Pulmonary Complications in the Postoperative Period with Particular Reference to the Overload Syndrome

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Pulmonary complications in the postoperative period can precipitate major metabolic abnormalities particularly in the critically-ill or debilitated patient. The resultant impairment in oxygen exchange or the increased work of breathing imposed on these patients has profound adverse effects on the balance of available oxygen and the utilization of oxygen in the body. Although atelectasis and pulmonary infections are common postoperative complications, they have not increased in frequency or severity over the years.¹ In recent years, however, the clinical syndrome of progressive hypoxemia with pulmonary edema and decreased compliance, the so-called “Wet Lung”, has plagued the physician because of its increasing lethality, when once developed.² This syndrome has been referred to by a number of names and the etiologic as well as pathogenic factors are quite complex.³–⁷ Nevertheless, the noteworthy feature that is common to all is the presence of pulmonary edema. Moreover, in many of the cases reported, the patients were over transfused, primarily with crystalloid solutions, and in these cases the problem might have been preventable. Because pulmonary edema is a constant feature in these patients, there has been a general misconception that congestive heart failure was the major feature of this syndrome. As a result, circulatory failure rather than pulmonary failure was the guideline for therapy. The purpose of this presentation is to re-evaluate the “Wet Lung” syndrome as it is affected by a volume overload both in the experimental animal and human, and to propose broad therapeutic guidelines for the treatment of this perplexing problem based on clinical and experimental studies which focus on the hypoxemia and H₂O excess in the lungs.

The typical radiographic appearance of pulmonary edema in the overload syndrome is illustrated in Figure 1. Note the diffuse bilateral infiltrates without any evidence of cardiomegaly. The infiltrates with time progress into a diffuse ground-glass appearance if the pulmonary edema is not corrected. The roentgenogram is indistinguishable from other etiologic factors causing the “Wet Lung” syndrome, such as fat embolization, post perfusion syndrome, pulmonary contusion, aspiration pneumonitis, oxygen toxicity and the Shock Lung. Thus the

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distress during the period of shock. Although three patients had underlying pulmonary or cardiac problems, the initial x-ray prior to the onset of the pulmonary edema showed normal lung parenchyma. Two of these patients had fractured ribs, one, of which had a pneumothorax treated by a closed tube thoracostomy. In neither was there radiologic evidence of pulmonary parenchymal abnormality. The third patient had undergone mitral valve prosthetic surgery but was not in congestive heart failure prior to the operation. The remaining three patients did not have chest trauma. The onset of symptoms occurred after intravenous fluid therapy had begun. In four patients this developed within eight hours. At the time of the respiratory distress, all six patients developed the typical pulmonary edema pattern on x-ray and were not in shock. The fluid intake and output of these patients indicated that they had been infused with large volumes of crystalloid solutions. The average amount of fluid administered had been 770 ml per hour. Note that the arterial \( \text{PO}_2 \)'s were markedly decreased despite the fact that all of the patients were on mechanical ventilatory assist with high concentrations of oxygen. The patient with the \( \text{PaO}_2 \) of 75 mm of mercury had already been on 100 percent oxygen concentration at the time of the measurement. Hypoventilation was not a problem, and only one patient demonstrated \( \text{PaCO}_2 \) retention. The total serum protein level was decreased in all patients, thus lowering the colloid oncotic pressure.

Table II represents ventilatory studies.
performed while on mechanical ventilatory assistance as these patients could not be taken off the ventilators. These patients were on high minute and tidal volumes which were artificially induced to compensate for the increased dead space. THE VD/VT was quite elevated despite the high tidal volumes. Alveolar ventilation was also increased. Simultaneous cardiopulmonary function studies were performed in four patients, and

VENTILATION - PERFUSION RELATIONSHIP
4 PTS OVERLOAD SYNDROME

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value 1</th>
<th>Value 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>VA - L/min</td>
<td>7.1</td>
<td>(4.6 - 12.0)</td>
</tr>
<tr>
<td>Q - L/min</td>
<td>7.1</td>
<td>(6.2 - 8.1)</td>
</tr>
<tr>
<td>VA/Q - %</td>
<td>94</td>
<td>(66 - 154)</td>
</tr>
<tr>
<td>Qs/Qt - %</td>
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<td>(20 - 44)</td>
</tr>
<tr>
<td>PaO₂ (100% O₂ - mmHg)</td>
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<td>(93 - 460)</td>
</tr>
<tr>
<td>A-a DO₂ - mmHg</td>
<td>420</td>
<td>(239 - 584)</td>
</tr>
</tbody>
</table>

Table 3

Table III is a summary of these studies. The cardiac outputs were all increased. There was increased venous admixture, alveolar to arterial oxygen tension gradient, and decreased arterial PO₂ while on a 100% oxygen. None of the four patients demonstrated an elevated CVP. In fact, only one of the six patients revealed a transient evidence of myocardial failure with an elevated CVP. This was the patient with the mitral valve prosthesis who required transient myocardial support with a vasoactive agent. In the other five patients, cardiomegaly was not present. The blood pressures were normal. The EKG was normal or revealed only non-specific S-T and T changes of ischemia. Circulatory failure was not apparent by the conventional standards of measurement in these patients.

