Extracorporeal Life Support for a Patient With Severe Asthma

To the Editor:

We read with great interest the article by Michael et al.,1 which reported a patient with life-threatening respiratory acidosis due to status asthmaticus who was successfully treated with extracorporeal life support (ECLS). Since the authors claimed that their case was the first patient with this disease treated with ECLS, we would like to call their attention to our case report that was published in 1991.2

Our patient was a 20-year-old man with a history of bronchial asthma. He had a severe asthma attack while driving a car and was transported to a hospital. Despite maximal efforts made to relieve his attack with conventional drugs and mechanical ventilation, his condition deteriorated rapidly. Arterial blood gas analysis revealed severe respiratory acidosis and hypoxemia (pH=6.82, PaCO2=223 mm Hg, PaO2=44 mm Hg, FiO2=1.0). When we decided to put the patient on extracorporeal lung assist (ECLA) 40 min after admission, he suddenly fell into cardiac arrest. Veno-arterial ECLA was started immediately. His conditions improved dramatically.

Although blood access was different (veno-venous vs veno-arterial), our report clearly showed the effectiveness of ECLA or ECLS for severe asthmatic patients. We chose veno-arterial bypass for our reported case because he had cardiac arrest. However, we use veno-arterial bypass for patients with respiratory failure without circulatory problems if oxygenation is not satisfactory with veno-venous bypass.

Already we had reported a case of successful treatment by applying ECLS to a severe asthma attack in 1991. Michael et al said, "We believe that represents the first application of this technology to this disease in an adult patient." But it was his error. We must assert that it should be corrected.

Shoji Ito, M.D., and
Hirotada Katsuya, M.D., F.C.C.P.,
Department of Anesthesiology and Resuscitology,
Nagoya City University Medical School,
Nagoya City, Japan

REFERENCES

β-Agonists and Bronchial Asthma

To The Editor:

I read with interest the correspondence from Dr. Sears1 and Dr. Ziment2 in the August, 1993, issue of Chest. I would like to strongly support Dr. Sears’ position.

There are two important observations. First, it is a paradox that asthma morbidity and mortality are rising at a time when sales of increasingly potent asthma therapies are rising whereas a reciprocal relationship should be seen. Second, the most used asthma therapy, namely inhaled β2-agonists, have been reported to have negative effects in asthma including worsened asthma control,3 worsened airway responsiveness,4-7 accelerated decline in lung function,8 tachyphylaxis to important airway effects,9,10 a dose-response relationship between β-agonist consumption and mortality,11 as well as a tendency to be overused for symptom relief while underappreciating severity and undertreatment with anti-inflammatories.12 When the asthma paradox is viewed in the light of the negative effects of inhaled β2-agonists in asthma, the conclusion would seem to be inescapable. It is possible if not probable that some or all of rising asthma morbidity, mortality, and possibly even prevalence, is the result of the increasing and regular (or frequent as needed) use of inhaled β2-agonists.

Dr. Ziment’s letter contains two comments that merit response. First, Dr. Ziment states, “...millions of asthmatics use routine inhaled β-agonists and...do very well.” This is undoubtedly true but cannot be used as an argument to justify the safety of routine β-agonist use. I am reminded of the similar misguided logic that is frequently used by cigarette-smoking patients (correctly) claiming that some or many cigarette smokers do so quite happily without any adverse events!

The second comment concerns the dangerous implications in paragraph 3 that resting bronchoconstriction is common in chronic asthma and that the alternatives are to ignore it or to treat it with regular inhaled β2-agonists. Nothing could be further from the truth. Resting bronchoconstriction signifies uncontrolled airway inflammation and, therefore, identifies asthmatics who are poorly controlled. I would advocate neither of the treatments preferred by Ziment. Rather, I would use resting bronchoconstriction as an indication to add or increase prophylactic therapy such as inhaled corticosteroids.

Despite these concerns, it is critical to remember the important occasional life-saving value of inhaled β2-agonists in the management of acute severe asthma1 while awaiting the delayed relief from corticosteroids.

Donald W. Cockerell, M.D., F.R.C.P.
Department of Medicine,
Division of Respiratory Medicine,
Royal University Hospital,
Saskatoon, Saskatchewan,
Canada