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Local Anesthesia and Narcotic Drug Interaction in Pediatric Dentistry

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An uncooperative 3-year-old male child weighing 18.2 kg presented to a private office for multiple dental restorations. The medical history and physical examination revealed no apparent abnormalities. The child was classified American Society of Anesthesiologists Physical Status I.

At the first visit, a 10 mg dose of alphaprodine (0.55 mg/kg) plus atropine (0.2 mg) were injected into the mucobuccal fold. The degree of sedation and cooperation was clinically insufficient and the child was given another appointment. At the second appointment, a submucosal injection of 14.0 mg alphaprodine (0.77 mg/kg) plus 0.2 mg atropine into the mucobuccal fold of the posterior left maxillary arch was made. Within five minutes the patient was quiet and accepted the injection of half a standard dental cartridge of 2% lidocaine with 1 : 100,000 epinephrine. No physical restraint was required. A rubber dam was applied and the first cavity preparation begun.

Ten minutes after administration of the alphaprodine, the patient was responsive to verbal stimuli, although a decrease in respiratory rate was observed by the anesthesiologist. Within one minute, the airway became partially obstructed, as noted by audible snoring sounds. The patient became progressively unresponsive to verbal and tactile stimuli, necessitating discontinuation of the dental procedure. The rubber dam was immediately removed and a triple airway maneuver performed. Respirations were no longer evident, and an Ambu-bag with full face mask was immediately applied to provide positive pressure ventilation of 100% oxygen. Simultaneously, naloxone hydrochloride 0.2 mg was administered by deep intramuscular injection into the anterior thigh. Heart rate and blood pressure remained stable throughout the apneic period. Within five minutes of administration of the naloxone, respiratory depression was completely reversed and positive pressure ventilation was discontinued. The child was retained for observation for an additional 60 minutes, and then dismissed in an alert and oriented state with vital signs stable and within normal limits.[1]

Alphaprodine and other synthetic narcotics may augment the convulsant effects of local anesthetics either directly or indirectly. Because toxic doses of alphaprodine induce central nervous system changes (CNS) similar to local anesthetics (convulsions and respiratory depression), this interaction may represent, in part, a direct pharmacologic effect. However, the significant respiratory depressant effects of the narcotic, by inducing increased P_{CO_2} and decreased pH and P_{O_2}, may play an even more important role.[4] Elevated P_{CO_2}s appear to decrease the central nervous system threshold concentrations needed for local anesthetic-induced convulsions. Elevated P_{CO_2}s also increase cerebral blood flow, thereby increasing the CNS distribution of the local anesthetic. Additionally, acidosis can decrease the plasma protein binding of local anesthetics. With this release of previously bound drug, more local anesthetic is now available for redistribution to the CNS. All four mechanisms may perpetuate continuation of the convulsion and further increase CNS concentration of the local anesthetic.

What is clear regarding the various possible mechanisms for local anesthetic/narcotic interactions is that convulsions are not only more likely to occur but, after convulsions begin, the effects of further respiratory acidosis severely complicate recovery. Cardiac arrest becomes more likely as pH and P_{O_2} decrease and P_{CO_2} increases. The necessity of positive pressure O_2 in treating local anesthetic-induced convulsions cannot be overemphasized. Reducing the elevated P_{CO_2} caused by the inadequate ventilation during convulsions and/or the respiratory depression induced by the toxic levels of the narcotic and local anesthetic, is pivotal to recovery.

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