In order to gain further insight of the circulatory components of the overload syndrome, some experimental data are presented from the laboratory. Figure 2 reveals sequential changes in intracardiac pressures, recorded during continuous volume loading of a blood-saline mixture in eight anesthetized intact dogs. Simultaneous increase of right and left intracardiac pressures were observed during volume loading with the left intracardiac pressures greater than the right. When the pressures on the right side were multiplied by two and when the zero reference of the right atrial pressure was corrected for the posterior position of the left atrium, the graphic plot on the right approximated those on the left side.

Figure 2 — Sequential intracardiac pressures during continuous infusion of volume demonstrates progressive increase of all pressures. The pressures on the left are always greater than those on the right.

Figure 3 — The intracardiac pressures shown in Figure 1 are replotted by the pressures on the right by a factor of two and correcting the zero reference by -2 to account for the posterior position of the left atrium. The right intracardiac pressure now approximate the left intracardiac pressures.

(Figure 3). On statistical analysis, there was a high degree of correlation in the response of all intracardiac pressures to volume loading. An analysis was made
based on 64 paired measurements in the eight dogs. The correlation between changes in the left atrial and right atrial pressure measurements (R = .93) and the correlation between the left ventricular enddiastolic and the left pressure measurements (R = .95) was not significantly

CORRELATION COEFFICIENT

8 DOGS - 64 PAIRED MEASUREMENTS

$\overline{LA} \text{ vs } \overline{LVED} \ r = .95 \ N.S.$

$\overline{LA} \ vs \ \overline{RA} \ r = .93$

CONSERVATIVE ESTIMATE OF 95%

LIMITS (.73, .97)

PH SKU 2/68

**Table 4**

This provided evidence that the predictability of changes in left ventricular enddiastolic pressure on the basis of mean left atrial pressure was essentially the same as the predictability of the left atrial pressure on the basis of the right atrial pressure. Thus the CVP is a reliable guide to changes on the left side of the heart during volume loading.

The hemodynamic measurements in three dogs are tabulated in Table V.

**Table 5**

Cardiac output, stroke volume and stroke work increased in response to 60 minutes of volume loading and the mean circulation times decreased. These data indicate that under the conditions of this study, ventricular performance increased during volume loading. The ventricular function curve in which left ventricular stroke work was plotted as a function of left ventricular enddiastolic pressure for the three animals which were volume loaded over a period of sixty minutes is shown in Figure 4. This did not display a descending component of the Frank-Starling curve. Thus, although the intracardiac pressures increased during volume loading, the myocardial performance improved consistent with Starling's Law. Myocardial failure was not demonstrated in these studies.

The metabolic data in these dogs revealed a significant drop in PaO₂ during this study from an average of 62 to 37 mmHg. The total serum protein decreased from 6.0 to 3.8 gm percent in the one dog that this measurement was taken, thus lowering the colloid oncotic pressure in the blood. Severe pulmonary edema was demonstrated at the time of autopsy in these animals.

The practical considerations derived from these experimental studies stress the importance of regarding the central venous pressure (CVP) as a reflection of the dynamic relationship between the blood volume and the competence of the heart to accept the venous return. Clearly
the central venous pressure reflects ventricular function in relationship to the venous return i.e. the volume presented to the heart rather than to the total blood volume. The importance of a dynamic assessment of the central venous pressure is also evident in the reports by Landis\(^9\) and Johnson\(^11\) who demonstrated increases in the central venous pressure during volume loading and a decrease in the pressure when the infusion was discontinued. Although the CVP reflected the functional competence of the ventricle to eject the volume load that is presented to it, whether it reflected progressive myocardial failure during the volume overload is not established. When very large volume loads were placed on the heart during our experiments and in the patients that were presented, myocardial performance was not impaired. How then did the pulmonary edema occur in these patients or in the dogs?

Pulmonary edema probably occurred because of the concomitant reversal in the hydrostatic and colloid oncotic gradient in the pulmonary capillaries. The importance of the relationship between plasma proteins and hydrostatic capillary pressure in the production of pulmonary edema was demonstrated by Guyton in 1959.\(^{12}\) In his experiments in dogs, he demonstrated that in the presence of normal colloid oncotic pressure, pulmonary edema did not occur until the left atrial pressure was raised above 25 mmHg. When the oncotic pressure was reduced by approximately 50 percent, pulmonary edema occurred when the left atrial pressure exceeded 12 mmHg. Thus, pulmonary edema can occur with normal left atrial and right atrial pressures if there is significant lowering of the colloid oncotic pressures by hemodilution. The dynamic relationship of the CVP and the colloid oncotic pressures must be taken into account in the production of pulmonary edema.

