### Table 74.2  Progressive onset of major systemic symptoms and signs of untreated envenomation (in massive envenomation or in a child, a critical illness may develop in minutes rather than hours)

**< 1 h after bite**
- Headache
- Nausea, vomiting, abdominal pain
- Transient hypotension associated with confusion or loss of consciousness
- Coagulopathy (laboratory testing)
- Regional lymphadenitis

**1–3 h after bite**
- Paresis/paralysis of cranial nerves, e.g. ptosis, double vision, external ophthalmoplegia, dysphonia, dysphagia, myopathic facies
- Haemorrhage from mucosal surfaces and needle punctures
- Tachycardia, hypotension
- Tachypnoea, shallow tidal volume

**> 3 h after bite**
- Paresis/paralysis of truncal and limb muscles
- Paresis/paralysis of respiratory muscles (respiratory failure)
- Peripheral circulatory failure (shock), hypoxaemia, cyanosis
- Rhabdomyolysis
- Dark urine (due to myoglobinuria or haemoglobin)
- Renal failure

### Neurotoxins
Presynaptic and postsynaptic neuromuscular blockers present in all dangerous venomous snakes. May cause paralysis. Postsynaptic blockers readily reversed by antivenom. Presynaptic blockers are more difficult to reverse, particularly if treatment is delayed. Some presynaptic blockers are also rhabdomyolysins.

### Prothrombin activators
Present in many important species. Cause disseminated intravascular coagulation with consumption of clotting factors including fibrinogen. Intrinsic fibrinogen lysis generates fibrinogen degradation products. Significant risk of haemorrhage.

### Anticoagulants
Present in a relatively small number of dangerous species. Prevent blood clotting without consumption of clotting factors.

### Rhabdomyolysins
Some presynaptic neurotoxins also cause lysis of skeletal and cardiac muscle. Apart from loss of muscle of mass, may cause myoglobinuria and renal failure.

### Haemolysins
Present in a few species. Rarely a serious clinical effect.