I. Essay: (2 x 15 = 30)

1. Define Firearm. Draw and describe the parts of the Shotgun Cartridge. Describe the pattern of entrance wounds produced by a shotgun at various ranges.

Definition of Firearm

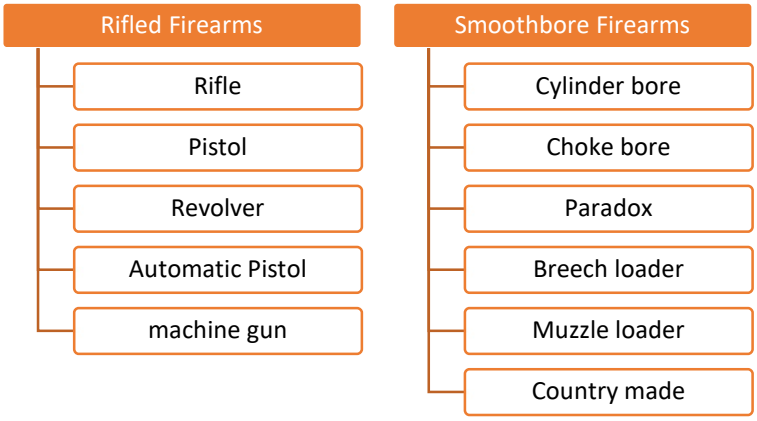
A firearm is a weapon from which a projectile is discharged by the expansive force of gases produced by combustion of gunpowder.

Mechanism

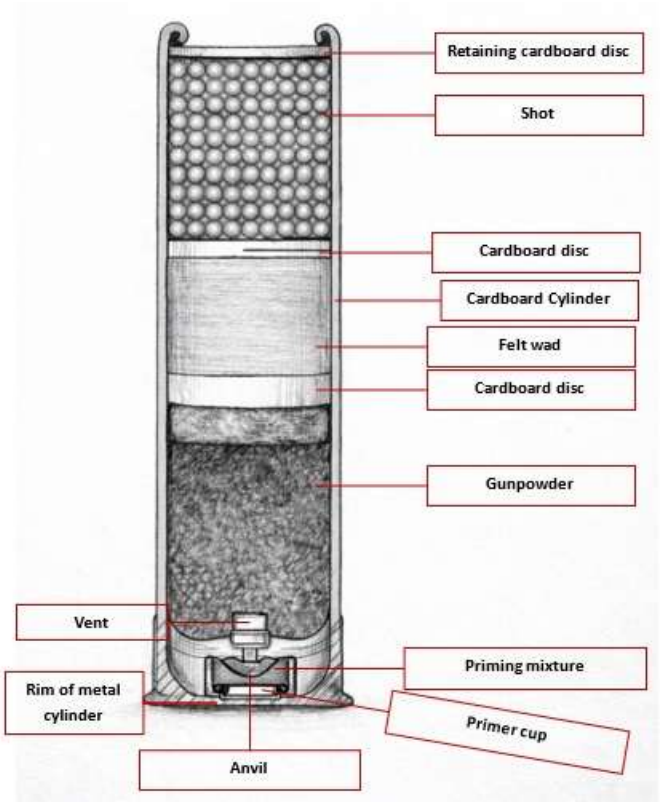
When the trigger is pulled, the hammer or firing pin IS released and strikes the primer of the cartridge, causing ignition of the primer compound. This ignition sets fire to the propellant powder present inside the cartridge case. Combustion of the propellant produces rapidly expanding high-pressure gases. These gases exert force on the base of the bullet or shot, driving it forward through the barrel. As the projectile moves along the barrel, it gains velocity due to continued gas expansion. Finally, the projectile exits the muzzle at high velocity, followed by hot gases and residues.

