Communications for this section will be published as space and priorities permit. The comments should not exceed 500 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Intermittent Positive-Pressure Breathing in Asthma

To the Editor:

Loren et al\(^{1,2}\) have concluded that "IPPB did not offer any advantage over simple nebulization in patients with severe, reversible airway obstruction." Based on the described method of administration of therapy with intermittent positive-pressure breathing (IPPB), it is not appropriate to make this conclusion, as there is a major flaw in the method of Loren et al\(^1\).

Several years ago, when "puff parlor" IPPB was practiced, it was common to administer therapy with IPPB in an assembly-line fashion, as described by Loren et al\(^1\). Despite differences in the caliber of their airways, their pulmonary volumes, and their degree of bronchospasm, all patients received therapy from a machine at an arbitrarily selected value of 10 cm H\(_2\)O for peak pressure. I do not believe that Loren et al\(^1\) intended to imply that these 23 asthmatic children (ages, 6 to 16 years), have identical lungs, although the way in which the therapy with IPPB was administered would indicate so.

Therapy with IPPB must be individualized to the requirements of each patient. In order to do this, it is essential that the volume delivered by IPPB be adjusted to exceed that of the patient’s spontaneous tidal volume (TV). This is not achieved by arbitrarily selecting 10 cm H\(_2\)O as the required peak pressure for all patients. In fact, in pediatric asthmatic patients a peak pressure of 20 to 40 cm H\(_2\)O commonly is required to deliver a breath that is deeper than the patient is capable of moving spontaneously. In my experience, utilizing therapy with IPPB at a peak pressure of 10 cm H\(_2\)O results in volumes very near the patient’s spontaneous TV. This would account for the nearly identical degree of bronchodilatation achieved by Loren et al\(^1\) when comparing IPPB at 10 cm H\(_2\)O with a simple nebulizer and the Freon-propelled nebulizing devices.

The potential problems of pneumothorax and pneumomediastinum are real in patients with severe asthma; however, there is no good evidence indicating that the incidence of barotrauma in asthmatic patients is influenced by the use of therapy with IPPB. Barotrauma is a complication seen in asthmatic patients who are not treated with IPPB. Bierman\(^2\) reported 16 cases of barotrauma with severe asthma. Of these 16 patients, only four had received therapy with IPPB. It appears that the barotrauma is just as likely a result of the disease as of therapy with IPPB.

Treatment with IPPB is yet to be properly evaluated in a study that administers the IPPB in an individualized manner by personnel trained in its proper and safe administration. Until it is realized that "plugging in" a patient and letting the machine "do its thing" is unsatisfactory, there will be conflicting reports about the effectiveness of therapy with IPPB in the treatment of any respiratory disease. I believe that a proper conclusion for the study of Loren et al\(^1\) would have been as follows: therapy with IPPB at 10 cm H\(_2\)O in the pediatric patient with acute asthma is like no IPPB at all for the purposes of administering an aerosol bronchodilator drug.

Melvin A. Welch, Jr., R.R.T., Program Director
School of Respiratory Therapy
UCLA Hospital-Santa Monica College, Los Angeles

REFERENCES

To the Editor:

It becomes a matter of personal experience how one should best individualize and standardize treatments with nebulization in the asthmatic patient with severe obstruction of the airways. Welch suggests that one should adjust the inspiratory distending pressures between 20 and 40 cm H\(_2\)O to maintain tidal volume (TV) above the spontaneous TV of the patient. There have been no studies that have demonstrated any advantage of administering therapy with intermittent positive-pressure breathing (IPPB) set at these pressures. In an uncontrolled study, Yanda\(^1\) had compared various distending pressures of IPPB (from 10 to 25 cm H\(_2\)O) to the generation of TV and found no correlation with pressures of IPPB and measurements of TV.

