An End to “ARDS”

Few medical acronyms have become so popular and yet are so misleading as that of the “adult respiratory distress syndrome” or “ARDS.” Certainly, no one would argue that this rather common pulmonary disorder could not occur in children, and in its milder forms, it seems likely that there may be relatively little evidence for pulmonary insufficiency. The term was originally adopted to indicate a respiratory disorder characterized by the acute onset of tachypnea, hypoxemia, and decreased compliance. On chest x-ray films, pulmonary infiltrates were visibly suggestive of local edema, and histologic studies of pulmonary tissues obtained from these patients frequently showed hyaline membranes covering the pulmonary epithelium and both interstitial and intra-alveolar hemorrhage and edema. This early study recognized the basic physiology, course, and incidence of this illness and the therapeutic advantages of ventilation with positive end-expiratory pressure. It can be said without exaggeration that the report permanently changed the practice of pulmonary medicine and helped give it a central role in intensive care medicine.

Reference to an “adult” form of respiratory distress was used to distinguish it from the neonatal illness, which was also characterized by noncardiogenic pulmonary edema and hyaline membrane formation. The basic problem in the neonate is attributable to the immaturity of the lung at the time of birth and is probably related to a deficiency of surfactant production. Although there may also be a deficiency in surfactant in the adult disease, and although there may eventually be some role for administering surfactant in these patients, the cause of the illness is presumably related to an injury involving the lungs which results in formation of edema.

However, there is a somewhat more subtle problem with the term which has led to some confusion in both clinical and investigational circles. Although it had long been recognized that specific agents such as toxic fumes could cause pulmonary injury and edema in the absence of congestive heart failure, it was not recognized just how common an event this could be until the introduction of the Swan-Ganz catheter into clinical practice some 15 years ago. Prior to that time, it had been common practice to attribute the development of edema following injuries or surgical catastrophes to some obscure failure of the heart. This became untenable when low pulmonary arterial occlusion pressures were measured in many of these patients. The causes of edema were presumably related to some form of pulmonary injury which was sustained in patients with severe injuries related either to wartime or civilian activities, but the pathogenesis of the injury was and remains obscure. This mysterious illness obviously and embarrassingly must have been prevalent for many years but remained unrecognized because left atrial pressures were not readily estimated and arterial oxygen tensions were difficult to obtain. Furthermore, the frequency of the problem undoubtedly increased when emergency services improved and patients could be delivered to the hospital with much greater dispatch. It was natural to assume that this very serious problem was a specific disorder of the lungs with a well-defined set of causes and a natural history of its own.

Difficulties quickly arose with this approach. The list of potential factors that can precipitate ARDS grew dramatically with experience. From the diagnostic point of view, it became particularly difficult to say when a patient had a severe bacterial viral pneumonia or a picture of ARDS, and complaints were expressed that the term referred to a miscellaneous collection of disorders which might have little to do with one another. Furthermore, it has gradually become evident that the edema found in the lungs of these patients might simply represent the most obvious and indeed the most serious manifestation of a generalized capillary injury.

This sequence of events must be familiar to even the most casual medical historian. Following the introduction of the clinical thermometer in the latter part of the nineteenth century, considerable attention was given to a variety of “fevers” which were assumed to represent specific illnesses. Advances in bacteriology soon made it obvious that fever was simply a manifestation of the disease, which would require specific therapy; at best, treatment of the fever itself could provide only temporary relief. Now that it has become technically practical to rule out congestive heart failure as the cause for pulmonary edema, care must be taken that a similar error is not made. For the sake of clarity, it would seem preferable to make a specific statement regarding the pathogenesis of the edema, rather than referring to it as “ARDS.” For example, if no cardiac cause for the edema can be found, then it is “noncardiogenic pulmonary edema.” If the clinician is convinced that the edema is related to some injury to the endothelial or epithelial membranes (or both) which leads to the leakage of protein and fluid into the interstitium or alveolar spaces, then it would be appropriate to refer to an “exudative” or an “inflammatory” edema, rather than a “transudative” or “non-

10 Dahms TE, Bolin JF, Slavin RG. Passive smoking: effects on bronchial asthma. Chest 1981; 80:530-34
inflammatory” edema. Much can be learned from the manner in which pleural effusions are usefully divided in a comparable fashion.

There would be several potential advantages to the adoption of this nomenclature. The question of whether viral or bacterial pneumonia had become ARDS could be reduced to the question of whether an exudative edema had appeared in regions of the lung which were remote from the infected tissues. Furthermore, it would emphasize the possibility that prolonged elevations of intravascular pressures might eventually lead to injury of tissue and the formation of an exudative edema and the fact that pulmonary injury can occur independently in patients with congestive heart failure. Even from an investigational perspective, this change in semantics might be expected to be fruitful, since it would emphasize that studies of “ARDS” represent no more and no less than a study of the phenomena of injury and inflammation in the lung, which may differ in some respects from inflammation elsewhere, but more than likely involve many of the same mechanisms.


REFERENCES


Risk-Benefit Analysis in Chest Medicine

A New Feature

In this issue of Chest (see page 276) we begin a new regular feature which will attempt a critical analysis of many of the current modalities of diagnosis and treatment used in chest medicine. The basis of the series is the belief that a major task faced by medicine is to become more safe and effective and responsive than is currently the case. This statement is not intended as a simple recognition that all human activities, including patient care, can be improved. It is a recognition of the fact that while medical care often results in major improvements in patient welfare, it also uses practices which are tangential to patient welfare and not uncommonly sanctions practices which are harmful. Both the good and the bad seem to be increasing as technology flourishes without major changes in the way that new science is incorporated into medical practice.

If there are major problems with the present state of medicine, what might be done to improve safety, effectiveness and responsiveness? One key is a wider and more knowledgeable use of risk-benefit analysis by physicians. It is generally recognized that all medical encounters have risks as well as benefits. Physicians are routinely called upon to make decisions for which they should perform risk-benefit analysis, but too often fail to recognize the extent to which, in even simple decisions, both risk and benefit are involved. Nor do they recognize that their decision cannot be made in an absolute sense, but depends upon some sort of probability estimate. In fact, they commonly assume that their own decision reflects the most optimal risk-benefit analysis.

For many or most diagnostic and therapeutic procedures, the data needed for a rational decision are simply not available. Even though a method does exist for rationally determining the probabilities of risk versus benefit (an appropriately designed clinical trial to be described in subsequent columns), little of current practice has been tested in this crucible. The recognition that a given form of practice has not been validated should by itself improve safety. Even when available, information derived from an acceptable clinical trial is often not widely known, and, sad to say, even when a procedure is widely known to be more risk than benefit, its use may not be abandoned. For example, several adequate clinical trials have shown that electronic fetal monitoring (EFM) produces no improvement in fetal salvage, but is associated with a high rate of (unnecessary) cesarean sections and increased maternal and fetal risks. The use of EFM continues to be almost mandatory. Firm data exist that perhaps 50 percent of coronary bypass surgery is performed on patients who would do better on medical management. The national mortality for bypass is about 2 percent, but bypasses continue to be performed in inappropriate groups of patients.

It should be recognized that risk-benefit analysis can be fruitfully approached by thinking about risk/benefit ratios. The less the benefit, the closer the risk/benefit ratio approaches infinity. For trivial patient benefits, even the most trivial patient risks are unacceptable.

In this series we will be reviewing the status of a number of management approaches in chest disease.

For some approaches, clinical trials establish the safety and efficacy of the intervention. The careful use of captopril for the treatment of intractable congestive heart failure is an example.

For some approaches, adequate clinical trials have established that risks exceed benefits. EFM and coro-