

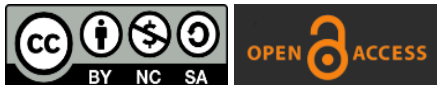
# Guyton's Theory of Venous Circulation: Relevance and Application

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## Abstract

*Guyton proposed the physiology of venous return and its characteristic curves nearly seven decades ago. This article aims to understand their relevance today—particularly the bedside application—by reviewing the literature. His concepts and experiments have withstood the test of time and profoundly influenced our physiological understanding of both normal and pathological states. Researchers have built upon these principles to further elucidate venous circulation under various clinical conditions. However, unlike the arterial circulation—which lies at the heart of hemodynamic monitoring and shock management—Guyton's venous return curves have not translated into bedside utility. Central venous pressure (CVP) remains integral to hemodynamic monitoring, though its concept did not emerge from Guyton's framework. Further studies are warranted to explore the clinical utility of Guyton's theory at the bedside.*

**Keywords:** Guyton; Venous return; Curves; Physiology; Application; Relevance.

## 1. Introduction

One may assume that blood returns to the heart simply because the left ventricle pumps it forward through the circulation. The Frank-Starling law explains this in terms of forward cardiac output. However, Guyton showed that it is the venous system that determines blood return to the heart, delineating physiological changes in various situations. The Guytonian framework deepened our understanding of venous circulation and its contribution to cardiac output [1].

## 2. Application of Guyton's Theory of Venous Circulation

Venous capacitance (distensibility) is about eight times greater than that of arteries. Approximately 70% of blood volume resides in the venous system, with 70% of this as unstressed volume and 30% as stressed volume. The

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unstressed volume does not distend veins, while the stressed volume contributes to venous pressure. The splanchnic circulation acts as a major reservoir of unstressed volume.

Despite a small pressure gradient (~6 mmHg) only, venous blood flows effectively to the right atrium (RA) because venous resistance is low. The pressure propelling this flow is called the mean systemic filling pressure (PMSF), and venous return (VR) depends on the gradient between PMSF and right atrial pressure (RAP):  $VR = (PMSF - RAP) / RVR$ . Venous resistance (RVR) is less understood than arterial resistance but relates to venous diameter—smaller diameters increase resistance. Guyton defined the 'peripheral resistance to venous return' as the sum of venous and arterial resistance [2]. External pressures, such as positive intrathoracic pressure during mechanical ventilation, increase RVR by compressing the inferior vena cava (IVC). High PEEP in hypovolemic patients can even collapse the IVC. Abdominal masses or thoracic outlet obstruction (e.g., cervical rib) can similarly increase venous resistance.

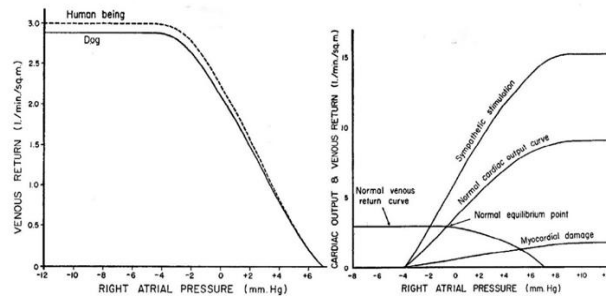
### 3. Clinical Scenarios Where Guyton's Concepts Apply

- Hypovolemic shock: Vasoconstriction recruits unstressed volume from the splanchnic reservoir, increasing PMSF and venous return.
- Venoconstriction paradox: Though venoconstriction raises RVR, the greater rise in PMSF predominates, increasing VR [2].
- Fluid resuscitation: Increases stressed volume and PMSF, enhancing VR if the patient is fluid responsive. Septic shock: Fluid administration raises PMSF without increasing RVR due to vasodilation, widening the PMSF–RAP gradient.
- Non–preload-dependent states (RV failure, cardiogenic shock): PMSF and RAP rise together, so the gradient does not change or may decrease.

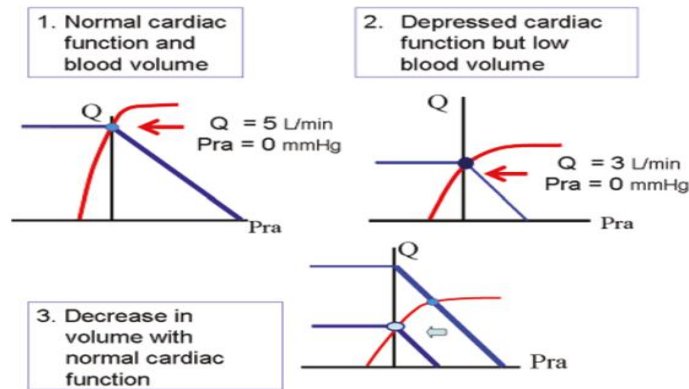
In prone ventilation, cardiac output improves in ~80% of patients. Proning increases intra-abdominal pressure, recruiting unstressed splanchnic volume (raising PMSF) and decreasing RAP through reduced pulmonary vascular resistance (PVR). The resulting higher PMSF–RAP gradient enhances VR. However, in nonresponders—due to high PVR, cardiac dysfunction, or hypovolemia—the gradient may fail to rise or the IVC may be compressed, impeding VR. This explains why the passive leg-raising test can be unreliable in intra-abdominal hypertension [3].

However as much as Guyton's theory helps us to understand the venous circulation physiology, does it help the physician at the bedside? The central component is RAP which is reflected in the CVP. The CVP was first used in 1966 by Ryan and Holland to see if blood loss during surgery could be estimated by CVP measurements [4]. An editorial note by Richard Theye followed this article: "Central venous pressure is a pressure measurement and is useful only as an index of right ventricular diastolic filling pressure.....CVP measurements are useful in diagnosing the basis and guiding the therapy of a reduction in cardiac output". The moot point here is that this done was independent of the Guytonian theory. The CVP works still well in estimating venous return given its limitations.

What we use at bedside is CVP as a way to understand the venous return and none of the other parameters (Figs. 1 and 2).



**Fig. 1.** a) Predicted venous return curves for the dog and human being. b) Equating the normal venous return curves with cardiac output in human beings. Adapted from Guyton AC et al. 1957.



**Fig. 2.** Guyton’s venous return–cardiac function curves showing how central venous pressure can be low with different cardiac outputs. Adapted from Magder S. Crit Care. 2012.

**Table 1:** Key Points Summary.

Concept	Description	Clinical Implication
Venous Return (VR)	Determined by PMSF–RAP gradient	Reflects preload and cardiac filling
PMSF	Mean systemic filling pressure driving VR	Increases with fluids or venoconstriction
RVr	Venous resistance depending on vein diameter	Rises with PEEP, IVC compression
Unstressed vs. Stressed Volume	Stressed volume contributes to venous pressure	Vasoconstriction or fluids shift volume to stressed compartment
Clinical Utility	CVP measurable; PMSF & RVr theoretical	Explains physiology but limited bedside use

#### 4. Conclusion

Guyton's theory of venous circulation remains foundational to physiological understanding but has yet to find direct, practical bedside application. Future developments may make its parameters more clinically measurable and useful to intensivists.

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