# Passive Leg Raising

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## Introduction

Passive leg raising involves the elevation of the lower limbs from the horizontal plane. It was used as an empiric rescue therapy for acute hypotension long before intensive care units (ICUs) were created. The hemodynamic effects of passive leg raising have been progressively elucidated. In view of its simplicity, there is renewed interest in passive leg raising as a means of predicting fluid responsiveness in the critically ill.

## Hemodynamic Effects of Passive Leg Raising

During elevation of the legs to 45°, gravity causes a translocation of venous blood from the legs toward the intrathoracic compartment. A study conducted in healthy subjects using a radionuclide method, showed that the volume transferred during this postural maneuver was 150 ml [1], but no other study has addressed this particular physiological issue. The transfer of blood from the legs operates through the splanchnic venous network and likely increases the mean circulatory pressure, i.e., the driving pressure of the systemic venous return toward the right atrium [2]. Passive leg raising thus increases right ventricular (RV) preload [3]. If the increase in RV preload is sufficient to increase the RV output – which is generally the case -this results in an increase in left ventricular (LV) filling and preload. In this regard, passive leg raising has been reported to induce significant increases in the pulmonary artery occlusion pressure (PAOP) [4–6] as well as in the LV end-diastolic volume (LVEDV) [7, 8], under various hemodynamic conditions.

Although passive leg raising has been demonstrated to increase cardiac preload parameters in previous studies, it did not increase cardiac output in all the studied patients [8, 9]. Indeed, in response to an increased left cardiac preload, cardiac output should increase only in patients with cardiac preload dependency, according to the Frank-Starling relationship [8, 9]. Moreover, Wong and colleagues found that the passive leg raising-induced increase in cardiac output of healthy subjects was 7% in normal conditions, but increased to 11% after a 500 ml blood withdrawal [10]. Hence, passive leg raising can be considered as a 'self-volume challenge' that could be used to assess fluid responsiveness.

Passive leg raising has the advantage of a short time delay during which it exerts its hemodynamic effects and a complete reversibility. In a study in critically ill patients, we measured blood flow in the thoracic aorta by means of esophageal Doppler monitoring during a passive leg raising maneuver [11]. The changes in aortic blood flow – an estimate of cardiac output – occurred within the first 30 seconds

in all the 71 patients included in this study [11]. Additionally, when the patients' legs were lowered, cardiac output returned to its baseline value within a few seconds: as the original body posture was restored, the changes in cardiac output completely vanished [11]. Thus, passive leg raising should be considered as a reversible 'self-volume challenge', testing the volume response without administering fluid.

The time during which passive leg raising is sustained must also be considered. Some studies performed in healthy subjects found that the increase in cardiac output induced by passive leg raising disappeared after a few minutes although the legs were maintained elevated longer [12]. This may be due to redistribution of venous blood or to lung sequestration of the blood transferred toward the intrathoracic compartment. While the effects of a short duration passive leg raising vanish rapidly after down-tilt, it must be acknowledged that some degree of pulmonary sequestration could appear if passive leg raising is prolonged for minutes and the volume translocated toward the great veins could progressively fall. In such conditions, the translocation of blood toward the intrathoracic compartment would not be completely reversible [12, 13]. To summarize, for correct assessment of the hemodynamic response to passive leg raising, one must be able to observe its effects in a short time. In particular, one must choose a technique of hemodynamic monitoring that is able to track the rapid and transient changes in cardiac output induced by passive leg raising.

## How to Perform Passive Leg Raising?

A major advantage of the passive leg raising maneuver as a 'self-volume challenge' is its easiness to perform. To avoid any risk of gastric regurgitation, caution must be kept to down-tilt the trunk of the patient at the horizontal level and not lower; passive leg raising is not a Trendelenburg maneuver. Passive leg raising and Trendelenburg maneuvers may have different hemodynamic effects [14], since with the Trendelenburg position, an unknown amount of venous blood is sequestrated in the head compartment while another amount from the lower body compartment is transferred toward the thorax. Furthermore, the baroreceptor stimulation observed during a head-down tilt may not occur during passive leg raising. More importantly, gastric regurgitation and ensuing aspiration pneumonia may occur with the Trendelenburg position.