When patients are treated with large amounts of crystalloid solutions, the colloid oncotic pressure is decreased with concomitant increase in the capillary hydrostatic pressure which is also reflected by a rise in CVP. Normally the hydrostatic pressure in the capillaries is 10 mm of mercury and the oncotic pressure is between 25 and 30 mmHg. When the hydrostatic pressure exceeds the colloid oncotic pressure, pulmonary edema occurs. These changes develop in a dynamic fashion at the time of the volume loading. When the volume load is discontinued, the CVP may be down to normal levels.

The lymphatic drainage of the lungs acts as a safety valve attempting to decompress the pulmonary vascular bed whenever the hydrostatic pulmonary capillary pressure rises. However, this mechanism of preventing pulmonary edema is ineffective when large volumes are infused continuously.

Another important factor producing the pulmonary edema picture is the capillary permeability of the vessels. This may be one of the etiologic factors of other forms of pulmonary edema, but its role here is not clearly defined.

The manifestations of the overload syndrome are summarized in Table VI.

| OVERLOAD SYNDROME |
|-------------------|------------------|
| **CLINICAL**      | RESPIRATORY DISTRESS WITH PULMONARY EDEMA AND HYPOXEMIA |
| **PULMONARY**     | INCREASED DEAD SPACEoha/0 ABNORMALITY SHUNTING |
|                   | INCREASED A - a \(O_2\) GRADIENT |
| **CIRCULATORY**   | CVP ELICIATED WITH NORMAL ONCOTIC PRESSURE |
|                   | CVP MAY BE NORMAL IF ONCOTIC PRESSURE LOW OR IF STATIC MEASUREMENT |
|                   | CARDIAC OUTPUT NORMAL OR HIGH UNLESS UNDERLYING CARDIAC DISEASE PRESENT |

Table 6

Clinically, occurrence of progressive respiratory distress with pulmonary edema and severe hypoxemia are noted after the infusion of large volume of fluid. Pulmonary function abnormalities indi-
cate decreased PaO₂ and increased dead space, ventilation to perfusion abnormalities, venous admixture and alveolar to arterial oxygen tension gradient. The circulatory components to the overload syndrome can be noted by an elevated CVP in the presence of a normal colloid oncotic pressure. The CVP may be normal if the oncotic pressure is low or if the CVP is taken as a static measurement. The cardiac output is normal or high unless underlying cardiac disease is present.

Treatment of the overload syndrome is focused on correction of the severe hypoxemia as well as the mobilization of the increased fluid in the lungs. Table VII summarizes the broad concepts of treatment for the overload syndrome. The first is prevention. Cautious administration of crystalloid solutions with the simultaneous monitoring of the CVP and the colloid pressure in a dynamic fashion would help in preventing many of the overload complications. When the complications of pulmonary edema are already present, hypoxemia dominates the clinical picture. Treatment is then focused on the correction of the pulmonary derangements, preferably with mechanical ventilatory support, in order to increase the oxygen exchange. Fluid restrictions to less than 1,000 ml per day as well as the use of potent diuretics should be instituted to remove excess fluid. Once an adequate diuresis is maintained, the administration of salt poor albumin can be used to mobilize the fluid from the extravascular compartment. Secondary forms of therapy are the use of vasoactive drugs. These should be used only if the patient develops clinical evidence of myocardial failure. The use of cortical steroids have been recommended by some investigators but its beneficial effects have not been clearly defined. If an adequate urine volume or diuresis cannot be accomplished because of poor renal function, the use of peritoneal dialysis or hemodialysis should be considered to mobilize the fluid.

In the six patients presented here all were treated with prolonged mechanical ventilation, with tracheostomies in five patients and prolonged nasal tracheal intubation in one patient. The volume controlled respirators gave the best results in all of the six patients. Five of the patients required end expiratory resistance or pressure. The total fluid intake was restricted to less than 1,000 ml per day. Potent diuretics were used to promote a diuresis. The mean average urinary output in all of these patients was 3,500 ml in the first 24 hours after therapy. Digitalis was not routinely administered. Only one patient, the patient undergoing the mitral valve prosthetic replacement required transient positive inotropic support. Isoproterenol was infused for 15 hours shortly after the open-heart surgery, and he also received maintenance Digitalis for his underlying cardiac problem. With these combined therapeutic measures, the pulmonary edema gradually resolved in all of the patients with significant clinical improvement. Five patients survived and eventually were discharged from the hospital in satisfactory condition. One patient died nine days after he was admitted to the Shock Trauma Center. This patient developed bronchopneumonia and expired suddenly following a precipitous hypotension and an elevated CVP to a level of 24 mm of mercury. Pulmonary embolism was suspected but a post mortem examination was not performed on this patient.
IN SUMMARY:
The postoperative complications related to the overload syndrome have been discussed primarily as they influence the production of the “Wet Lung” syndrome. Broad therapeutic guides have been proposed to emphasize the importance of focusing on the pulmonary function abnormalities as well as the mobilization of fluid from the lungs rather than on the support of myocardial function as the proper approach to the management of this syndrome.

REFERENCES