Pulmonary Arterial Pressure Increases during Cardiopulmonary Bypass, a Potential Cause of Pulmonary Edema

ROBERT J. BYRICK, M.D.,* DONALD C. FINLAYSON, M.D., F.R.C.P. (C),† WILLIAM H. NOBLE, M.D., F.R.C.P. (C)†

Elevated pulmonary arterial pressure during cardiopulmonary bypass has been shown to lead to pulmonary edema in dogs in the immediate post-bypass period.1 In their animal model, Littlefield et al.1 demonstrated the significance of elevated pulmonary-artery hydrostatic pressure as a cause of pulmonary edema following cardiopulmonary bypass. However, in these dogs, the aorta and the pulmonary artery were clamped, at times high bronchial arterial flow was created, and the colloid osmotic pressure was maintained at 25 torr. These conditions are very different from our handling of patients undergoing cardiopulmonary bypass. During bypass using crystalloid priming solutions, the colloid osmotic pressure is reduced.2 Hence, if Starling's relationship regarding the transfer of water and electrolytes across a semipermeable membrane applies during extracorporeal circulation, pulmonary edema must develop at lower levels of pulmonary-artery hydrostatic pressure than would normally occur.

Nahas3 and others have commented on the reduction in the incidence of “post-perfusion lung syndrome” in patients since the introduction of left ventricular venting to reduce hydrostatic pressure in the pulmonary capillary bed during bypass. However, the routine use of left ventricular venting has not been universally accepted,4 and some investigators even argue against its use.

The difficulty in quantitating extravascular lung water has made it difficult to evaluate the etiologic factors involved in the development of pulmonary edema.5 Using a new double indicator dilution technique (extravascular thermal volume of the lung, ETVL),6,7 to study this problem, we found a patient in whom a large increase in lung water developed within an hour of the completion of a coronary-artery operation. The only factor distinguishing this patient from a larger group in which this was not the case,8 was his persistent elevation in pulmonary arterial pressure during bypass.

Since the development of flow-directed pulmonary arterial catheters the pulmonary-artery-occluded pressure (PAP) has been widely used in cardiac surgical patients to evaluate left ventricular performance. The following report demonstrates the value of this catheter in monitoring a patient with persistently high pulmonary arterial pressure during bypass. This patient had gross pulmonary edema within an hour of the completion of the surgical procedure. Data from this case are compared with those from a more typical case, representative of a larger group, in which pulmonary arterial pressure was not elevated during bypass.

REPORT OF TWO CASES

Patient 1. A 50-year-old man who had coronary-artery disease was scheduled for aortocoronary bypass. The patient was a heavy smoker but had no evidence of chronic pulmonary disease or pulmonary edema. At cardiac catheterization minimal aortic insufficiency was found. ETVL,6,7 and shunt fraction, Qs/Qo,10 were determined following induction of anesthesia but prior to operation.

The pulmonary arterial pressure and PAP were monitored preoperatively, and at the lung-water measurement one hour after operation. The pulmonary arterial pressure was also monitored during cardiopulmonary bypass. Colloid osmotic pressure was calculated11 from plasma protein measurements taken at the same times.

Patient 2. A 62-year-old man who had symptomatic coronary-artery disease and chronic pulmonary dysfunction underwent bypass and was studied in the same fashion.

A Travenol bubble oxygenator (VF-1) primed with two liters of lactated Ringer's solution was used for both patients. Flow rates were 60 ml/kg/min. Ventricular fibrillation was induced while the patient was on cardiopulmonary bypass.

RESULTS

During the period of extracorporeal circulation the colloid osmotic pressures of both patients decreased, as expected8,9 (fig. 1). The pulmonary arterial pressure of Patient 1 was persistently elevated (= 20 torr) throughout the procedure (fig. 2). In Patient 2, pulmonary arterial
of the air-entrainment type, inserted via the pulmonary vein. It has proven generally satisfactory in our hands; however, in Patient 1 left ventricular distention was noticed by the surgeon throughout the procedure. This was followed by a striking increase in both ETV_{l} (61.3 per cent) and Q_{s}/Q_{t} (143.5 per cent) from the preoperative measurement to the immediate postoperative determination (table 1).

