force in the horizontal plane, and/or 3) the presence of posterior bites may suggest that VCG could, in the future, be the non-invasive diagnostic test of choice for acute true posterior myocardial infarction.

I wonder whether the authors intend to apply their result in patients with acute posterior infarction, and what future plans they have to validate their result in patients with differentially located infaracts, especially during the acute period.

Wangden Carson, M.D.,
Cardiac Department,
John Radcliffe Hospital,
University of Oxford,
Headington, Oxford,
United Kingdom

References

To the Editor:

Unfortunately, none of the patients studied in our series was acute or had a recent myocardial infarction. The observation of Doctor Carson is extremely interesting and illustrates one of the many problems encountered in the analysis and study of posterior wall myocardial infarction. Unfortunately, most of our patients were referred to us and did not have their acute event at our institution. We are trying to gather enough information to address this important issue.

Isaac Eisenstein, M.D.,
Lakewood, California

New Modes of Ventilatory Assistance

To the Editor:

The recent article by Prakash and Meij (Chest 1985; 88:403-06) describes a new ventilatory technique that may have characteristics which can benefit some patients who require ventilatory assistance. However, I am concerned that terminology suggested by Prakash and Meij may be confusing and may lead to inappropriate clinical application of the technique. Dr. Moshin pioneered ventilator classification based on cycling mechanisms¹ in order to better understand mechanical ventilator function. I believe that such an analysis of pressure support ventilation is indicated in order to appreciate the mechanical characteristics and potential physiologic effects of this technique.

Pressure support ventilation may be initiated by the patient's inspiratory effort. Thus, the inspiratory phase is patient-triggered and pressure-cycled. As long as the patient's inspiratory effort continues, a pre-set airway pressure will be maintained by a variable flow of gas from the ventilator. When the patient's inspiratory effort wanes, flow will decrease. When the effort ceases, flow will terminate and airway pressure will return to the preset baseline pressure. Thus, the inspiratory phase is terminated, or cycled, by flow (or lack thereof). Thus, pressure support ventilation is really "assisted mechanical ventilation" in its purest form. As long as the patient attempts to inhale, he will receive assisted ventilation, which is pressure limited but flow, rather than volume or pressure, cycled. Some may feel that this is a semantic exercise of little physiologic consequence. However, I think it is not.

Analysis of figures 2 and 3 in the article by Prakash and Meij demonstrates similar tidal volumes during conventional ventilation and pressure support ventilation. Initial flows are greater during pressure support because of the higher pre-set airway pressure; as inspiration proceeds, flow decreases during pressure support, in contrast to the constant flow that is terminated at a pre-set volume during conventional ventilation. Thus, in both examples, patients are receiving mechanical ventilatory support and are not breathing "spontaneously" in the usual sense.

Pressure support ventilation was most likely designed to counter the excessive work of breathing associated with many currently available IMV ventilators that supply gas for spontaneous respiration on demand.¹¹ Unfortunately, unless the pressure support level is precisely set to match the exact pressure drop created by circuit resistance and inertia of demand-valve flow, positive pressure ventilation will result.¹ Even low levels of pressure support may cause significant mechanical ventilation and may possibly mislead the clinician who is evaluating the patient for potential extubation. For example, a patient receiving 3 cm H₂O pressure support with a lung-thorax compliance level of 50 ml/cm H₂O and is breathing 20 times/min, may receive an additional 150 ml of alveolar ventilation with each "spontaneous" breath, or 3 L/min. The patient may, or may not, be able to provide adequate assisted alveolar ventilation during total spontaneous respiration following extubation. This point is illustrated in table 1 of the article; here, the mean PaCO₂ increased from 40±2 mm Hg during pressure support to 47±5 mm Hg more than two hours following extubation.

It may be that variable tidal volume, constant pressure, patient triggered mechanical ventilation (term inspired pressure support during spontaneous respiration by Prakash and Meij) will open a "new horizon in clinical respiratory care," as they suggest. However, it is clear that the physiologic effects of inspiratory pressure support are likely to mimic those observed during mechanical ventilation, and patients receiving pressure support should not be considered to be breathing spontaneously. Care should be taken to insure patient safety, and less confusing nomenclature may be advisable.

John B. Downs, M.D., F.C.C.P., Professor & Vice Chairman, Department of Anesthesiology, Ohio State University, Columbus

Reprint requests: Dr. Downs, Department of Anesthesiology, Ohio State University Hospital, Columbus, 43210-1223

References
2 Downs JB. Inappropriate applications of IMV. Chest 1980; 78:897
4 Katz JA, Kraemer RW, Gjerde GE. Inspiratory work and airway pressure with continuous positive airway pressure delivery system. Chest 1985; 88:519-26

To the Editor:

In this letter, Dr. Downs takes up some interesting aspects to new modes of ventilatory assistance. One aspect is that Dr. Moshin contributed greatly by supplying the scientific community with a nomenclature that was widely accepted. However, Dr. Moshin could not anticipate all new modes of ventilatory assistance that are now becoming available.

In our paper we used the phrase inspiratory pressure support during spontaneous ventilation, I-PSV. The patient triggers each inspiration with an effort that lowers the airway pressure below the preset expiratory pressure. Inspiration is then supported by a preset positive pressure in the ventilator circuit. When inspiratory flow rate has fallen to 25 percent of the peak flow rate, expiration starts. We have not been able to describe this mode in the terminology of Dr.
Mushin. We also foresee other modes of ventilation which are still more difficult to describe in classical terms. There is a need for new modes of ventilation which let the patient, to a large extent, influence tidal volumes, flow patterns and duration of inspiration and expiration—hence also breathing frequency. The ventilator can support ventilation in various ways without imposing unnecessary restriction upon the patient, who can contribute to ventilatory control in more or less natural ways. We strongly believe that this approach does open up a new horizon—but do not state that we have reached it.

