Various pathologic conditions may lead to acute failure of the right ventricle. The most common cause is pulmonary artery thromboembolism. Another relatively frequent etiology of right heart failure is occlusion of the right coronary artery with dysfunction of the right ventricle. Severe types of right-sided heart failure can develop after isolated infarction of the right ventricle.

Uncertainty may exist when the term “acute right heart failure” is used by older clinicians to indicate a terminal condition. Today this definition is no longer applicable.

In addition to these infrequent types of primary acute right ventricular failure, there are the more common forms of secondary right-sided heart failure resulting from chronic left ventricular failure. During the course of all three major clinical presentations – end-stage coronary artery disease, valvular defects, and cardiomyopathies – acute forms of right-sided heart failure frequently develop. Finally, right ventricular heart failure and primary pulmonary hypertension associated with chronic obstructive lung disease should be mentioned.

The clinical picture of patients with acute right heart failure presents with signs and symptoms of congestion before the right ventricle. Distended jugular neck veins and engorgement of the liver are characteristic signs of right heart failure. Acute congestion leads to hepatic tenderness secondary to distention of the hepatic capsule. Peripheral edema in the lower extremities and even anasarca develop in the later stages of the disorder. Chronic overload of the right heart with enlargement of the right ventricle eventually results in insufficiency of the tricuspid valve. Tricuspid regurgitation presents clinically with systolic pulsations of the liver. Severe forms secondary to pulmonary thromboembolism or right ventricular infarction may be associated with cardiogenic shock.

I. Pulmonary Thromboembolism

1. Etiology

In pulmonary thromboembolism the thrombotic material generally originates from the deep pelvic and femoral veins. The region of the superior vena cava is involved in only 10 to 15 percent of cases. Pulmonary embolism is particularly common following surgery or trauma with fractures in the lower extremities. Pulmonary embolism is not a rare event even after minor surgery such as cholecystectomy or pyelolithotomy. At present, standard therapy which emphasizes rapid mobilization of the post-operative patient clearly reduces the risk of thromboembolism.
The etiology of pulmonary embolism in surgical patients is related to venous stasis and absent muscular pumping in the lower extremities during anesthesia and in the early postoperative phase. Pulmonary embolism frequently occurs at 6 to 12 days postoperatively, in part associated with vigorous mobilization of the patient or prolonged bed rest. An increased risk of thromboembolism exists during pregnancy and with the use of oral contraceptives.

In a minority of patients, pulmonary embolism is followed by hemorrhagic infarction with a wedge-shaped infiltrate on chest x-ray. Frequently this leads to infarction pneumonia with painful pleurisy and fever.

2. **Clinical Presentation**

Massive pulmonary embolism is accompanied by dyspnea and symptoms of shock. This may be related to overdistention of the right ventricle and the acute onset of arterial hypoxia. The symptoms may mimic those of acute myocardial infarction with precordial pain and a sensation of impending doom. The major signs of massive pulmonary thromboembolism are severe dyspnea, tachypnea, cyanosis, tachycardia, hypotension, increase in central venous pressure, dysrhythmias and chest pain. Sudden death may occur from cardiogenic shock, bradycardia, or asystole.

Smaller pulmonary emboli may present with variable symptoms. In most cases, tachycardia occurs that cannot be readily explained. Patients complain of dyspnea with exercise.

The diagnosis of severe pulmonary embolism is made when significant dyspnea at rest and a reduced arterial PO$_2$ occur and the ECG indicates overload of the right ventricle with right ventricular injury. The findings in the electrocardiogram are similar to those seen with a diaphragmatic posterior wall infarct. Correspondingly, there are ST-segment elevations in lead II, aVF and III. The precordial leads V$_1$-V$_3$ also reveal signs of precordial right-sided ischemia. They can be clearly seen in the right ventricular leads (V$_r$/V$_r$) as well. A normal chest x-ray does not exclude the diagnosis of pulmonary thromboembolism, but may help to rule out left-sided heart failure or shock lung. In pulmonary embolism the x-ray rarely shows diagnostic pathologic changes. Occasionally the pulmonary vascular markings are reduced indicating diminished perfusion to a portion of the lung parenchyma.

3. **Diagnostic Procedures**

a) **Shock Symptoms**

When signs and symptoms of shock are present, the diagnostic procedure of choice consists of immediate pulmonary angiography with the simultaneous recording of pulmonary circulation pressures and cardiac output.

The indication for surgical intervention and embolectomy is present when there is nearly complete occlusion of one of the two major branches of the pulmonary artery with embolic material. However, if the embolic material is widely scattered and only visible in the periphery, surgical intervention is limited. A major therapeutic alternative is treatment with intravenous streptokinase.