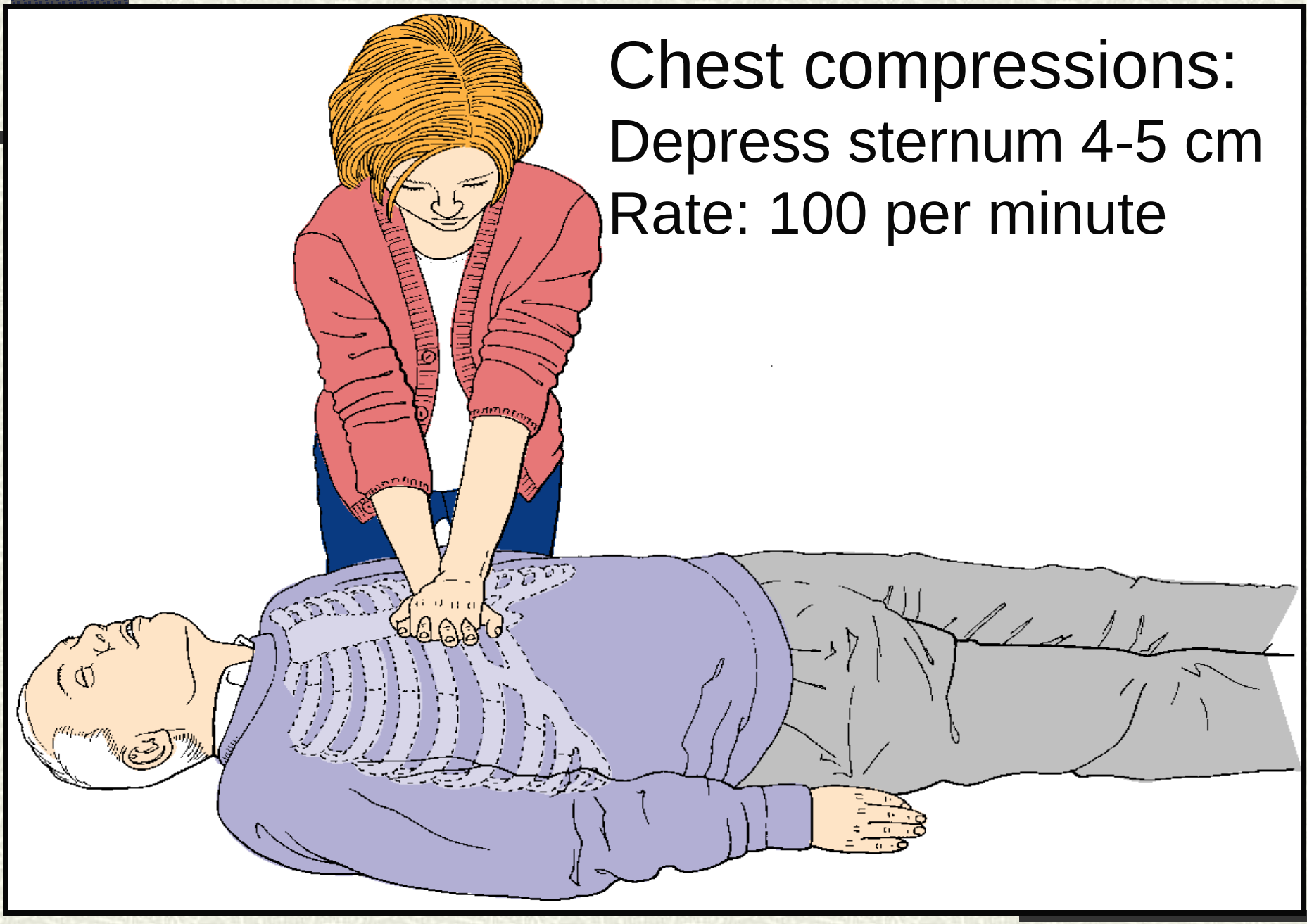
Expired air ventilation

- Occlude victim's nose
- Maintain chin lift
- Take a deep breath
- Ensure a good mouth-to-mouth seal
- Blow steadily (2 sec) into victim's mouth
- Watch for chest rise
- Maintain chin lift, remove mouth
- Watch chest fall



