SEPTICAEMIA.

Bacteraemia : presence of viable bacteria in blood, no signs of disease. Dental procedures, tooth brushing may be a cause. Not significant except in heart valvular disease.

Septicaemia : presence of bacteria or their products in blood associated with disease.

Term used nowadays is sepsis :

Sepsis is the systemic inflammatory response to an infection where organisms or their toxins invade the blood stream causing illness manifested by fever or hypothermia (in elderly and very young), tachycardia, tachypnoea and may be associated with various degrees of injury to one or more major organs, Disseminated intravascular coagulation and microthrombi. Mild, moderate and severe. In its extreme it is defined as septic shock.

Decreased urinary output. Elevated blood sugar. Metabolic acidosis. Drop in blood pressure causing shock. Nausea and vomiting, diarrhoea. There is confusion and oedema.

Common primary sources of infection : lungs, abdomen, urinary tract. A source may not be apparent. Most commonly bacterial, but sometimes it may be viral or fungal.

Prompt diagnosis is essential. Blood culture for aerobic and anaerobic bacteria should be immediately taken and from other suspect sites of infection e.g. CSF, urine, wounds, respiratory secretions, IV devices.

This should not delay antibiotic treatment. Other investigations CT and X-rays . Exclude surgical emergencies that need prompt intervention.

Blood gases, lactate level, if BP is low then central venous pressure line and IV fluids.

DIC, damaged endothelia.

End organ dysfunction : brain, lungs, heart, liver, kidneys.

Pathogenesis :

Bacteria : LPS in gram negative. Gram positive exotoxins (superantigens), lipoteichoic acid, peptidoglycan, bacterial DNA, flagellin. Pattern recognition receptors leading to activation of cells of the immune system. Toll like receptors.

Bacterial toxins, (viruses) may act as superantigens, binding to MHC II and the beta chain of TCR leading to polyclonal T cell activation : interferon gama and activation of macrophages leading to TNF, IL-1 and IL-6.

Cytokines TNF, IL-1 and IL-6 can activate procoagulation factors in endothelium leading to endothelial damage and DIC. Adhesion molecules, neutrophils, release of enzymes, endothelial damage.

Role for complement. PAF, Leucotrienes. Neutrphil and platelet activation may all participate in the pathogenesis.

Impaired endocrine function. Hyperglycemia, Waterhouse-Fredericson syndrome.

Gram –ve : E. coli, Pseudomonas.

Gram +ve : S. aureus and Streptococcus.