EDITORIALS

Positive End-Expiratory Pressure
Is No Panacea

In 1967, Ashbaugh et al. described the use of positive end-expiratory pressure (PEEP) in the management of noncardiogenic pulmonary edema in adults. Subsequently, the usefulness of this therapeutic approach has been widely acclaimed, and numerous authors have attested to the beneficial effects of therapy with PEEP in the management of acute respiratory failure. The pulmonary disorders which have responded most dramatically to therapy with PEEP have been characterized by decreased compliance of the lung, reduced functional residual capacity (FRC), microatelectasis, and pulmonary edema without left ventricular failure. It has been recognized that the major beneficial effects appear to emanate from an increase in FRC, with resultant reduction in microatelectasis and intrapulmonary shunt.

As with many successful types of therapy, the assumption is often made that if some is good, then more is better. This not only relates to the absolute levels of PEEP that may be applied to the airways but also to the specific indications for its use. Today in many medical centers, any patient with significant hypoxemia is likely to receive therapy with PEEP, regardless of the cause of the pulmonary disease, if his "numbers" satisfy some predetermined criteria for treatment. These criteria usually relate to the fractional concentration of oxygen in the inspired gas, the alveolar-arterial oxygen gradient, or the intrapulmonary shunt. Included among these patients are those with cardiogenic pulmonary edema and low cardiac output, as well as other patients with primary obstructive pulmonary disease where some acute insult has resulted in an increased intrapulmonary shunt. In many of these patients, the therapeutic gain may be negligible, but the clinician is often reassured by an increase in arterial oxygen tension. The total effect of PEEP on the transport of oxygen, pulmonary mechanics, and cardiac output is frequently unclear and sometimes ignored.

Knowledge in this area is further confused by the failure of many authors to provide information concerning differences in pulmonary pathophysiology when reporting on the success or failure of therapy with PEEP. The lumping of patients into broad categories, such as the adult respiratory distress syndrome or acute respiratory failure, prevents the identification of subgroups that may respond quite differently to therapy with PEEP.

Therapy with PEEP is commonly employed in the postoperative management of patients who have experienced thoracic, cardiovascular, or abdominal surgery. In most instances, this is being done as "routine" practice, without scientific data to support its use. Some physicians regularly prescribe PEEP for all patients who have undergone a particular surgical procedure, and the prescription may call for a predetermined level of PEEP for a period of time which may be fixed or subsequently judged appropriate at the rising of the sun or the hour of morning rounds.

One of the areas where atelectasis is most predictably encountered is in the patient undergoing open-heart surgery. Here, atelectasis of the left lower lobe is not an unexpected postoperative finding. If prophylactic therapy with PEEP is helpful in preventing postoperative atelectasis, then this is an area where positive effects should be evident. In this issue of Chest (see page 397), Good et al. describe a controlled study of 24 patients undergoing open-heart surgery who were randomly assigned to receive PEEP or no PEEP following surgery. Good et al. found that therapy with PEEP had no beneficial effect in the prevention of atelectasis during the postoperative period.

Fowler et al. recently reported that therapy with PEEP can be dramatically effective in correcting refractory lobar atelectasis, and the failure of PEEP to prevent postoperative atelectasis in patients who have undergone cardiac surgery may seem contradictory. There are several possible explanations for this apparent inconsistency. The therapeutic effectiveness of any treatment used in the management of an acute disorder does not justify its prophylactic use. We have learned this lesson many times before; penicillin, which is a superb drug in the treatment of certain pneumonias, is a miserable failure as a
prophylactic agent to prevent pneumonia. Additionally, the type of atelectasis observed in many patients after surgery is plate-like or subsegmental and is sometimes difficult to identify on the chest x-ray film. The observations of Fowler et al. were specific for lobar atelectasis without obstruction of the large bronchi. It is of interest that the only patient to develop lobar atelectasis in the report by Good et al. was in the group with no PEEP, but this one case does not justify the use of prophylactic therapy with PEEP in these or any other patients.

A question that must be answered in the future is whether or not therapy with PEEP is potentially harmful in a patient after surgery, even when administered at relatively low pressures in an attempt to avoid alveolar overdistention and barotrauma. Does the use of PEEP unnecessarily increase the cost of medical care or introduce hidden complications by virtue of more extensive invasive monitoring, prolonged intubation, or reduced local perfusion? Are there additional direct and indirect pulmonary or extrapulmonary effects which may be unrecognized in these patients? The answers to these questions remain obscure, but clearly there is little scientific justification for the widespread use of prophylactic therapy with PEEP as part of routine postoperative care.

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REFERENCES

Is There a Selective Increase in Pulmonary Capillary Permeability following Cutaneous Burns?

More than a generation ago, the exudative and transudative reaction at the site of the trauma due to a burn was described in animals. More elegant proof of altered permeability of capillaries to protein and fluid at the site of a burn was demonstrated by injecting radioactive bromide and radioactive colloids into the bloodstream and measuring their appearance in the lymph. The first physiologic clue that trauma due to burns might cause an increase in the permeability of capillaries at sites distant from the burn was provided by Arturson. By infusing dextran of different molecular sizes, he demonstrated alterations in the cervical flow of lymph following a burn on the paw. The greatest change in the permeability of capillaries in unburned areas affected permeability to substances with molecular weights of between 10,000 and 40,000. Arturson observed no significant increases in the concentration of albumin (molecular weight, 69,000) or other plasma proteins with higher molecular weights in the cervical lymph.

The observations are central to our understanding of the concept of resuscitation via administration of fluids in the burned patient. The common clinical dictum that resuscitation requires the infusion of volumes of fluid larger than that lost through the cutaneous burn alone implies that additional fluid is lost into unburned tissues. Indeed, the generalized edema of a severe cutaneous burn has been attributed to an impaired microvascular barrier to protein and an increased conductance of water across the capillary. Despite the recent increased clinical awareness of pulmonary edema due to altered permeability, there is little evidence that following a severe burn of the skin, proteins are confined to or leave the vascular compartment in man in unburned areas. In practical terms the question of whether crystalloid or colloid is the optimal form of resuscitation following burns hinges largely on how protein is handled by the barrier of the capillary. The lung is an ideal organ to assay these effects because of the flimsy air-tissue interface to oppose edema; however, characterizing the effects of a peripheral cutaneous burn at distant sites, especially the lung, on the permeability of water and proteins has been difficult. Sheep with a chronic lymphatic fistula, which were first described by Staub et al., provide an innovative preparation to study the pulmonary microvasculature and the filtration of fluid and protein in the lung. In this issue (see page 448), Demling and associates have applied this technique to measure the alterations in the flow of fluid across the lung following cutaneous burns.

The work of Demling et al demonstrates two salient features. First, the increased flow of fluid across the lung at low intravascular pressures early during a burn may imply an increase in transendothelial conductance to filtration of fluid at sites distant from the burn. Although normal pulmonary intravascular pressures were present, acute reduction in plasma colloid osmotic pressure occurred as a result of resuscitation. Depression of colloid osmotic pressure alone with unchanged capillary hydrostatic pressure has been shown by Zarins et al. to increase the flow of pulmonary lymph in baboons. Unfortunately, in the present study of Demling et al,