Neuromuscular-blocking drug

Neuromuscular-blocking drugs block neuromuscular transmission at the neuromuscular junction, causing paralysis of the affected skeletal muscles. This is accomplished either by acting presynaptically via the inhibition of acetylcholine (ACh) synthesis or release or by acting postsynaptically at the acetylcholine receptors of the motor nerve end-plate. While some drugs act presynaptically (such as botulinum toxin and tetanus toxin), those of current clinical importance work postsynaptically.

In clinical use, neuromuscular block is used adjunctively to anesthesia to produce paralysis, firstly to paralyze the vocal cords, and permit intubation of the trachea, and secondly to optimize the surgical field by inhibiting spontaneous ventilation, and causing relaxation of skeletal muscles. Because the appropriate dose of neuromuscular-blocking drug may paralyze muscles required for breathing (i.e., the diaphragm), mechanical ventilation should be available to maintain adequate respiration.

Patients are still aware of pain even after full conduction block has occurred; hence, general anesthetics and/or analgesics must also be given to prevent anesthesia awareness.