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Until recently, the right ventricle (RV) was considered as a moderately passive conduit between the systemic and pulmonary circulations. This belief was supported by studies showing that complete destruction of the right ventricular free wall in dogs had no detectable impairment on overall cardiac performance (1). However, investigations in the 1970s demonstrated that right ventricular failure (RVF) has significant hemodynamic and cardiac performance effects (2).

Right ventricular failure has a similar incidence to that of left-sided heart failure, with each affecting about 1 in 20 of the population (3). In contrast with left-sided heart failure, which is often a chronic, progressive disease with a mortality four to eight times greater than that of age-matched general population (4), the outcome of RVF is broadly dependent on the underlying cause, resulting in either an acute or chronic condition.

The importance of the right ventricular involvement in heart failure is illustrated by the fact that ischemia following a myocardial infarction involving both the right and the left ventricle results in a greater mortality than isolated left ventricular ischemia (5,6).

Special attention is needed to avoid the development of RVF in intensive care unit (ICU) patients. Indeed, compared with the left ventricle, RVF progresses quickly from compensated stage to end-stage because of a vicious cycle (7). Indeed, the elevated right atrial and ventricular end-diastolic pressures eventually lead to an increased right ventricular end-diastolic volume, and tricuspid regurgitation. The latter aggravates organ congestion and decreases cardiac output; the heart is, therefore, unable to maintain an adequate function. Thus, the auto-aggravation of right ventricle function becomes an irreversible vicious cycle. In addition, decreased venous return to the left ventricle reduces left ventricular preload. This further exacerbates the situation as it causes decreased left ventricular output and systemic blood pressure and hence further impairment of organ perfusion, including the coronary arteries.

The RV plays a pivotal role in hemodynamic homeostasis, and changes in right ventricular function can have profound effects on the pulmonary and systemic circulation. Understanding the normal physiology and pathophysiology of the RV seems to be essential for the management of right ventricular failure. It should allow a quick and accurate diagnosis. The principal therapeutic goals of the early management of RVF depend on its underlying etiology, but primarily involve breaking the vicious circle of reduced cardiac

output. This will allow restoring adequate oxygen delivery to the myocardium and reducing right ventricular overload.

Treatment of RVF, therefore, should focus on alleviating congestion (limit volume loading), increasing right coronary artery flow, improving right ventricular contractility, and reducing right ventricular afterload (avoiding mechanical ventilation and high airway pressure). An example of a management algorithm is shown in the Figure below.

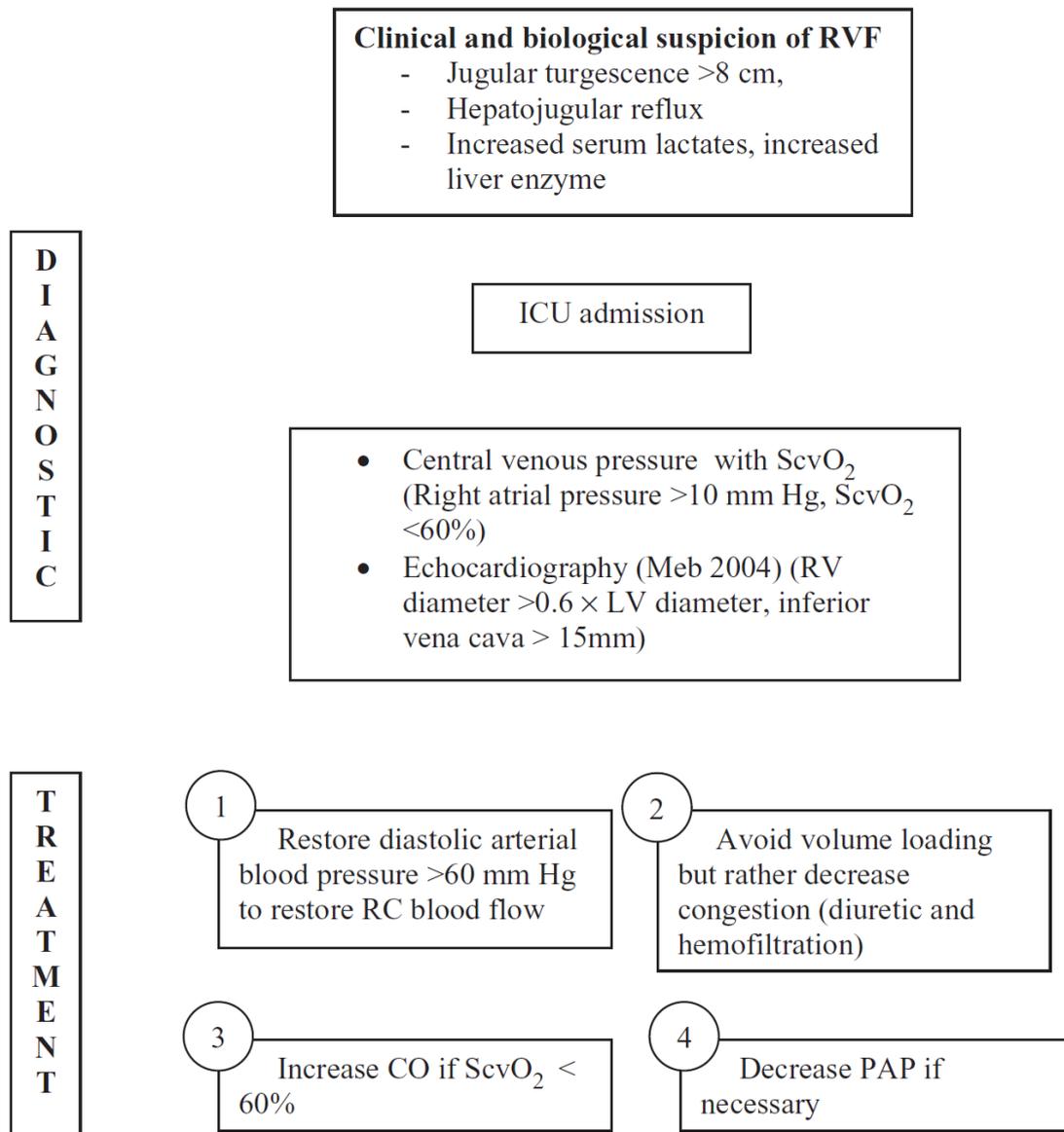


Figure. Example of management algorithm of right ventricular failure (RVF). ICU, intensive care unit; PAP, pulmonary arterial pressure; ScvO<sub>2</sub>, central venous oxygen saturation .

## References

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