High-frequency pulsation (HFP) in a patient with Guillain-Barré syndrome

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Abstract. A patient with Guillain-Barré syndrome (GBS) developed a respiratory crisis despite recurrent treatment with plasma exchange. Thus mechanical ventilatory support became necessary. As an alternative to conventional ventilatory techniques high-frequency pulsation (HFP), a modified high-frequency jet-ventilation technique was used. According to the observations HFP may be a valuable technique for the continuous adaptation to the patient's individual respiratory demands in GBS.

Key words: High-frequency jet-ventilation — High-frequency pulsation (HFP) — Guillain-Barré syndrome (GBS)

Rapidly ascending motor paralysis due to neuronal inflammation is characteristic in patients with Guillain-Barré syndrome (GBS). When respiratory muscles are paralyzed pulmonary complications (atelectasis, bronchopulmonary infections) are frequently observed [1]. Since the early use of plasma exchange (PE) has been established as a main goal of neurologic therapy, the number of pulmonary complications has significantly decreased. Additionally these patients require less mechanical ventilatory support and are weaned earlier from the ventilator [2, 3]. Thus a reduction in the specific problems related to mechanical ventilation is provided. However, if neuromuscular respiratory failure occurs, mechanical ventilation becomes an increasingly important therapeutic adjunct to PE in the management of GBS. Although various conventional ventilatory techniques are able to tide a patient over a respiratory crisis, high-frequency ventilatory techniques are said to provide additional benefits in terms of continuous adaptation to the patient’s respiratory demands. The following report presents a patient with GBS who, apart from routinely given therapies (PE, respiratory physiotherapy) required ventilatory support due to respiratory insufficiency. As an alternative to conventional ventilatory techniques high-frequency ventilation was used. In this special case high-frequency pulsation (HFP), a modified high-frequency jet-ventilation technique, was applied.

Case report

A 39-year-old man with suspected GBS had been admitted to the neurologic ward. Clinical neurologic examination, cerebrospinal fluid analysis (protein 87 mg/dl, lymphocytes 2 cell/mm³) as well as pathologic velocity of nerve conduction supported the diagnosis. Initially he was given one single oral dose of corticosteroid as immunosuppressive (Urbason® 40 mg). During the following days four PE procedures were performed (volume exchanged: 1700 ml, 1800 ml, 1420 ml, 2117 ml; replacement fluid — albumin). After a slight temporary improvement his neurologic status deteriorated. The patient was transferred to our ICU due to respiratory deterioration.

On admission (day 24 after onset of GBS) arterial blood gas analysis (BGAa) was performed [FiO₂ approx. 0.5 (face mask – 10 l/min): PaO₂ 9.7 kPa, PaCO₂ 4.9 kPa] (Fig. 1). Fiberoptic bronchoscopy showed massive viscous secretion and obstruction of the right lower lobe bronchus, whereas chest X-ray revealed paracardiac opacification with elevation of the diaphragm on the right. After careful consideration intermittent CPAP (+ 5 cmH₂0) and intensive respiratory physiotherapy was felt to be justified.

On the next day (day 25), however, a decrease in oxygenation was observed [BGAa–FiO₂ 0.7: PaO₂ 8.2 kPa, PaCO₂ 4.9 kPa] (Fig. 1). At CPAP HFP (F=150/min) CPAP

Fig. 1. Alterations in PaO₂ and PaCO₂ during oxygen therapy, CPAP (interm: intermittent CPAP via face mask; cont: continuous CPAP via tube) and HFP in a patient with Guillain-Barré syndrome. PE: plasma exchange;*: fiberoptic bronchoscopy;+: intubation;–: extubation; X-ray: chest X-ray
this point endotracheal intubation was performed. In addition further aggressive therapeutic efforts [continuously applied CPAP (+5 cmH₂O), recurrent fiberoptic bronchoscopy] were made. Several hours later BGAa had not improved to the expected extent [BGAa = FiO₂ 0.7; PaO₂ 9.1 kPa, PaCO₂ 5.1 kPa; "Quotient": 0.85] [4]. Moreover chest X-ray revealed an intensifying paracardiac opacity (Fig. 3b, c) it was possible to continuously draw by intermittently performed suction manoeuvres.

**Technique of high-frequency pulsation (HFP) (Fig. 2)**

HFP is operated by means of a combination of a specially designed patient-adaptor [5] with a commercially available high-frequency jet-ventilator (Universal Jet Ventilator AMS 1000, Acutronic, Switzerland). This HFP-adaptor is attached to the top of a conventional endotracheal tube. High pressure gas impulses are injected through a nozzle on top of the horizontal branch of a T-piece. The jet pulses discharge into the Venturi-throat that is part of the vertical branch of the T-piece. Airway pressures are measured at the patient adaptor.