Assess Circulation

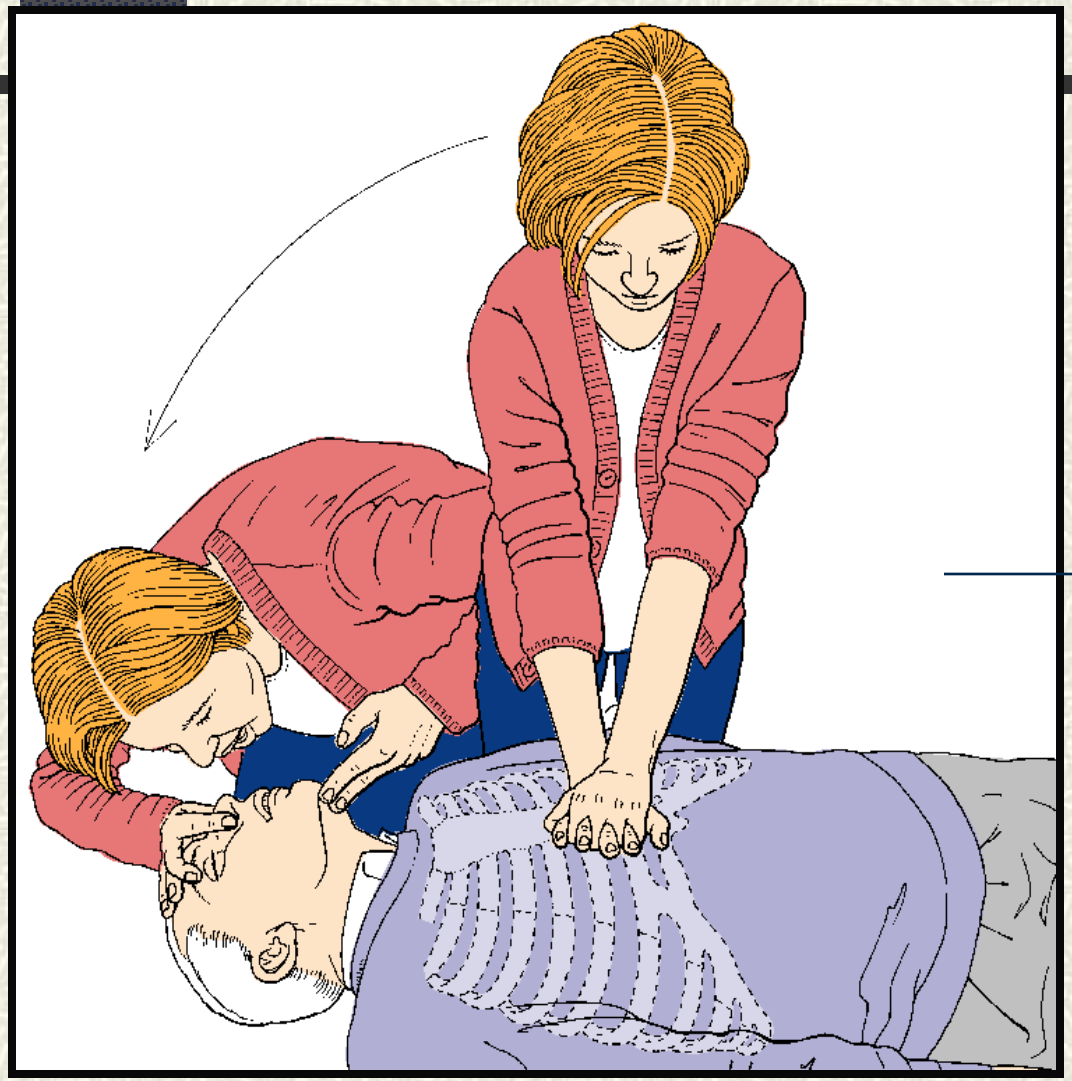
- Look, listen and feel for normal breathing, coughing, or movement by the victim
- Check the carotid pulse (if trained)
- Take no more than 10 seconds.
- If normal wait for help
- If absent-



