PROLONGED POSTOPERATIVE APNEA

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The initial management of the postoperative apneic or hypoventilating patient should include an assessment of the adequacy of both oxygenation and ventilation followed by appropriate supportive ventilatory management. Further evaluation proceeds in a systematic fashion beginning with a review of the past medical history and pertinent physical examination of the patient, including assessment of neuromuscular blockade and neurologic function. The anesthetic record and other perioperative records may give a clue to medications and/or untoward intraoperative events that may influence the postoperative course. If apnea persists, examine pertinent laboratory values, including serum electrolytes, calcium, magnesium, blood glucose, acid/base status, ABG, and renal/hepatic function.

A. Review the records for narcotic analgesic dosages, including any premedication. In the trauma patient, consider preinjury drug and/or alcohol ingestion. Naloxone, 0.1–0.2 mg IV, may reverse narcotic-induced respiratory depression but is not entirely innocuous. Nalbuphine may also reverse narcotic-induced respiratory depression. Flumazenil, 0.1–0.2 mg IV, may be titrated to reverse sedation caused by benzodiazepine administration. Physostigmine, 15 μg/kg/IV, may reverse depression from sedatives or tranquilizers. Residual neuromuscular blockade may result from relative overdose or impaired excretion of nondepolarizing relaxant and may be avoided by monitoring neuromuscular blockade intraoperatively. Repeating anticholinesterase (ACE) agents (neostigmine, 60 μg/kg up to 5 mg, or pyridostigmine 0.35 mg/kg up to 25 mg) may be effective. Residual succinylcholine effect can be seen in patients with abnormal pseudocholinesterase (PCE). Typically, *deficiency* of PCE activity (severe liver disease, malnutrition, pregnancy or after plasmapheresis, anticholinesterase therapy for myasthenia gravis [MG], echothiophate eye drops for glaucoma, and/or the use of metoclopramide[1]) does not prolong succinylcholine-relaxation beyond 30–60 minutes. Neurally applied opiates, such as morphine, may cause a biphasic respiratory depression and delayed awakening after perioperative administration.[2] The early phase reflects systemic absorption and produces respiratory depression and sedation of a similar magnitude to equipotent doses of parenterally administered narcotics. The later phase reflects the rostral spread in the CSF to depress the brainstem respiratory centers. Aminoglycoside antibiotics may cause postoperative apnea. Drugs that cause paralysis include virtually all the "mycin" drugs and others. Reversal is inconsistent and unpredictable.

B. Patients who chronically retain CO_2 may hypoventilate when exposed to high FiO_2. Reduce FiO_2 when ABG results dictate. Damage to the CNS, such as increased ICP or CVA, and cervical cordotomy for chronic pain, may cause apnea. Neurologic consultation is recommended, and diagnostic neuroimaging may be necessary. Renal and/or hepatic dysfunction may prolong the effects of many anesthetic agents and delay emergence. Patients with MG or other neuromuscular diseases may exhibit postoperative apnea, and anticholinesterase therapy may be effective in those cases. Patients with hereditary hepatic porphyrias may develop porphyric attacks, manifested by neuromuscular weakness, after being given thiopental; ventilation is supported until the weakness resolves. Electrolyte derangements, including abnormalities in calcium and magnesium, as well as hypo/hyperglycemia, may contribute to prolonged awakening. Laboratory evaluation and correction of any abnormalities should facilitate return of motor and mental function.

C. Hypothermia exacerbates neuromuscular blockade, and chilled patients may become apneic even in the absence of muscle relaxants.[3] Rewarming usually reverses these abnormalities. Intraoperative hyperventilation leads to loss of CO_2 from the body. Postoperatively, $PaCO_2$ may rise slowly and cause a lack of respiratory stimulus for ventilation. Careful observation of capnography intraoperatively should prevent this; however, when hyperventilation is intentionally induced, it may be difficult to avoid this situation.