Although we have not actually measured TV in asthmatic patients during severe bronchospasm, in our opinion, a pressure of 10 cm H\(_2\)O at a rate of flow of 80 L/min was adequate in exceeding the spontaneous TV of most asthmatic patients who were receiving therapy with IPPB. There were a number of patients with asthma more severe than that of the patients we studied; these patients with more severe asthma were immediately given parenteral therapy with bronchodilator drugs. In the opinion of the nursing staff and staff physicians, these patients were unable to tolerate any form of treatment with nebulization.

Welch is correct in saying that there are no studies indicating that barotrauma in asthmatic patients is influenced by the use of IPPB. There is a suggestion in the work by Bone and associates\(^1\) that elevated TV and
pressure may contribute to barotrauma. In a brief communication, these investigators reported that 20 out of 50 patients with the adult respiratory distress syndrome who received therapy with mechanical ventilation at high TV and high positive end-expiratory pressure (PEEP) developed pneumothorax and pneumomediastinum. Patients who received therapy with mechanical ventilation at lower TV and lower PEEP did not develop these complications.

We agree with Welch that patients in status asthmaticus have a higher incidence of these complications, compared to patients not in status asthmaticus. The point we are trying to raise is that patients who are already at a high risk of developing complications may be placed at a potentially higher risk by receiving therapy with IPPB.

Michael L. Loren, M.D., and Hyman Chai, M.D.
National Asthma Center, Denver

REFERENCES
1 Yanda RL: Quality control of inhalation therapy: The results of therapy, with and without control, and methods of developing such control, in a community hospital. Chest 69:61-66, 1974

Correction
To the Editor:

We would like to call your attention to two cases reported in our abstract entitled "Specific Elastase Inhibitory Activity of Serum in Chronic Bronchitis and Emphysema" (Chest 72:260, 1977). Original observations placed the values for serum elastase inhibitory activity in these two patients at 1.9 and 3.1 standard deviations below the mean of their cohort. Subsequently, we have determined that the values in all patients analyzed fall within 2 standard deviations of the mean of the group tested.

Roy H. Eriksen, M.D., and Robert A. Norum, M.D.
Division of Pulmonary Diseases and Division of Human Genetics, Department of Medicine
New York Hospital-Cornell Medical Center, New York

Formula for Total Pulmonary Arteriolar Resistance
To the Editor:

In their article in Chest, Bracchi et al. stated that the formula for total pulmonary arteriolar resistance was:

\[ R = \frac{1,332 \times (\text{mean PAP} - \text{PCWP})}{\text{CO}} \]

and they expressed their units in dynes-sec-cm^{-5} (PAP, pulmonary arterial pressure; PCWP, pulmonary capil-
lary wedge pressure; and CO, cardiac output). This formula and the units used are incorrect, as the formula only expresses results in dynes/min/cm². In order to obtain correct units and to express the cardiac output in ml/sec/cm², the cardiac output is multiplied by 6,100. Therefore, the correct formula, to the nearest decimal point, is:

\[ R = \frac{(\text{mean PAP} - \text{PCWP}) \times 80}{\text{CO}} \] dynes/sec/cm^{-5}

where PVR represents pulmonary vascular resistance.

Desmond A. Leen, M.D., Resident in Anesthesia
St. Boniface General Hospital, Winnipeg, Manitoba

REFERENCES

Cardiac Tamponade and Nonhemorrhagic Pericardial Fluid in Dressler’s Syndrome
To the Editor:

The report by Tew and associates with respect to cardiac tamponade complicating Dressler’s syndrome is of great interest; this complication has been seen by me on at least one occasion and was briefly referred to in a recent review of this syndrome. Although Tew and his co-workers believed that their patient represented the first reported case with cardiac tamponade and nonhemorrhagic pericardial fluid as a result of Dressler’s syndrome, this combination was, in fact, reported by Lawrence and Wright in 1972.

Alan Bernstein, M.B.
Consultant Physician, Hope Hospital
University of Manchester School of Medicine
Salford, England

REFERENCES

To the Editor:

The report by Tew and colleagues entitled “Cardiac Tamponade with Nonhemorrhagic Pericardial Fluid Complicating Dressler’s Syndrome” (Chest 72:93-95,