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Drug-induced respiratory depression

Dr Bruno Mégarbane

Department of Medical and Toxicological Critical Care, Lariboisière Hospital, Paris Cité Université,

Aim and objectives: Drug-induced respiratory depression represents a life-threatening complication in the poisoned patient. Consistently, the current epidemic of opioid overdose fatalities is the consequence of opioid-induced depression of the brainstem centres of ventilation control. We aim to clarify the various mechanisms of drug-induced respiratory depression using examples of usually involved drugs and the consequent implication for the poisoned patient management.

Methodology: Narrative review of the literature.

Results: Respiratory depression leads to a type-2 respiratory failure combining hypoxemia + hypercapnia and results from various mechanisms including central hypopnea, upper airway obstruction, bronchospasm, and neuromuscular blockage. At the bedside, investigation based on the measurement of simple physiological parameters can allow easily distinguishing central (such as with opioids) from peripheral mechanisms of respiratory depression (such as with benzodiazepines). Identification of respiratory depression is based on the measurement of respiratory rate, SpO_2 and arterial blood gas. SpO_2 and respiratory rate are surrogate indicators of ventilatory drive but provide limited information on drug-related effects on ventilatory control. $PaCO_2$ and minute volume are direct measures of ventilation but difficult to assess continuously. Exposure to benzodiazepines, opioids, baclofen, and gamma-hydroxybutyrate represent public health concerns in the European countries and elsewhere. Understanding the exact mechanisms involved in respiratory toxicity is helpful to explain the observed clinical features and improve management of the resulting poisoning. In poisoned patients with consciousness impairment (Glasgow coma score <8), tracheal intubation is required although not systematic. Decision should consider the drug properties and its pharmacokinetics as well as the level of encephalopathy and respiratory/circulatory findings. Antidotes (naloxone and flumazenil, according to the toxidrome) can avoid intubation in selected patients. Their use is safe if respecting contra-indications.

Conclusions: Drug-induced respiratory toxicity represent a serious consequence in poisoning and should be systematically identified at the bedside and the more adequately managed in relation to the involved mechanisms.