Classification

Based on Barrel Type



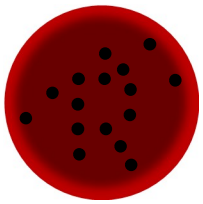
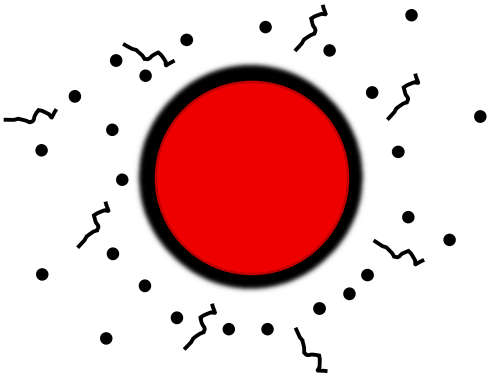
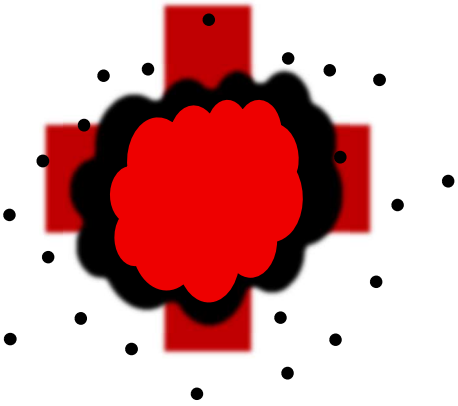
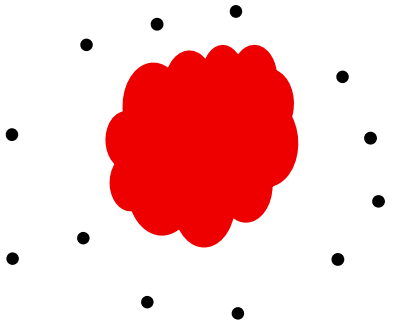
Shotgun Cartridge – Parts

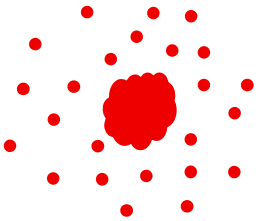
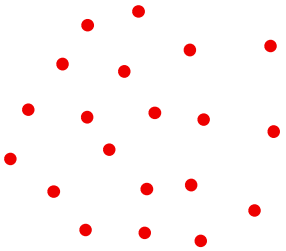


Parts of a Shotgun Cartridge:

1. **Cartridge case (shell)** – made of Paper/plastic/metallic and contains primer, shots, etc.
2. **Primer (cap)** – Contains priming mixture and anvil which initiates burns and ignites gun powder. Primer contains compounds of lead, antimony and barium.
3. **Gunpowder –** ignition of gun powder produces rapidly expanding high-pressure gases. It could be black powder, smokeless powder or semi- smokeless powder.
4. **Wad & cardboard disc**– Separates powder from pellets to block escape of gases and heat for prevent fusion of pellets.
5. **Shot (pellets)** – made of Lead (Soft shot). Some times hardened with antimony (Hard shot).
6. **Top wad / Retaining cardboard disc**– Holds pellets in position

Pattern of Entrance Wounds by Shotgun at Various Ranges

1. Contact Shot • Single large, circular wound • Blackening, burning, tattooing present inside wound • Wad may enter body • Severe tissue destruction	
2. Close Range **(up to 30 cm)** • Central single hole • Surrounding singed hair, blackening and tattooing • Pellets still act as a single mass • Wad will be inside wound	
(30 - 60 cm) • Central single hole with irregular rim gives Rat hole appearance • Surrounding blackening and tattooing • Pellets still act as a single mass • Wad opens up and produce Maltese cross type of bruise surrounding entrance wound.	
(60 cm to 1 metre) • Central single hole with irregular rim gives Rat hole appearance • No burning or blackening, but minimal tattooing present • Pellets still act as a single mass • Wad will be inside wound	

3. Short Range (1–2 metres) • Single central hole with irregular edges surrounded some satellite pellet holes. • Wad will be inside wound upto 2 meters. • No burning, blackening or tattooing. •	
4. Intermediate Range (2–4 metres) • Shot mass begins to expand. • Central hole with irregular edges surrounded multiple satellite pellet holes. Towards 4 meters, central hole becomes smaller and number of satellite pellet holes increases. • Wad strike below the wound.	
5. Distant Range (>4 metres) • Multiple discrete pellet holes • No burning, blackening or tattooing • Pellet spread increases with distance • Wad may or may not strike the body.	

