pulmonary recovery." Actually, that quote is not to be found in our paper. We did write that we wished to test the hypothesis that 

recruitive PEEP would hasten pulmonary recovery. The hypothesis was not supported by our data.

Secondly, they misquote our paper as follows: "Patients with hypoxemia due to atelectasis or unilateral lung disease, must be treated with very high PEEP" What we wrote which may have provoked their reaction was "... patients were treated whose hypoxemia was a result of hydrostatic pulmonary edema, postoperative atelectasis, and pneumonia. However, those conditions often are not distinguishable from early ARDS, may occur together with ARDS, and may also benefit from PEEP."

Thirdly, they attribute to us the quote, "Highest PaO2 is the best indicator of titrating PEEP". There is no statement in our paper remotely similar, making a direct response difficult. However, we can say that there appears to be no scientific evidence which indicates to what endpoint PEEP should be titrated. Indeed, it was our intent to compare two methods in a clinical trial in order to begin to identify endpoints for PEEP titration.

Fourthly, our critics chide us for attributing increased mortality to our use of recruitive PEEP. They incorrectly point out that our data indicate statistically equivalent death rates in each of our groups. They missed the point which was that they had equivalent death rates only after PEEP treatment had ended. During treatment of 22 patients subjected to recruitive PEEP died, compared to only one of 28 patients subjected to supportive PEEP. That difference is indeed significant (χ² = 3.9, p = .05).

Finally, Drs. Dedhia, Schiebel and Teha agree with the editorial views expressed by Dr. Civetta that "this poorly conducted study" will lead clinicians improperly, to stop using PEEP.

We object to the notion that conclusions are invalid simply because they may be extrapolated improperly. We appreciate the need to interpret our results carefully, but reaction to our study seems to have gone far beyond—to have generated frank animus.

For example, in our section titled "PEEP Application Mode" we reported that two of our 22 recruitive PEEP patients and three of our 28 supportive PEEP patients got PEEP by face mask. Yet Dr. Civetta Ignored that information and implied in his editorial that our recruitive PEEP group did worse because they were denied face mask-type CPAP. Furthermore, that section was added to the paper at his request as reviewer of the paper! Perhaps Drs. Dedhia, Schiebel and Teha became so inflamed by the editorial that they proceeded extensively to misread our paper.

In any case, we believe the root cause of the hostility which so obscured our critics' discernment is a simple misperception. They appear to have the opinion that we are challenging a tenet upon which all of us base our clinical practice: the endpoint of PEEP titration must simultaneously permit safe inspired oxygen tensions, nearly complete arterial saturation, and ensure that this oxygenated blood is delivered to the tissues. Those goals are so obvious they are trivial. Indeed, they are pursued vigorously in all our patients at all times. We certainly were not so benighted as to try to compare PEEP titrated to levels which did not maintain adequate arterial oxygen transport to that which did.

In actual clinical practice, PEEP tolerance depends on intravascular volume. It is usually possible to give intravenous fluids sufficient for patients to maintain oxygen transport with a wide variety of PEEP levels. Therefore, it is possible to test the more interesting and clinically relevant hypothesis: Does PEEP help heal the lung or merely support PaO2? Accordingly, we titrated PEEP to levels which produced blood gas changes associated with lung recruitment (recruitive PEEP) because recruitment seems to be the leading hypothesis for the mechanism of PEEP's putative healing property. Fluids and occasionally pressors were given as needed. Those 22 patients so treated had a significantly greater mortality than the supportive PEEP patients (p = .05). As stated in our paper, four patients randomized to the recruitive PEEP group actually received no PEEP. (Only if those four are included is the p = .08, as stated in the editorial cited by our critics. We felt it unethical to proceed under the circumstances.)

In conclusion, we hope objective interpretation of our paper convinces the reader that PEEP may be beneficial but should not be titrated above the point at which PaO2 is satisfactory at a nontoxic FIO2 level.

Gilbert C. Carroll, M.D.;
Kenneth J. Tuman, M.D., F.C.C.P.;
Berton Braserman, Ph. D.;
William G. Logas, D.O.;
Normal Wool, M.D., F.C.C.P.;
Marshall Goldin, M.D., F.C.C.P., and
Anthony D. Ivanekovitch, M.D., F.C.C.P.;
Departments of Anesthesiology, Surgery,
and Cardiovascular and Thoracic Surgery,
Rush-Presbyterian-St. Luke's Medical Center,
Chicago

Perfusion Scan Defects

To the Editor:

I read with interest the report by Berkowitz and colleagues describing a unilateral ventilation-perfusion defect due to a bronchogenic cyst.1 Because of our divisional interest in chronic thrombotic occlusion of the pulmonary vasculature, I have had occasion to collect reports documenting uncommon causes of pulmonary artery (PA) obstruction. I would like to point out that Watts et al have published a similar case.2 Their manuscript details a 21-year-old woman who, like Berkowitz's patient, presented with dyspnea and pleuritic chest pain. Although having previously been labeled as suffering from congenital right PA stenosis based upon cardiac examination and catheterization at age 6 years, she was subsequently found to have a subcarinal bronchogenic cyst which impeded flow through the right PA. Preoperatively, there was no flow to the right lung apparent on either scan or angiogram. Postoperative perfusion scan was normal (results of ventilation scans were not reported). Thus, Berkowitz's case is the second illustrating this uncommon cause of perfusion scan defect, and at least the fourth of a bronchogenic cyst causing pulmonary artery stenosis.3,4

Defining the etiology of pulmonary arterial obstruction can be surprisingly difficult. In selected cases, fiberoptic angioscopy can add information not currently available by noninvasive means.5

Benjamin Kanter, M.D.,
Division of Pulmonary and Critical Care Medicine,
University of California School of Medicine,
San Diego

REFERENCES


CHEST / 95 / 1 / JANUARY, 1989 253