Chest compressions:

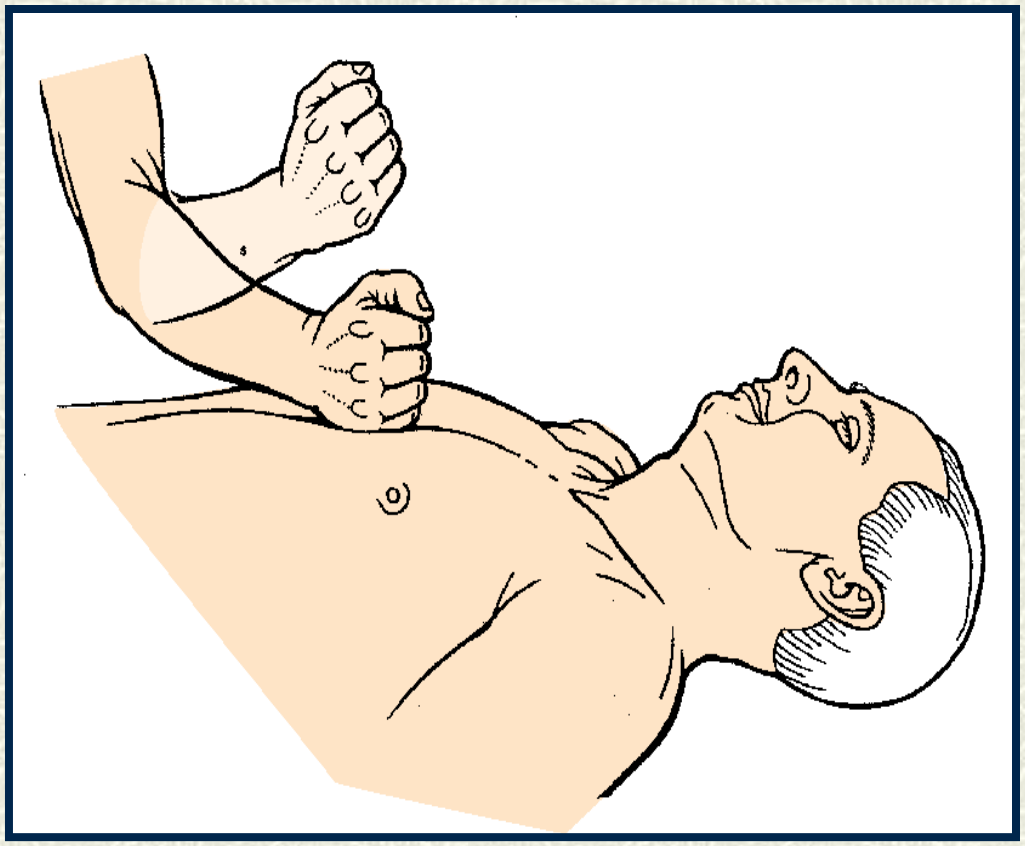
Depress sternum 4-5 cm

Rate: 100 per minute



- 15 compressions : 2 breaths for
  - 1-person CPR

Pericardial thump.



