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Respiratory Dysfunction Associated with Acute Cerebrovascular Events

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Cerebrovascular events (CDVE) are a major cause of morbidity and mortality worldwide. Most patients with CVE do not develop significant respiratory problems but when present, they may be a marker of severe neurologic derangement. In one study (1), only 8% of patients presenting with acute carotid territory stroke were electively intubated and mechanically ventilated due to neurologic deterioration. "Good" outcome in terms of survival and neurological status of patients with hemispheric strokes who required mechanical ventilation have been reported in approximately 20% of cases (2). Respiratory disturbances associated with strokes can result from discrete or diffuse lesions to key components of the respiratory controller. The clinical spectrum of respiratory disorders in stroke include abnormal breathing patterns, hypoxemic and hypercapnic respiratory failure, aspiration pneumonia due to an inability to protect the airways and clear the airway by coughing, and acute pulmonary embolism due to prolonged immobilization. There is a variety of altered respiratory patterns associated with strokes. There is a variety of altered respiratory patterns associated with strokes (3-5). These changes are not only important in determining the location of the neuroanatomic lesion, but they have also been regarded as outcome predictors in CVE. This paper reviews the evaluation, management, and effect of respiratory care interventions, management, and effect of respiratory care interventions on a variety of respiratory system problems in patients with CVE.

SUPRATENTORIAL STROKES

Hemispheric strokes have been associated with a modest reduction of the contralateral chest wall and diaphragm movements (6,7). On the other hand, bilateral hemispheric dysfunction has been typically associated with Cheyne-Stokes respiration (8); a periodic breathing pattern characterized by progressive hyperpnea followed by hypopnea and apnea. Cheyne-Stokes respiration is frequently described in patients with hemispheric CVE and significant cardiac and pulmonary diseases but it has also been noted in patients with brainstem strokes.

The exact mechanism of this form of breathing pattern is not completely understood. In patients with bilateral hemispheric lesions, an increased response to carbon dioxide (CO₂), presumably due to loss of the normal cortical inhibition, has been described (5). In patients with congestive heart failure and pulmonary disorders, circulatory slowing and hyperventilation with or without hypoxemia are instability. Cheyne-Stokes respiration is typically present in the transition from wakefulness to sleep (drowsiness). Cheyne-Stokes respiration is typically present in stroke patients with reduced level of consciousness of in the transition from wakefulness to sleep (drowsiness) and the first two stages of non-rapid eye movement sleep (NREM). Cheyne-Stokes respiration with hypocapnia has been associated with high mortality (9,10). Conversely, intermittent Cheyne-Stokes respiration does not necessarily indicate a poor prognosis and it usually disappears with the improvement in neurological status.

INTRATENTORIAL STROKES

Central neurogenic hyperventilation (CNH), is described in patients with midbrain and upper pontine tegmentum lesions (11,12). To be considered neurogenic in origin, such hyperventilation must be accompanied by a normal PaO₂, as response to hypoxic drive can mimic this breathing pattern. True CNH seems to occur rarely and it has been associated with poor clinical outcome. Most hyperventilation in stroke patients can be explained by reflex stimulation of the respiratory center by hypoxia, activation of

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lung and chest wall receptors, and metabolic factors (i.e., metabolic acidosis with Kussmaul's breathing).

Apneustic breathing (13) with its prolonged inspiratory pauses indicates a pontine stroke. Damage to the low pons or high medulla can produce an abnormal breathing pattern characterized by normal breaths separated by irregular pauses or cluster breathing. Grossly irregular breathing occurs in patient with medullary lesion. This ataxic breathing pattern is an ominous sign and usually precedes the patient's demise.