References

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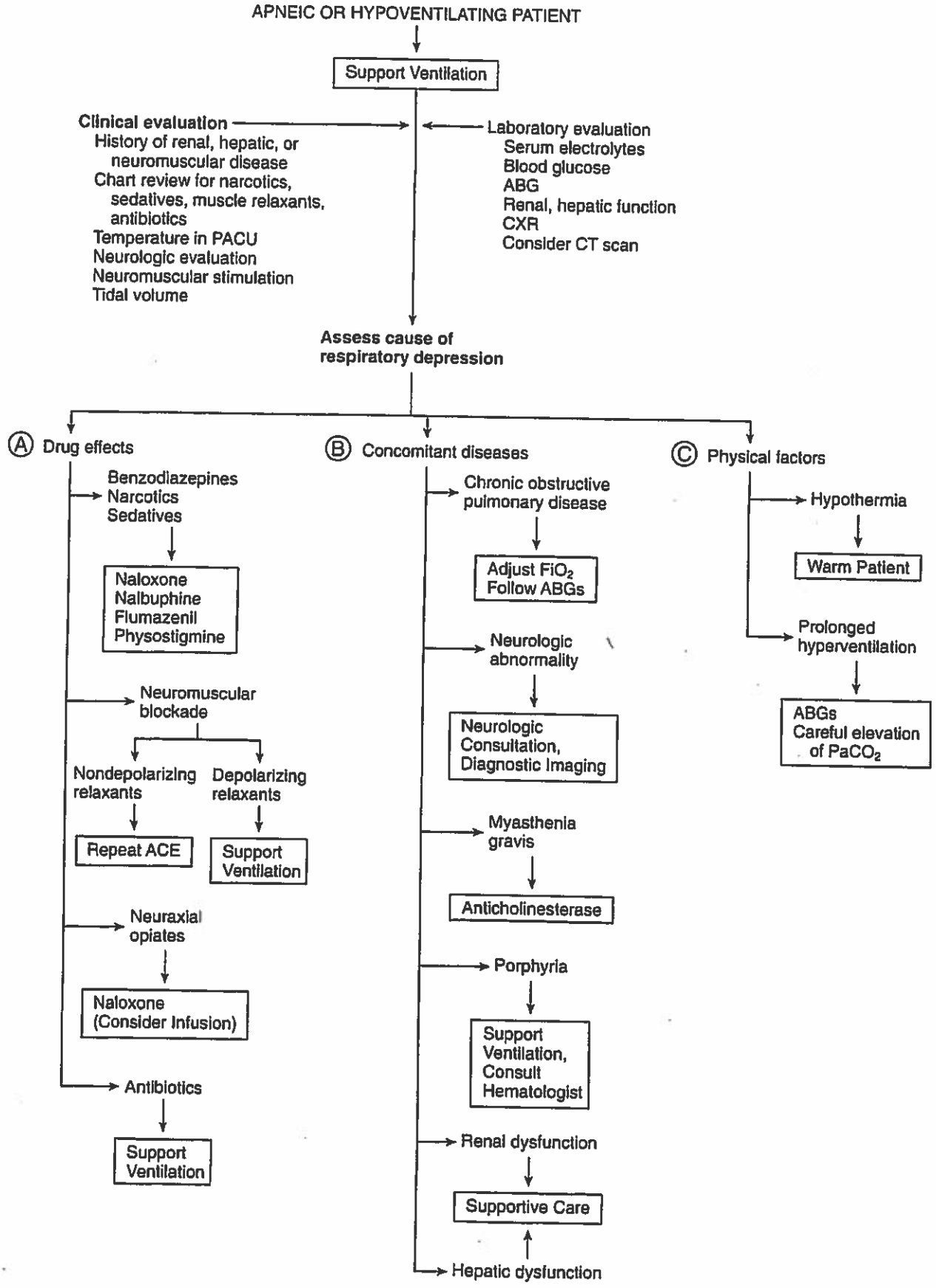
2. Etches RC, Sandler A, Daley MD: Respiratory depression and spinal opioids. Can J Anaesth 1989; 36:165.
3. Denlinger JK. Prolonged emergence and failure to regain consciousness. In: Gravenstein N, Kirby RR, eds. Complications in anesthesiology. Philadelphia: Lippincott-Raven, pp 441–450, 1996.


APNEIC OR HYPOVENTILATING PATIENT
Support Ventilation
Clinical evaluation
History of renal, hepatic, or neuromuscular disease
Chart review for narcotics, sedatives, muscle relaxants, antibiotics
Temperature in PACU
Neurologic evaluation
Neuromuscular stimulation
Tidal volume
Laboratory evaluation
Serum electrolytes
Blood glucose
ABG
Renal, hepatic function
CXR
Consider CT scan
Assess cause of respiratory depression
Ⓐ Drug effects
Benzodiazepines
Narcotics
Sedatives
Naloxone
Nalbuphine
Flumazenil
Physostigmine
Neuromuscular blockade
Nondepolarizing relaxants
Depolarizing relaxants
Repeat ACE
Support Ventilation
Neuraxial opiates
Naloxone (Consider Infusion)
Antibiotics
Support Ventilation
Ⓑ Concomitant diseases
Chronic obstructive pulmonary disease
Adjust FiO_2
Follow ABGs
Neurologic abnormality
Neurologic Consultation, Diagnostic Imaging
Myasthenia gravis
Anticholinesterase
Porphyria
Support Ventilation, Consult Hematologist
Renal dysfunction
Supportive Care
Hepatic dysfunction
Ⓒ Physical factors
Hypothermia
Warm Patient
Prolonged hyperventilation
ABGs
Careful elevation of $PaCO_2$