Treatment of Pulmonary Edema by Means of Temporal Occlusion of Inferior Vena Cava with Balloon-Technique

K. Taguchi
Department of Cardiovascular Surgery, Hiroshima Citizens Hospital, Hiroshima, Japan

Intending to invent a newer and more effective technique to treat pulmonary edema being developed in the pre- and postoperative courses, and during operation of cardiac surgery, the author has pursued the broad experimental and clinical studies and has found the following new and simple technique being suitable for the above purpose.

The experimental study on this new technique, in which the inferior vena cava was occluded temporarily at the height of mid of 4th lumbar vertebra by means of balloon-catheter under heparinization for 2 to 4 hours repeatedly, disclosed that experimentally created pulmonary edema could be treated successfully. This technique has been called as "Balloon-Technique" by author. In addition to this, both of ligation of inferior vena cava and of assisted circulation were also investigated. However, the superiority of balloon technique compared with other two techniques were demonstrated. Ligation of inferior vena cava did not show the prolonged effect, only for 20 days, and also showed the inconvenient hemodynamics in case of total cardiopulmonary bypass by means of lung-heart machine. Although the assisted circulation showed significant improvement of experimental pulmonary edema as much as in balloon technique, this required many staffs and time loss. Thus, the author abandoned these two techniques in both of experimental dogs and of clinical patients.

Forty dogs with experimentally created mitral insufficiency have been induced into acute pulmonary edema by means of rapid intravenous administration of saline solution and of adrenalin. Although 75% of dogs treated without this technique could not survived, 90% survive with aid of this subsidy technique. Rapid disappearance of bronchial secretion and rapid decrease of elevated pulmonary artery pressure have been the most impressive observations. Also, the prompt and marked decrease of cardiopulmonary ratio has been demonstrated in x-ray examination of the experimental dogs.

In the clinical application, 25 patients with both of acute and chronic pulmonary edema have been treated by temporal occlusion of inferior vena cava by means of balloon-catheter. Among them, 13 patients developed the pulmonary edema in their preoperative courses and 7 developed in their postoperative courses. Remaining 5 patients developed acute pulmonary edema during cardiac surgery. The significant and remarkable improvement of severe advanced pulmonary edema was observed in all of these 25 patients. As the balloon technique was very easy to repeat in the same patient, twice to several times repeating were carried out in these patients. The clinical manifestations seen in these patients were decreased pulmonary edema, improved liver function, increased urine flow, decreased venous pressure, disappearance of dyspnea and improved cardiac hypertrophy. There occurred only 3 deaths in this surgical series of patients with severe advanced cardiac failure combined with acute and chronic pulmonary edema.
Thus, the author believed that this balloon-technique would be an effective tool to extend the surgical indication of far advanced cardiac patients especially combined with acute and chronic pulmonary edema.

**Concluding Remarks**

D. M. Aviado

Department of Pharmacology, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania, U.S.A.

An examination of the titles submitted for this meeting reveals that research in pulmonary edema has been devoted mostly to its pathogenesis rather than to therapy. The existing information on the pathogenesis of pulmonary edema is summarized in four figures devoted to left ventricular failure, intracranial lesions, epinephrine-induced edema, and alloxan-induced edema. In each situation, the combined participation of increased capillary permeability and elevated capillary hydrostatic pressure is indicated. The vagus and sympathetic innervation to the lungs participate in the formation of edema, but the intermediate events triggered by the autonomic nerves have not been identified. The enclosed figures will be used to indicate the new information reported in the conference and the suggested approach to therapy of pulmonary edema. (Figures are reproduced from: D. M. Aviado: The Lung Circulation. Vol. II. Pathologic Physiology and Therapy of Diseases, Chapter 18: Acute Pulmonary Edema, pages 865 to 932, Pergamon Press, Oxford, 1965).

Pathogenesis of acute pulmonary edema arising from left ventricular failure

<table>
<thead>
<tr>
<th>PREDISPOSING:</th>
<th>PRECIPITATING:</th>
</tr>
</thead>
<tbody>
<tr>
<td>ARTERIOSCLEROTIC HEART</td>
<td>TACHYCARDIA</td>
</tr>
<tr>
<td>MITRAL STENOSIS</td>
<td>↑ VENOUS RETURN</td>
</tr>
<tr>
<td>AORTIC INSUFFICIENCY</td>
<td>↑ BLOOD VOLUME</td>
</tr>
<tr>
<td>RELEASE OF COMPENSATORY VASOCONSTRICTION</td>
<td>ACUTE RESP. INFECTION</td>
</tr>
</tbody>
</table>

\[
\text{SYM. VASOCONSTRICTION} \rightarrow \text{RECEPTORS} \\
\rightarrow \text{CAROTID SINUS} \\
\rightarrow \text{AORTIC ARCH} \\
\rightarrow \text{LT. VENTRICULAR} \\
\rightarrow \text{EDEMA} \\
\rightarrow \text{SYM. TACHYCARDIA}
\]

\[
\downarrow \text{CARDIAC OUTPUT} \\
\uparrow \text{LT. ATRIAL B. P.} \\
\uparrow \text{PULM. VENOUS B. P.} \\
\uparrow \text{PULM. CAP. B. P.} \\
\uparrow \text{PULM. ART. B. P.}
\]