If dangerous levels of pressure are reached a fast responding automatic shut off system is activated. The essential part of this system is a bias flow passed through to the T-piece. This bias flow (25 l/min) of humidified and heated gas is used to provide entrainment of fresh gas at adjusted FiO₂ levels. This will guarantee constant gas mixture and prevent rebreathing of CO₂ by the patient. Humidification of the high humidified and heated gas is used to provide entrainment of flesh gas. This HFP is operated by means of a combination of a specially designed humidifier (Heated Humidifier AMS 1020, Acutronic, Switzerland). Humidification as well as heating of the bias flow gas are performed by special equipment (Conchatherm®, Kendall, West Germany).

After onset of HFP in our patient (initial ventilator settings: f = 150/min, driving pressure = 1.1 bar, FiO₂ = 0.7, I:E = 50% - adapted to continuously monitored oxygen saturation and intermittent BGAa) PaO₂ increased persistently at sufficient CO₂ elimination (Fig. 1). The evident increase in bronchial secretion was consequently withdrawn by intermittently performed suction manoeuvres.

Due to normal recurrent BGAa and a gradual dispersion of opacification in chest-X-ray (Fig. 3b, c) it was possible to continuously reduce the support from HFP (Fig. 1). Thus an immediate change from controlled mechanical ventilation to spontaneous breathing was rendered feasible.

On day 27 it became obvious that the last two PE treatments (four PE had been performed during ventilatory therapy; volume exchanged = 2146 ml, 2332 ml, 2745 ml, 2764 ml; replacement fluid – albumin) had effected an improvement of the patient’s neurologic condition [only paraplegia of the lower limbs was diagnosed as opposed to tetraparesis with bulbar symptoms (day 26) similar to the day of admission to our ICU (day 24)]. Thereafter, the patient was rapidly weaned from the ventilator by progressively decreasing the driving pressure. After a brief change from HFP to continuous CPAP augmenting spontaneous respiration the patient was extubated.

On the 28th day the patient was transferred back to the neurologic ICU.

**Discussion**

This report presents a patient with GBS and respiratory insufficiency. Although PE was effected early he developed respiratory failure necessitating mechanical ventilatory support. As an alternative to conventional ventilatory techniques we decided to use high-frequency pulsation (HFP, a modified high-frequency jet ventilation technique, which has recently been proven to be safe in terms of gaseous exchange and hemodynamic side effects [6].

In the acute phase of GBS respiratory failure due to respiratory muscular weakness is induced by a deterioration in lung and chest wall mechanics and a reduction in lung volumes. This leads to a loss of alveolar surface area. Therefore a deterioration in the oxygenation properties of the lungs occurs. If weakness progresses the fall in tidal volume reduces alveolar ventilation which leads to hypercapnia and further hypoxemia. Both impaired gaseous exchange and increased energy demands due to the increased work of breathing may shift the process providing energy for the respiratory muscles from aerobic to anaerobic metabolism thus accelerating the development of respiratory muscle fatigue [7]. In addition to the neuronally mediated muscular weakness this mechanism may further increase the extent of respiratory failure.

In our patient HFP was advantageous because, in contrast to conventional ventilators, no change of ventilatory system was necessary throughout the weaning process. This is due to the open system of HFP which guarantees immediate adaptation to continuously changing ventilatory requirements. Initially, during the period of respiratory muscle paralysis and fatigue, controlled mechanical ventilation, as the main goal of respiratory therapy, was granted. However, as sufficient muscular strength was gained due to recurrent PE, spontaneous breathing activities were possible throughout the ventilatory cycle. Thus respiratory muscle training was provided and the risk of muscle atrophy in case of long term ventilatory dependence reduced. Moreover HFP enabled our patient to compensate for the increased inspiratory work that was evident while he was breathing spontaneously through the endotracheal tube. Thus a further impact on respiratory muscular fatigue interfering with successful weaning from mechanical ventilation was avoided.

If mechanical ventilatory support is required in patients with GBS the applied ventilatory technique should be able to counteract the loss in lung volume as both the recruitment of alveolar surface area and the maintenance of lung volume are essential for oxygenation. As can be seen from our patient (Fig. 1) the onset of HFP immediately improved oxygenation. This effect can be explained by intrinsic PEEP phenomena regularly occurring during