Patient 2 had no increase in either Q_{s}/Q_{t} or ETV_{l}, (table 1). This conforms to the pattern of lung water accumulation in our previous series.8

**DISCUSSION**

In a previous study of lung water changes in patients following bypass,8 we found no significant increase in Q_{s}/Q_{t} or ETV_{l} from the control determination to the immediate postoperative measurement. In Patient 1 the high pulmonary arterial pressures during extracorporeal circulation undoubtedly followed inadequate venting of the left atrium and ventricle. The pulmonary arterial pressure of Patient 1 during cardiopulmonary bypass was double his colloid osmotic pressure at the same time. This led to increases in both ETV_{l} and Q_{s}/Q_{t}. The increases in ETV_{l} and Q_{s}/Q_{t} probably reflect pulmonary edema at the stage of alveolar filling,12 as pulmonary edema was evident on the radiograph. In Patient 2 (fig. 2) momentary increases in pulmonary arterial pressure to above colloid osmotic pressure associated with manipulation of the heart were not sustained long enough to create pulmonary edema.

Immediate post-bypass pulmonary edema of the extent seen in Patient 1 must be a rare event. In a similar group of patients previously reported, no pulmonary edema was evident at this stage.8

The "post-perfusion lung syndrome" has been attributed to a variety of factors. Some of these are surfactant suppression,13 pulmonary vasculitis,14 microaggregate formation,15 protein denaturation,16 and circulating humoral substances.4,17 Many of these may be contributory, but since bronchial artery flow persists during cardiopulmonary bypass there is a variable but continuous infusion of blood via bronchopulmonary anastomoses into the pulmonary circulation. Sustained pulmonary hypertension during cardiopulmonary bypass in the

| Table 1. Changes in Extravascular Thermal Volume of the Lung (ETV_{l}) and Q_{s}/Q_{t} |
|---------------------------------|-----------------|-----------------|
|                                 | Patient 1       | Patient 2       |
| ETV_{l} Preoperative           | 5.22 ± 0.17 ml/kg | 6.43 ± 0.09 ml/kg |
| Postoperative                  | 8.42 ± 1.1 ml/kg  | 5.3 ± 0.2 ml/kg   |
| Q_{s}/Q_{t} Preoperative      | 13.55 per cent   | 31.96 per cent   |
| Postoperative                  | 32.99 per cent   | 20.34 per cent   |

pressure fluctuated about a mean of 10 torr (fig. 2) during bypass close to the colloid osmotic pressure (10 torr). With surgical manipulation the pulmonary arterial pressure briefly rose above 20 torr (fig. 2). The cause of the persistently increased pulmonary arterial pressure in Patient 1 appeared to be a combination of aortic insufficiency and poor vent function. The vent used in both patients was
animal model (>60 tomm) was accompanied by microscopic evidence of peribronchial, perivascular and alveolar edema and hemorrhage after simultaneous occlusion of the pulmonary artery and aorta. In the same study, left atrial decompression effectively lowered the pulmonary arterial pressure and prevented pulmonary edema and hemorrhage. Patient 2 (fig. 2) had a sudden drop in pulmonary arterial pressure following insertion of the left ventricular vent. Venting may protect the lungs as well as the heart during bypass. Brief elevations of hydrostatic pressure during bypass of the type seen in Patient 2 do not, in our studies, result in significant elevations in lung water in the immediate postoperative period.

The duration of extracorporeal circulation was longer for Patient 2, in whom pulmonary edema did not develop one hour after operation (fig. 2). The edema in Patient 1 was not the result of prolonged cardiopulmonary bypass. The development of pulmonary edema 24 to 48 hours postoperatively results from factors other than intraoperative elevation of pulmonary arterial pressures, such as alterations in the balance of Starling forces or alveolo-capillary permeability.

The reliability and usefulness of flow-directed balloon-tipped catheters in monitoring patients before and after cardiac surgery has been amply demonstrated. This report indicates that adequate left ventricular venting is important in preventing the development of pulmonary edema immediately postoperatively. When a Swan-Ganz catheter is in place during a procedure the pressure should be monitored during cardiac bypass and considered in relation to the colloid osmotic pressure.

 Decompression of the left ventricle and atrium reduces the hydrostatic pressure in the pulmonary vascular bed during bypass. Monitoring the pulmonary arterial pressure during bypass will detect elevations in pulmonary arterial pressure that may result in pulmonary dysfunction in the immediate postoperative period.

The authors thank Dr. Claire Baker for allowing them to study his patients and Dr. John Hart for reviewing the manuscript. Expert technical assistance was provided by Mr. J. C. Kay, Mrs. N. Bell, Mr. Gary Carmichael, and Mr. Gary Caskanette.

REFERENCES