To the Editor:

I must make the assumption that Winter would only advocate carotid body resection for patients with severe asthma; one of his criteria refers to failure to adequately respond to "conventional therapy." There is no such thing as "conventional therapy" for severe asthma. Multiple mechanisms are involved; more often than not, therapy requires two different bronchodilators (theophylline and a β₂-sympathomimetic) neomycin sodium, inhaled or orally administered corticosteroids or both of these. Therapy with theophylline, in addition, must be tailored to the individual, and around-the-clock administration may be mandatory.

Asthmatic subjects who do not fall into this category of severity can have their asthma well controlled with bronchodilator therapy tailored to their needs, and adequacy of therapy can be confirmed by measurements of pulmonary function at frequent intervals. Tests of pulmonary function performed before and after surgery and thereafter (many months or a year later) have little validity in assessing a response to therapy in a dynamic disease such as asthma.

It is obvious that one of the major problems associated with the treatment of severe asthma is a lack of compliance by the patient. Whether this failure is best managed by resorting to carotid body resection or by efforts to promote understanding of the patient's disease by the patient is another matter.

I do not agree with the fact that some asthmatic subjects who have severe poorly controlled disease of long duration have poor ventilatory drives to hypoxia. These are the asthmatic subjects who fail to develop clinical discomfort in the face of increasing hypoxia due to increasing bronchospasm. These are also the subjects who apparently have "fulminating" attacks of asthma that are so often easily responsive to an inhaled or injected dose of a β₂-sympathomimetic drug or ephedrine. The attacks appear to fulminate because the patients are symptomatically unaware of the increasing bronchospasm and, axiomatic, increasing hypoxemia until the attack is so severe that the attacks appear to "fulminate." The risks of asymptomatic clinical unresponsiveness can be great.

The fact that control could be achieved with adequate around-the-clock therapy plus treatment with cromolyn to avoid the exercise component in our patient is by no means an indication that the carotid resection was successful. It has every chance of being an indication that the initial resection was unnecessary and reflected poor prior medical management.

In regard to the lack of supportive data in our case report (ie, no blood gas levels and no neurologic studies), I think that Winter would agree that if the child was cyanotic, his arterial oxygen pressure would be well below 50 mm Hg, and he has to be desaturated. Our worry was due to the fact that the child had no symptomatic warning prior to these events, a fact very unusual with even the most severe asthma that we see but common with those who have a depressed ventilatory drive.

Of course we cannot know what our patient's ventilatory drive was before the carotid resection. Of course we realize that there may have been an inborn defect in control of oxygen-dependent ventilatory drive. We realize equally well that the addition of a carotid body resection has no possibility of improving the situation if indeed the patient had a developmental deficit, and hence resection could only make things worse.

I find it difficult to accept the suggestion by Winter that other symptoms, such as headache, malaise, "pressure" in the head, and palpitations, are adequate substitutions for discomfort in the chest and dyspnea as clinical warnings.

Finally, I would like to comment on two of Winter's4,8 abstracts that he cited. In both studies, most subjects were patients with chronic obstructive pulmonary disease (COPD), with few asthmatic subjects. In the first study,70 percent of the subjects were said to be "better." No objective data were given, nor was it stated how much "better." More importantly, 23 percent of the subjects were worse. To have COPD to start with is bad enough, but to stand a one-in-five chance of being worse with an irreversible procedure hardly commends the procedure. The second study4 was somewhat confusing. Thirty-two subjects had impairment of the airways (large airways) which was stated to be normal (?), slight, or moderate. Seventy-two subjects had severe impairment. The success or failure was apparently based on pulmonary scans (xenon-xenon or xenon-aggregated albumin was not stated). How this relates to ventilatory drive I cannot understand, as the basic defect clarified by these procedures is at the levels of ventilation and perfusion. The other references by others are to the physiology of ventilatory drive.

Our view remains the same. The benefits to be derived from carotid resection are difficult for us to understand, other than subjects were "better." The probable risks are very clear. An irreversible procedure resulting in a possible 23 percent of patients being worse is unacceptable in asthma, and much more so in COPD in my view. In this day of better understanding and "tailored" therapeutic approaches, I consider the procedure of resection to be unnecessary and the risks unacceptable.

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REFERENCES

