

INHALED BRONCHODILATORS IN AIRFLOW OBSTRUCTION TOO MUCH OF A GOOD THING?

by John Marini MD



No one could reasonably argue that bronchodilators are not among our most useful interventions for outpatients with airflow obstruction. But as currently used, I am less certain about their efficacy in the ventilated patient with decompensated COPD or status asthma. In recent years it has become fashionable to deliver high doses of bronchodilators through the ventilator's connecting circuitry at frequent intervals - even continuously in the most worrisome cases. However, the dose actually delivered in this fashion and its effectiveness cannot be easily determined. Apart from logistical issues and the associated expense, this practice troubles me for other reasons. Bronchodilator overdosing is a potentially dangerous

practice, and except in a minority of cases, the primary pathological problem in these conditions is not bronchospasm but mucosal inflammation, edema and retained secretions.

Patients afflicted by asthma or COPD are saddled with these conditions for years. Consequently, many are quite knowledgeable about their disease, its treatment, and their idiosyncratic responses. Most utilize adrenergic bronchodilators at home and have already escalated their dosing prior to arriving to the hospital. Indeed, by their very presentation for emergency care they announce that bronchodilators have failed them. Exhausted and yet physiologically stressed, their levels of adrenergic stimulation are predictably high. Pulmonary catecholamine receptors, bombarded for days to weeks by adrenergic bronchodilators, are desensitized to their action. Delivering the bronchodilator to the distal airways where they might do the most good presents a major challenge. Narrowed by chronic structural disease and acutely clogged by mucosal edema and thick secretions, compromised airways allow little of the intended inhaled dose to reach its intended target. The open airways do accept the drug, however, where the twin circulations of the lung—pulmonary and bronchial—absorb them into the blood. Once in the general circulation, the catecholamine spillover has unintended actions. Beta-2 'selective' agents stimulate peripheral muscle to increase tremor and, with it, oxygen consumption and CO₂ production. More importantly, the associated cardiac stimulation often manifests as tachycardia or arrhythmia, and presents an ischemic stress as the cardiac triple product rises. In those with the potential for diastolic dysfunction, lung congestion and edema add to the ventilatory burden, impair gas exchange, and even accentuate bronchospasm. In pressing forward with greater intensity with a failed intervention we sometimes bring with more trouble. As someone once said, the definition of insanity is to do exactly the same thing repeatedly while expecting a different result.

Whereas using bronchodilators for patients with acutely decompensated obstructive disease is certainly rational, their overuse is decidedly not. What should be done differently? In my view, these patients who require intubation and ventilatory support launch themselves into a different management category. Having failed adrenergic bronchodilators, a changeup---not intensification---is the way to go. With regard to the adrenergic

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Last night, I went to bed at twenty of 11. That's about average for me. I always wonder who all the people are who stay up and watch David Letterman until midnight. I like Letterman, but you can't go to bed at 12:30 a.m. and still get enough sleep to go to work at a reasonable hour.

It's generally agreed that we need eight hours of sleep a night. This may be true, but I don't think we ought to be taking all eight hours at the same time. It seems to me it would make more sense to sleep four hours twice a day, or three hours and three hours and two hours, rather than eight hours all together. Once we fall asleep, we get over being tired in a hurry.

I don't know who came up with the eight-hour theory but it seems about right, although I seem to be able to make out with seven. If I get to bed by 11 p.m., I'm ready to get up at 6 a.m. Getting up early is one of the easiest ways I know to get a head start on the rest of the world. Most people don't get up until 7:30 or 8 a.m. To be truthful with you, though, I confess that I have a comfortable couch in my office that's six feet long and I'm only 5-foot-9. I've been known to drop onto it to drop off for a few minutes.

I'm a very good sleeper. I've given it some thought and I'm ready to say that one of the things I do best is nap. I don't lie in bed tossing and turning. Even when I'm worried about something, I can turn off my thoughts and go to sleep. Insomnia is an unknown condition to me.