As concerns pressure support of 3 cm H2O, Dr. Downs has calculated a theoretic contribution of this pressure to tidal volume. His calculation would have been correct if a static condition had been reached towards the end of inspiration. According to the principles of IPSSV, and as our illustrations show, this is not the case. Three cm H2O roughly corresponds to the pressure needed to overcome the resistance of the tube.

The increase of PaCO2 that was observed after extubation is rather typical after various weaning procedures. It was similar for patients weaned by IPSSV and controlled ventilation in our study.

We welcome the discussion that must go on until we have a much greater experience with various new modes of ventilation and find new nomenclature.

Omar Prakash, M.D.
Erasmus University
Rotterdam, The Netherlands

Reprint requests: Dr. Prakash, Thoracentrum, Erasmus Universiteit Rotterdam, 3015 GE Rotterdam, The Netherlands

Decrease In FEV, and FVC with Sodium Morrowate Esophageal Sclerotherapy

To the Editor:

Endoscopic sclerotherapy of esophageal varices is useful in portal hypertension and recurrent variceal hemorrhage. Complications occur in 10 to 15 percent of cases and include esophageal perforation and stricture, ulceration, bleeding, chest pain, pleural effusions and fever.

Sodium morrowate is a salt of fatty acids which are known to produce pulmonary toxicity when injected into animals. ARDS associated with sodium morrowate sclerotherapy has been reported. We present a case of a patient's reversible decrease in pulmonary function after sclerotherapy with sodium morrowate.

CASE REPORT

A 54-year-old black woman with a remote history of portal vein thrombosis, portal hypertension and recurrent esophageal variceal bleeds presented with hematemesis and an endoscopy-confirmed variceal hemorrhage. Chest x-ray examination showed a large paratracheal lymph node which contained non-caseating granulomas consistent with sarcoidosis. FEV1 was 1.87 L (67 percent predicted) and FVC was 2.19 L (67 percent). Helium lung volumes showed a TLC of 3.49 L (75 percent), VC 2.29 L (81 percent), FRC 1.65 L (63 percent), RV 1.20 L (81 percent). DLCO and blood gas levels were normal. Lungs were normal upon physical examination. Sodium morrowate sclerotherapy was begun and was complicated only by fever and malaise two to days after each procedure. On one occasion, pulmonary function tests performed three days after injection of 25 ml five percent sodium morrowate solution showed an FEV1 of 1.38 L and FVC of 1.67 L. This decrease in function persisted at two weeks (FEV1, 1.65 L, FVC 2.04 L), but was back to baseline at 12 weeks (FEV1, 1.93 L, FVC 2.16 L). Later, with administration of 18 ml of five percent sodium morrowate; solution FEV1 dropped from 1.93 to 1.59 L and FVC from 2.16 to 1.82 L. At 11 weeks, function was back to baseline (FEV1, 1.87 L, FVC 2.20 L). This patient has received no treatment for her sarcoidosis, which is clinically stable. Her only medication is iron sulfate.

DISCUSSION

Our case suggests that sodium morrowate sclerotherapy may cause a transient decrease in FEV1 and FVC. Because our patient has sarcoidosis, it is difficult to generalize this case to the normal population. A study examining the effects of sodium morrowate on pulmonary function is needed.

Stuart Katz, M.D.; Jeffrey Schneider, M.D., C.M.; and E. James Britt, M.D., F.C.C.P.
The Johns Hopkins Medical Institutions, Francis Scott Key Medical Center, Baltimore

REFERENCES


Pulmonary Microvascular Cytology
A New Diagnostic Application of the Pulmonary Artery Catheter

To the Editor:

We read with interest the report of Drs. Masson and Ruggeri (Chest 1985; 88:914) detailing the diagnosis of pulmonary disorders using samples of blood obtained from a wedged pulmonary artery catheter.

Recently, we were able to confirm a suspected case of amniotic fluid embolism by this technique.

A 26-year-old gravida 4 para 1-0-2-1 woman presented to the obstetric service after spontaneous rupture of membranes and onset of uterine contractions. Prenatal course had been unremarkable. The patient had smoked one pack of cigarettes per day during pregnancy. Acute fetal distress with late deceleration developed, necessitating emergency cesarean section with apgar scores of 8 and 9.

In the recovery room, the patient developed acute bronchospasm. Shortly thereafter, blood pressure began to rise, tendon reflexes were increased and urinary output fell to 5 ml/hr. Intravenous dexamethasone, furosemide and magnesium sulfate therapies were instituted. The patient was transferred to the intensive care unit.

The patient's respirations were labored with a rate of 28/min and a heart rate of 120/min with no S2. Blood pressure was 140/64 mm Hg, and temperature, 99.4°F. Her extremities were grossly edematous; inspiratory and expiratory wheezes were present. Pertinent laboratory data included a pH of 7.34, Pco2 of 27 and Po2 of 64 while breathing 4 L O2 by nasal cannula. Her creatinine level was 2.6, BUN 15, sodium 132, potassium 5.1, chlorides 103, and CO2 39. White blood cell count was 22,200 with left shift and the hemoglobin level was 7.3 with a hematocrit of 22. A pulmonary artery (Swan-Ganz) catheter was placed, maintaining normal cardiac output, capillary wedge pressure of 15 and pulmonary artery pressure of 40/25.

Additional laboratory data indicated a subclinical consumptive

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