A rare breathing disorder in which the volitional breathing controller is preserved but automatic breathing is impaired (Ondine's curse) (14,15) can be seen in ischemic lesions involving the lateral tegmentum points and medulla due to distal vertebral artery occlusion (lateral medullary stroke). Clinically, patients can maintain fair to adequate ventilation while awake but they develop severe hypoventilation and life-threatening central apneas during sleep (16). Invasive mechanical ventilation is often needed initially to maintain ventilation. Stable patients can be managed by noninvasive positive pressure ventilation during sleep. The opposite pattern, that is, preservation of automatic breathing with inability to voluntarily modify the breathing depth or frequency has been described in patients with extensive midpontine infarcts ("locked-in syndrome"), lacunar infarcts in the upper pontine corticospinal tract, and presumably in bilateral hemispheric lesions (17).

Finally, increased intracranial pressure leading to transtentorial herniation produces a typical rostrocaudal deterioration in which normal breathing is replaced by Cheyne-Stokes respiration, followed by neurogenic hyperventilation and subsequently, ataxic respiration that precedes apnea (18).

RESPIRATORY FAILURE IN STROKE

Patients with acute strokes may not be able to maintain adequate oxygenation and/or ventilation in the absence of an intracardiac shunt (acute respiratory failure) (19). In fact, respiratory arrest represents a primary cause of death in the first few days after a stroke (20). Depending on the pathophysiologic mechanism, patients may present with hypercapnic (pump failure) or hypoxemic respiratory failure. The hallmark of ventilatory pump failure is hypercapnia with acute respiratory acidosis. In acute stroke patients, this hypoventilation is most commonly due to a decreased total minute ventilation secondary to a reduced central respiratory drive. The alveolar-arterial oxygen gradient ($P(A-a)O_2$) is useful in determining if the hypoxemia present in this form of respiratory failure is only due to hypoventilation (normal gradient) or if there is additional parenchymal lung disease (elevated gradient). Hypercapnia may also result from concomitant abnormalities in one or several of the determinants of the carbon dioxide level in arterial blood [$PaCO_2 = k VCO_2/VE(1-VD/VT)$] (Table 1).

Table 1. Causes and pathogenesis of respiratory failure in acute stroke.

Increased CO_2 production (VCO_2) in patients with fever, sepsis, agitation or excessive carbohydrate load, associated with a limited ventilatory capacity (e.g., high VD/VT , low minute ventilation)

Increased dead space (VD/VT) in severe COPD, cystic fibrosis and severe asthma.

Decreased total minute ventilation (VE) due to other conditions associated with ventilatory pump dysfunction:

Decreased central respiratory drive: drugs (narcotics, sedatives, anesthetics), central hypoventilation, hypothyroidism.

Abdominal respiratory efferents

Spinal Cord: traumas, poliomyelitis, amyotrophic lateral sclerosis, tetanus, rabies. Neuromuscular: Myasthenia gravis, multiple sclerosis, botulism, Guillain-Barre Syndrome, hypophosphatemia, hypomagnesemia, drugs (streptomycin, amikacin, neuromuscular blockers), polyneuropathy of critical illness, bilateral phrenic nerve injury.

Abnormal chest wall and/or muscles: Severe kyphoscoliosis, ankylosing spondylitis, massive obesity, muscular dystrophy, polymyositis, respiratory muscle fatigue, acid maltase deficiency.

Airway obstruction: epiglottitis, fixed and variable upper airway obstruction due to tumors, post-extubation, tracheomalacia, bilateral vocal cord paralysis.

The hallmark of hypoxemic respiratory failure is an inability to adequately oxygenate the blood. The main pathophysiologic mechanisms involved are V/Q mismatch (response to 100% O₂) and intrapulmonary shunting (no significant improvement with 100% O₂). The patients typically exhibit a rapid shallow breathing pattern and a low or normal CO₂. This form of respiratory failure is commonly the result of a diffuse acute lung injury with high permeability pulmonary edema (ARDS), severe pneumonia, or cardiogenic pulmonary edema. Rarely, patients with acute brain injury can present with a form of non-cardiogenic pulmonary edema termed neurogenic pulmonary edema (NPE) (21). The exact mechanism of NPE is unknown but massive adrenergic overactivity with pulmonary vasoconstriction has been implicated (22,23).