I don't know what we should do about it, but we've made a mess of our 24-hour day and we ought to give some serious thought to reorganizing it. It won't be easy because it involves what time we eat breakfast, lunch, dinner, what we watch on television, when we work, when we play and when we sleep.

The problem with rearranging our sleeping time is obvious, of course. It's the intrusive nature of other things like cooking, television, doing the dishes and talking on the telephone. No one wants to put on and take off their pajamas twice a day.

We really ought to go to work twice each day, but the way our lives are arranged, it would not be practical. Sometimes, if we live in the country and our workplace is in the city, it may take an hour for us to get to work. Every business ought to have a room where employees can grab a nap. Napping has a bad reputation.

The French are expert at living. They enjoy life. They take a long break in the middle of the day, eat dinner and maybe have a nap before going back to work. The French may not accomplish as much as we do but they're happier. Most Americans would have a hard time getting back to work after drinking a bottle of wine and taking a nap in the middle of the day.

I'm smarter the first two hours at work in the morning than I am the rest of the day. Ideally, it would be better if I worked from 7 to 11 a.m., took four hours off and then worked again from 3 to 7 p.m.

It just seems wrong that our work day is determined by the dinner hour, or maybe by what's on television. It might take a while, but we would all eventually adjust to dividing our night's sleep into two parts.

Sleeping only after it gets dark must have started in the days before we had electricity. The world turned pitch black when the sun went down. We get daylight now with the flip of a switch and we ought to update our sleeping schedule.

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bronchodilators, these patients may need a brief drug holiday. To me, at least, substitution of ipratropium for albuterol makes good sense. Moreover, the emphasis must shift from bronchodilation to suppression of inflammation, clearance of secretions, resolution of airway edema, and reduction of the ventilatory burden. Moderate to high dose steroids are appropriate for both asthma and COPD (but in quite different doses—much lower for COPD). Excessive steroid doses are not only unnecessary, but cause these often elderly, weak and exhausted patients to become agitated, sleep deprived, and even delirious. Steroids unbalance glucose control and encourage consumption of muscle protein. Antibiotics—particularly those macrolides that have an anti-inflammatory action—are routinely given in my practice. Sedation is routine and is invariably necessary to reduce minute ventilation during the early phase of treatment. I do not attempt to lighten sedation until there are clear signs of improved airway patency—usually 2-4 days after intubation.

In some of these patients—particularly those with signs of retained secretions on the tidal flow and pressure tracings—bronchoscopic inspection of the airway not only reveals thick, immobile secretions but affords a chance to perform directed lavage. In such individuals, I boost humidification (by raising heated circuit temperature) and utilize mucolytics. I avoid using excessively rapid inspiratory flows and utilize square rather than decelerating waveforms to reduce the tendency for adverse ventilatory patterns to peripheralize secretions. I place emphasis on

reducing minute ventilation requirements to limit auto-PEEP and I use counterbalancing levels of added PEEP to keep the proximal airways patent, improve the distribution of ventilation, and improve the delivery of the inhaled bronchodilators that I do give. Episodic PEEP release is used during suctioning to aid coughing effectiveness. Repositioning into the position of optimum ventilatory efficacy makes good sense. Infrequently, I advise vibrotherapy and prone positioning to help secretions migrate from peripheral to central positions where they can be more easily extracted.

Trying to hurry the recovery process by forcing massive doses of adrenergic bronchodilators upon ventilated patients with decompensated airflow obstruction is a misguided stratagem. In many instances these actions produce just the opposite of the intended effect. Protocols based on 'average' patients may be a reasonable starting point in some settings but almost never enough and frequently are inappropriate for the situation at hand. These conditions are inherently complex. As with many difficult problems of intensive care, it is best to undertake an approach reasoned on the basis of a physiological analysis and employ "short loop" feedback cycles of treatment, careful observation of response, and thoughtful readjustment of management.

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