Dysfunction of the upper airways has only recently been recognized in patients with Parkinson's disease [2]. In most cases, this leads to impairment of static and dynamic pulmonary function. In some patients, laryngeal involvement was the main reason for airway obstruction [2]. In our patient, the most likely diagnosis is primary laryngospasm associated with Parkinson's disease.

Treatment by minitracheostomy connected to a continous flow CPAP resulted in a clinically relevant relief of stridor.

The mechanism of this effect might be the slight positive airway pressure of 2–4 cmH_2O in combination with a 4-mm free artificial airway. However, the latter cannot be the only explanation for the clinical relief of stridor, since the stridor increased while the patient was breathing through an open minitracheostomy without CPAP connection. Although minitracheostomy is most frequently used in the treatment of sputum retention, it allows an artifical airway to be combined with several other arrangements [3, 4].

In conclusion, laryngospasm caused by dysfunction of recurrent laryngeal nerves may be associated with Parkinson's disease. CPAP via minitracheostomy proved to be temporarily successful in the management of this problem. Tracheostomy may be inevitable in case of persistent relapses.

References

1. Vincken WG, Gauther SG, Dollfuss RE, Hanson RE, Darauay CM, Cosio MG (1984) Involvement of upper airway muscles in extrapyramidal disorders. N Engl J Med 311:438–442
2. Izquierdo-Alonso JL, Jimenez-Jimenez FJ, Cabrera-Valdivia F, Mansilla-Lesmes M (1994) Airway dysfunction in patients with Parkinson's disease. Lung 172:47–55
3. Squires SJ, Frampton MC (1986) The use of minitracheostomy and high-frequency jet ventilation in the management of airway obstruction. J Laryngol Otol 100:1199
4. Iapichino F, Gavazzeni, Mascheroni D, Bordone G, Solca M (1991) Combined use of mask CPAP and minitracheostomy as an alternative to endotracheal intubation. Intensive Care Med 17:47–49

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Inhaled nitric oxide is often efficient in severe ARDS

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Sir: In a recent issue of Intensive Care Medicine, Mira et al. [1] presented a study concerning the lack of efficiency of inhaled nitric oxide in ARDS. They reported a series of six patients with severe ARDS who did not respond to NO, three of whom responded to a subsequent trial of NO. It was suggested that soluble guanylate cyclase of pulmonary vasculature smooth muscle could be unresponsive to NO.

We used inhaled NO in 30 patients with severe ARDS ($PaO_2/FiO_2 = 81 \pm 8$ on FiO_2 1 PEEP 11±1; LIS = 3.45) [2]. NO (5 ppm) was administered early (mechanical ventilation for 7±2 days). Twenty-eight patients were considered to be NO responders (+20% PaO_2/FiO_2). We did not find any correlation between the improvement in arterial oxygenation and the decrease in mean pulmonary arterial pressure [PAPm = 29±3 at TO and PAPm = 28±2 at T1 hour (NS)]. The 2 NO non-responders had acute hemorragic pulmonary edema with refractory septic shock (SAP under 70 mmHg with high doses of epinephrine and norepinephrine). Owing to hemodynamic instability, PEEP levels were low and did not allow for alveolar recruitment. In such a situation, it is not surprising that an inhaled agent might be ineffecient.

According to published studies, 30–50% of patients are considered to be responders to inhaled NO [3, 4]. In ARDS, pulmonary hypertension is secondary not only to hypoxic pulmonary vasoconstriction, but also to increased Va/Q abnormality, atelectasis, loss of vascular bed and small vessel obstruction. Given this heterogeneous vascular insult, it does not seem surprising that pulmonary artery pressure was not dramatically changed. Several additional factors may interfere with the efficacy of NO in patients with severe ARDS: reduction or loss of the hypoxic pulmonary vasoconstriction (pulmonary infection, lung trauma, lung hyperinflation) and replacement of actively constricted small pulmonary vessels by fibrotic and irreversibly narrowed pulmonary vessels [5]. The last explanation may be of value for the patients presented by Mira et al. Indeed, as a reference center for LFPPV-$ECCO_2R$, they may have recruited a higher proportion of end-stage ARDS than we did. Modifications of pulmonary vasculature in late ARDS associated with large areas of non-ventilated, non-recruitable parenchyma may be responsible for a decreased response to inhaled NO.

Finally, the wide variation in responders to NO inhalation could be related to the patient' pulmonary vascular levels of guanylate cyclase, and also to the degree of pulmonary parenchyma and vascular fibrosis.

References

1. Mira JP, Monchi M, Brunet F, Fierobe L, Dhainaut, JF, Dinh-Xuan AT (1994) Lack of efficiency of inhaled nitric oxide in ARDS. Intensive Care Med 7:532
2. Levy B, Bollaert PE, Bauer P, Nace L, Audibert G, Larcan A (1995) Therapeutic optimization including inhaled nitric oxide in adult respiratory distress syndrome in a polyvalent intensive care unit. J Trauma 38:370–374
3. Wysocki M, Delclaux C, Roupie E, Langeron O, Liu N, Herman B, Lemaire F, Brochard L (1994) Additive effects on gas exchange of inhaled nitric oxide and intravenous almitrine bismesylate in the adult respiratory distress syndrome. Intensive Care Med 20:254–259
4. Lowson SM, McArdle P, Rich GF (1994) Variable responses to inhaled nitric oxide in patients with ARDS (abstract). Anesthesiology 81:A124
5. Marshall BE, Hanson CW, Frasch F, Marshall C (1994) Role of hypoxic pulmonary vasoconstriction in pulmonary gas exchange and blood flow distribution. Intensive Care Med 20:379–389

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