The management of acute respiratory failure is initially supportive aimed at the correction of hypoxemia or hypercapnia until specific actions are implemented to correct, if possible, the factors that lead to the respiratory failure. The main goal in managing hypercapnic respiratory failure is to improve alveolar ventilation through the use of mechanical ventilation. This is most commonly done through an endotracheal tube using a volume-controlled ventilator (usual initial settings are: tidal volume (VT) 10 ml/kg, assist-control mode (A/C), rate 8-10 breaths per minute, and fractional concentration of inspired oxygen (FiO₂) 1.0). Arterial blood gases (ABGs) must be checked 10-20 min later to detect inadvertent and potentially life-threatening acute alkalosis secondary to overcorrection of the hypercapnia and to adjust the FiO₂. Non-invasive mechanical ventilation could only be tried for cooperative, hemodynamically stable stroke patients with intact upper airway function without excessive secretions, regurgitation, or vomiting.

Patients with V/Q mismatch abnormalities without significant intrapulmonary shunt will usually respond to non-invasive O₂ supplementation (i.e., nasal cannula, Venturi mask). In patients with cardiogenic pulmonary edema, the use of CPAP (5-10 cmH₂O) via a face mask in addition to oxygen supplementation can be beneficial by reducing the transmural pressure of the left ventricle, and therefore afterload but also by decreasing the preload. The ventilatory management of patients with diffuse acute lung injury (i.e., ARDS) requires mechanical ventilation and should be viewed as a balance between adequate oxygenation on one hand, and the risk for barotrauma and cardiovascular compromise on the other. A detailed discussion of the ventilatory management of acute lung injury/ARDS is beyond the scope of this review, but a special effort should be made to deliver enough oxygenation and maintain adequate mean arterial pressure in stroke patients to avoid further increased intracranial pressure and ischemia.

Adequate arterial oxygen saturation is usually achieved by raising the FiO₂ and increasing the end-expiratory lung volume to recruit collapsed or flooded alveoli. This can be achieved by adding extrinsic end-expiratory positive pressure (PEEP) and/or setting the ventilator to create dynamic hyperinflation (auto-PEEP). It is unclear if one strategy is more effective than the other in ARDS but in stroke patients with increased intrathoracic pressure with its impedance of cerebral and systemic venous return (25,26). Since the effects of PEEP are not easily estimated, intracranial pressure monitoring is recommended if levels > 10 cm H₂O are used. Concomitant elevation of the upper part of the body (with the head in neutral position) also helps to optimize cerebral venous return. To avoid potential oxygen toxicity, it is recommended not to use 100% O₂ for more than a few hours and to maintain an FiO₂ for more than a few hours and to maintain an FiO₂ SYMBOL 163f "Symbol" 0.6. To avoid barotrauma, it is recommended to maintain plateau pressure (end-inflation hold pressure) less than 25 cm H₂O. The VT chosen should be one that prevents lung overinflation (i.e., plateau pressure <35 cm H₂O) and, alveolar derecruitment at the end of expiration (inadequate oxygenation). This usually means an initial VT of 7-8 ml/kg that may need to be reduced to 5 ml/kg.

Allowing CO₂ retention and respiratory acidemia in an effort to limit barotrauma (through reductions in rate and VT) is referred to as "Permissive Hypercapnia". Although widely used for the majority of ARDS patients, this strategy should be avoided in CVA patients due to the deleterious effect of CO₂ and acidemia on cerebral blood flow (27).

AIRWAY MANAGEMENT IN ACUTE STROKE

Strokes resulting in depressed level of consciousness or blbar dysfunction may cause abnormal swallowing mechanism and altered cough and gag reflexes that compromise the airway patency. Early identification of such high-risk patients may avoid aspiration of gastric or oropharyngeal contents. Simple routine measures to reduce the risk of aspiration include the use of frequent suctioning of secretions, elevation of the head of the bed, and avoidance of oral intake, at least in the acute period. Nearly 30% of patients experience neurogenic dysphagia following stroke (28,29). After the acute period resolves, swallowing study with modified barium protocol should be obtained if abnormal voluntary cough and gag reflexes or dysphonia are present (30,31), or if supervised feeding is associated with coughing, regurgitation or respiratory distress. According to the degree of dysphagia, oral feeding can still be possible if eating is done only in the upright position, optimal dietary texture is selected, and small size boluses (i.e., ≤ 1 teaspoon) are given under direct supervision. With severe swallowing problems or episodes of recurrent aspiration, placement of a gastric (i.e., percutaneous endoscopic gastrostomy PEG) or small bowel tubes is indicated.