2. A 30 year old male, painter by occupation presents to the Emergency Department with history of vomiting, headache, visual disturbances, restlessness and convulsion. On examination patient is anaemic with facial paleness, blue lines noted on the gums and left wrist drop.

a) What is your provisional diagnosis?

a) He is a painter. So there is a chance of lead poisoning as lead is a component of paint.
b) Symptoms like vomiting, headache, visual disturbances, restlessness and convulsion are symptoms of lead encephalopathy.
c) Signs like anaemic with facial paleness, blue lines noted on the gums (Burtonian line) and left wrist drop (feature of lead palsy) are seen in chronic lead poisoning.
So provisional diagnosis is Chronic Lead Poisoning (Plumbism)

b) Explain the various clinical manifestations of the underlying poison.

a) **Facial pallor**- earliest sign
b) Anemia
c) **Punctate basophilia or basophilic stippling-** It is the presence of dark blue pin head sized spots in the cytoplasm of red cells. This is due to the action of lead on the porphyrin metabolism by the inhibition of 5-pyramidine nucleotidase.
d) Polycythemia, polychromasia, reticulocytosis, poikilocytosis, anisocytosis and nucleated RBCs, eosinophilia.
e) **Lead line (Burtonian line):** A stippled blue line is seen on the gums due to the deposition of lead sulphide on the gum, usually on the upper jaw, near carious teeth, within a week of exposure.
f) **Lead colic**- Abdominal pain occurs in 85% of cases. More common at night and sometimes severe.
g) **Constipation** is the usual symptom
h) **Lead palsy**- it usually occurs late and characterised by numbness, paresthesia, cramps of the muscles of the lower extremities followed by paralysis of the muscles. Extensor muscles of wrist are effected and leads to Wrist drop.

i) **Lead encephalopathy**- it is common in children. Symptoms are vomiting, headache, visual disturbances, restlessness and convulsion. It is irreversible and causes permanent brain damage.

j) CVS – Hypertension

k) Kidney – chronic arteriosclerotic nephritis

l) **Reproductive system**- Amenorrhea, dysmenorrhea, menorrhagia, Sterility in both sexes, Loss of libido in males

m) **Lead osteopathy**- If children exposed for 4 weeks, lead is deposited in the epiphysis and seen as radio-opaque bands along metaphysis on X-rays.

c) Discuss in detail about treatment and post-mortem findings.

Diagnosis

1. History

2. Clinical features

3. X-ray - radio-opaque bands or lines at the metaphyses of long bones and along margins of iliac crest is seen in children.

4. Erythrocyte protoporphyrin (EP) level estimation.

5. Blood - Basophilic stippling.

6. Urine – Increased coproporphyrin III level, is a valuable screening test. In the blood, levels above 20 µg%, and in the urine 150 µg per litre is diagnostic.

Treatment

1. **Removal from source of exposure**
2. **Chelation Therapy**
    a) Treatment depends on the severity of poisoning and the blood lead (BL) level.
    b) In severe acute poisoning with encephalopathy, BAL is given immediately, followed by $CaNa_2$ EDTA intravenously.
    c) BAL is administered before EDTA to prevent worsening of symptoms due to mobilisation of lead from tissues.

d) In severe poisoning without encephalopathy, BAL and EDTA are used until BL levels fall below 40 µg/100 ml.
e) EDTA therapy is continued until BL levels reduce to safe limits or for a maximum of three months.
f) Moderate poisoning is treated primarily with EDTA, followed by oral chelation once BL levels decrease.
g) Mild poisoning is managed with oral chelating agents such as D-penicillamine or DMSA.
h) DMSA (succimer) is preferred for oral therapy as it is more effective and less toxic than EDTA.

3. **Supportive treatment**
    a) Thiamine 10 to 50 mg/kg to improve neurological manifestations
    b) Calcium gluconate i.v. for colic
    c) Magnesium or sodium sulphate 8 to 12 g will change unabsorbed lead salts to highly insoluble lead sulphate and hasten its passage in the stools
    d) Anticonvulsants
    e) Correction of anaemia

Post-mortem Findings

- Pallor of organs
- Blue line on gums
- The stomach and intestines may show ulcerative or haemorrhagic changes and are contracted and thickened.
- The brain is very pale and greatly swollen.
- Degeneration of peripheral nerves
- Lead deposition in bones (lead lines)

Histopathological examination

- Bone marrow - hyperplasia of leucoblasts and erythroblasts with a decrease in fat cells.
- Eosinophilic intranuclear inclusions may be seen in hepatocytes and proximal tubules of the kidneys.