- Indication:
  - –witnessed or monitored cardiac arrest

AIRWAY MANAGEMENT AND VENTILATION

ADJUVANTS TO BASIC AIRWAY TECHNIQUES

Oropharyngeal airway
Nasopharyngeal airway

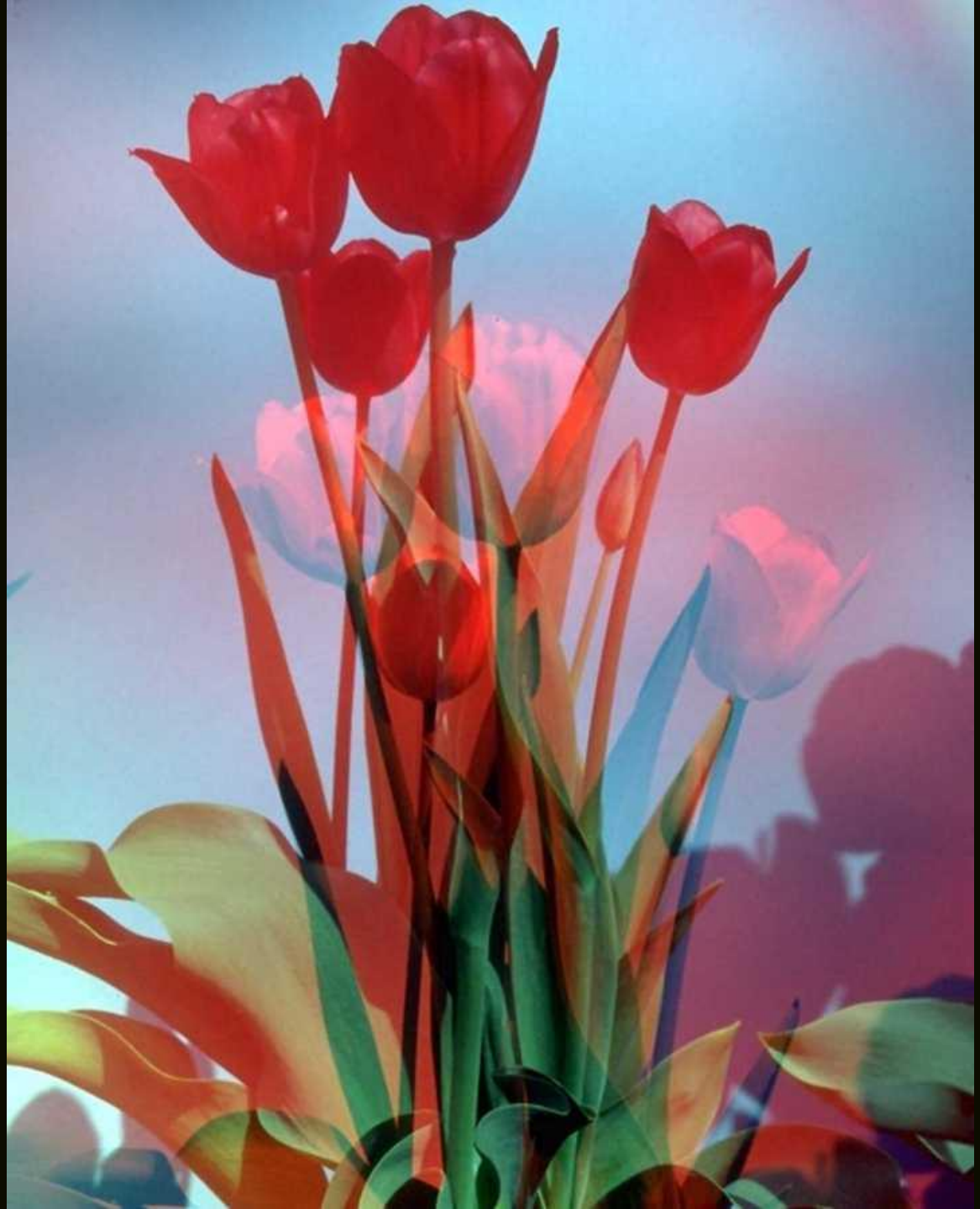
Oxygen
Suction

Ventilation

- Mouth to mask ventilation
- Self Inflating bag
- Laryngeal Mask Airway

Tracheal Intubation:

- Provides 100% Oxygen
- Clear and secure airway



THANK YOU

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