Prophylactic endotracheal intubation should be considered if patients are not able to protect their airways. This decision should take into consideration the severity and prognosis of the neurological damage, co-morbid conditions, and prior patientsi and proxyis wishes regarding invasive support (1,2,32,33). Other common indications for endotracheal intubation in stroke patients are tonic-clonic seizures or status epilepticus, respiratory failure due to pneumonia and pulmonary edema and therapeutic hyperventilation (1,2).

Physicians should be aware of the potential risks of performing endotracheal intubation in neurological patients who are prone to develop or already have increased intracranial pressure (34-36). In this patients, use of thiopental, nebulized or intravenous lidocaine, and an alpha/beta blocker such as labetalol would prevent further raising in intracranial pressure, coughing and sympathetic response to intubation (37).

INCREASE INTRACRANIAL PRESSURE AND MECHANICAL HYPERVENTILATION

Mechanical hyperventilation will acutely and transiently lower intracranial pressure (24). Cerebral blood flow is linearly related to PaCO₂ between 25 and 80 mm. Hg in normal brain. Lowering the PaCO₂ level to 25-30 mm Hg remains a useful emergency therapy for acutely elevated intracranial pressure although, worsening of ischemia due to vasoconstriction in infarct penumbral areas has been postulated as a potential risk (1). The effects of mechanical hyperventilation are short-lived, probably up to 24 hours. Return to normocarbica should be done gradually over 24-48 hours. In patients with otherwise normal lungs, noninvasive continuous monitoring with an infrared capnograph in line with the ventilator can avoid multiple ABGs determinations.

PATIENT-VENTILATOR INTERACTIONS IN STROKE

The output of the respiratory center, clinically expressed as respiratory muscles contraction, can be altered by an external stimuouss such as mechanical ventilation. Most patients intubated and mechanically ventilated continue to make spontaneous respiratory efforts. If the magnitude of the inspiratory efforts is sufficient, each effort triggers a machine breath, i.e., the respiratory pump entrains the ventilator. On the other hand, when teh amplitude os spontaneous inspiratory efforts is small relative to the sensitivity of the machine triggering mechanism, increases in spontaneous respiratory frequency are not expressed as changes in actual machine rate and minute ventilation (Figure 1). This is often the case in stroke patients requiring mechanical ventilation who have depressed sensorium with reduced respiratory drive. These "wasted respiratory efforts" represent a form of patient-ventilator asynchrony that is not only associated with increased in oxygen consumption and patient discomfort but they can also produce an irregular breathing pattern which could mislead the evaluthing pattern wich could mislead the evaluating physician.



Figure 1. Airway pressure (PAW) and flow (V) tracings in an intubated mechanically ventilated patient. Nota asynchronous spontaneous respiratory efforts (arrows) during machine deflation. (Dots: mechanical inflation)

Interactions between the ventilator and the patient's respiratory system can mimic breathing patterns observed in non-intubated patients with different levels of central neurological dysfunction. Figure 2 shows airway pressure and flow tracing in one of our patients with brainstem ischemic stroke. At first glance, it may seem that he exhibits a form of irregular periodic breathing. Careful evaluation of respiratory muscles contraction by physical exam and analysis of airway and flow tracings during the presumed "apneic" intervals demonstrate a regular tachypneic intrinsic pattern (Figure 2-arrows). Here, some inspiratory efforts are able to trigger machine breaths (dots). With each subsequent mechanical inflation, the operating lung volume increases (manifested by a progressive increase in peak pressures) to a point that the patient's effort cannot overcome the lung elastic recoil pressure plus the machine triggering sensitivity and there is a failure to trigger a mechanical breath. When this patient was allowed to breathe spontaneously with the assistance of continuous positive airway pressure (CPAP), the underlying tachypneic pattern was uncovered (Figure 3). This tachypnea was persistent despite normal oxygenation and lung mechanics, leading to a diagnosis of central neurogenic hyperventilation.

