Tetanus is caused by toxin from the bacterium Clostridium tetani and is characterised by muscle rigidity, spasms, and disturbance of the autonomic nervous system. Diagnosis is clinical, based on history and examination. Strychnine is a glycine agonist that may give rise to a similar clinical picture but muscle tone is usually normal between spasms and urinary strychnine will exclude this as a cause. Other differentials include oropharyngeal infections causing trismus, dystonic reactions or psychogenic illness.

General measures:
- Tetanus patients should be nursed in a quiet environment and all stimuli should be minimised.
- Prevent further toxin release; wounds should be cleaned and debried and antibiotics should be given.
- Airway management: airway management is a priority in tetanus.
- Generalised muscle spasm, laryngospasm, aspiration or large doses of sedatives may impair respiration and airway compromise should be anticipated.
- Copious bronchial secretions are seen in tetanus and frequent suctioning is required.

Antitoxin and immunisation:
- Antibiotics: metronidazole is the antibiotic of choice although penicillin is used throughout much of the world. Penicillin may worsen spasms; however, it is probably not associated with higher mortality.
- In tetanus, infection does not result in immunity; therefore, all patients should be actively immunised with a full primary immunisation course.

Further management:
- Consists of supportive care until the effects of bound toxin wear off.
- Sedation with benzodiazepines is the standard therapy for tetanus because they inhibit endogenous antagonists of GABAa receptors and may counteract the effects of tetanus toxin (large doses of 200mg per day or more are often required).
- Non-depolarising muscle relaxants may be required if spasms cannot be controlled with benzodiazepines. In this case, cardiovascularly inert agents like vecuronium are preferred to pancuronium because of the sympathomimetic side effects of the latter agent.
- Magnesium may be useful in treatment of spasms and may limit autonomic instability. Doses are titrated to a magnesium of 2 to 4 mmol/L.
- Autonomic instability is often difficult to treat and rapid fluctuations in blood pressure mean short acting agents are preferable.
- Patients with severe tetanus may require 2-3 weeks of IPPV until spasms subside and nosocomial pneumonia is an important complication that often arises in these patients.

Prognosis:
- If the disease is not treated, the mortality is >60% and higher in neonates.
- Mortality is 10-25% in units with good facilities.

Adverse prognostic features include:
- Incubation of <7 days
- Period of onset ≤48 hours
- Portal of entry from umbilicus, uterus, burns, open fractures or from IM injection
- Presence of spasm
- Temperature >38.4
- HR >120bpm in adults and >150bpm in neonates

Incubation is usually between 4 & 14 days with 90% of cases presenting within 15 days.
- Initial symptoms include muscle stiffness, with muscle groups with short neural pathways affected first; hence trismus and back pain are present in more than 90% of cases on admission.
- Involvement of the facial and pharyngeal muscles produce the characteristic risus sardonicus and dysphagia.
- Increased tone in the muscles of the trunk results in opisthotonus.
- Muscle groups adjacent to the initial site of infection are often particularly severely affected, producing an asymmetrical picture.

Pathophysiology:
- The toxin preferentially affects the GABA inhibitory interneurons afferent to the motor nerves in the spinal cord and the brainstem.
- By preventing inhibitory discharge, unrestricted motor nerve activity occurs, resulting in increased muscle tone and spasms characteristic of tetanus.
- In severe forms of tetanus, the autonomic nervous system is also affected, perhaps as a result of toxin action within the brainstem, giving rise to marked cardiovascular instability.

Clinical features:
- Muscle spasms are usually most severe during the 1st and 2nd weeks of illness but may persist for 3-4 weeks after which time rigidity may persist for many weeks.
- In severe tetanus, autonomic disturbance usually appears during the 2nd week.
- Signs of sympathetic overactivity usually predominate with periods of tachycardia & hypertension.
- Acute renal failure is a recognised complication of tetanus with dehydration, rhabdomyolysis due to spasms and autonomic disturbance all contributing.
- Other complications include tendon avulsions, vertebral fractures secondary to muscle spasm, GI bleeding, venous thromboembolism.