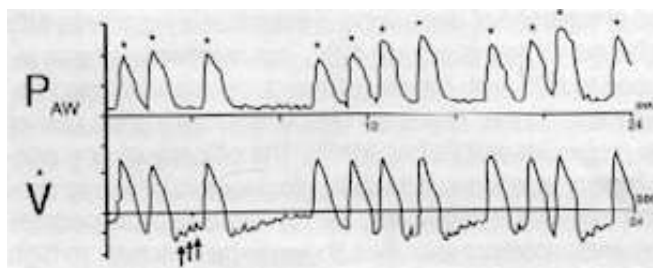


Figure 2. Patient-ventilator asynchrony in a patient with brainstem stroke mimicking irregular periodic breathing. (Dots: mechanical inflation; arrows: spontaneous respiratory efforts. See text for explanation)

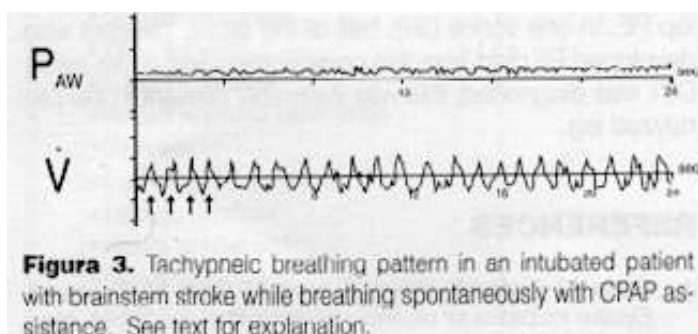


Figure 3. Tachypneic breathing pattern in an intubated patient with brainstem stroke while breathing spontaneously with CPAP assistance. See text for explanation.

An intrinsic periodic breathing pattern can be precipitated by the way physicians set the mechanical ventilator. This is particularly seen in patients with central neurologic damage and some degree of altered level of consciousness or during sleep. Most mechanically ventilated patients are maintained at settings resulting in nearly "normal" PaCO₂ which in turn maintain recruitment of the respiratory muscles. Following the transition to NREM sleep stage 1, the normal response is an increase in the recruitment threshold of respiratory muscles for CO₂. If the ventilator settings are unchanged during sleep time, it may be possible that PaCO₂ now falls below the operating recruitment threshold for CO₂ and apnea develops. If the patient's backup ventilator rate is low enough as it is usual in stable ventilator dependent patients, enough as it is usual in stable ventilator dependent patients (e.g., SIMV 4 breaths per minute or using pressure support ventilation (PSV mode)) this prolonged central apnea can cause cortical arousal and therefore, sleep disruption. The easiest way to overcome this ventilator-triggered

periodic breathing is to increase the ventilator frequency at night when using SIMV or A/C modes of ventilation and avoiding using PSV at night as the sole mode of ventilation.

VENOUS THROMBOEMBOLISM IN ACUTE STROKE

The prevalence of deep venous thrombosis in patients with ischemic strokes is around 42%. This number has been reduced to 26% with the use of low-dose unfractionated heparin and further down to 17% with prophylactic use of low-molecular-weight heparin (38). The efficacy of pneumatic leg compression and elastic stockings in ischemic stroke is not well established. But in neurosurgical patients pneumatic compression has shown to be effective. In high risk is contraindicated, an inferior vena cava filter (e.g., Greenfield filter) should be considered.

Data on the prevalence of pulmonary embolism (PE) after strokes is incomplete but it is estimated that 13% of all stroke patients or 22% of stroke patients with DVT will develop PE. In one series (39), half of the stroke patients who developed PE died from this complication. Moreover, when DVT was diagnosed, this was invariably present in the paralyzed leg.

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