In practice, passive leg raising should be performed simply by means of the automatic system of the patient's bed (Fig. 1). If the patient is managed in the semi-

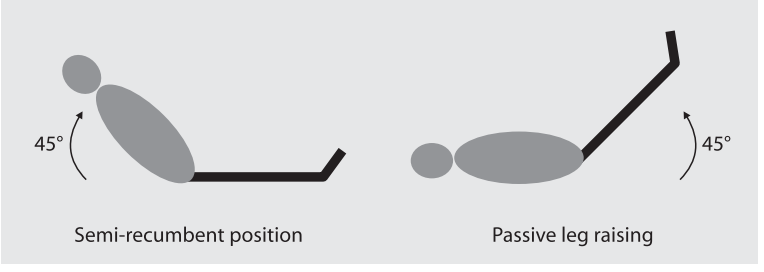


**Fig. 1.** How to perform passive leg raising? An automatic pivot of the bed allows the patient to be transferred from the semi-recumbent position to a passive leg raising posture with ease and without inducing pain. The lower limbs are raised at a 45° angle while the patient's trunk is tilted down to a supine position. Thus, the angle between the trunk and the lower limbs remains unchanged (135°).

recumbent position at 45° as recommended [15], passive leg raising only consists of pivoting the bed, without changing the angle between the trunk and the lower limbs. In this case, the trunk is lowered to the horizontal position while the lower limbs are tilted upwards to 45°. Compared to leg elevation performed by manually moving the patient's legs and holding them in position, automatic leg elevation avoids any clinician effort and enables the passive leg raising posture to be maintained for the time (around one minute) necessary to assess its full hemodynamic effects. A second great advantage of this technique is that it avoids any discomfort for the patient. Discomfort or pain may occur due to skin contact, body manipulation, or hip flexion and may induce sympathetic stimulation, which would have cardiovascular effects potentially leading to an erroneous interpretation of the hemodynamic effects of the passive leg raising. When performing passive leg raising using the automatic system of the electrical bed, we did not observe any significant increase in heart rate, and presumably there was no other significant sympathetic activation, even in patients who did not receive any sedation or analgesia [11].

## Prediction of Fluid Responsiveness by Passive Leg Raising in the Critically Ill

### The Issue of Predicting Fluid Responsiveness

Not all patients with circulatory failure respond to fluid infusion by a significant increase in cardiac output [16]. Indeed, in about half of the critically ill patients who are considered for fluid therapy, the heart is working on the upper and flat part of the curve describing the Frank-Starling relationship [16]: In such patients, any increase in cardiac preload cannot result in an increase in cardiac output and the patient is considered as a 'non-responder' to fluid infusion. If administered, volume could exert harmful rather than beneficial effects in such patients, such as an increase in lung water, worsening of gas exchange and lung compliance, increase in tissue edema, RV dilation with left shift of the interventricular septum. The need to avoid such deleterious effects of a volume infusion which would not increase cardiac output, has stimulated an interest in how to define predictors of preload responsiveness. Static measures of cardiac preload of any sort do not reliably predict a patient's response to fluid administration [17] and assessment of fluid responsiveness must rather be based on the response to dynamic tests which induce transient changes in cardiac preload [18].

For this purpose, a first method is to analyze the respiratory variation in hemodynamic signals [16]; changes in intrathoracic and transpulmonary pressures induced by mechanical ventilation induce cyclic and regular changes in cardiac preload that result in significant cyclic changes in stroke volume in the case of preload dependency. Thus, it has been hypothesized that such cyclic changes of stroke volume (or of its surrogate) could be taken as a marker of preload-responsiveness in mechanically ventilated patients [16]. Accordingly, respiratory variation of arterial pulse pressure [19], of subaortic outflow [20], of arterial pulse contour [21, 22], and of the descending aortic flow [23] have been demonstrated to be reliable markers of volume responsiveness. However, the predictive value of these respiratory variation indexes may be lost in the cases of spontaneous breathing activity or arrhythmias. In such frequent situations, the variation in stroke volume may not be due to concomitant changes in cardiac preload but rather to the heterogeneity of the intrathoracic pressure variation or of the cardiac cycle length. Accordingly, we demonstrated that in patients with spontaneous breathing activity, the respiratory variation of arterial

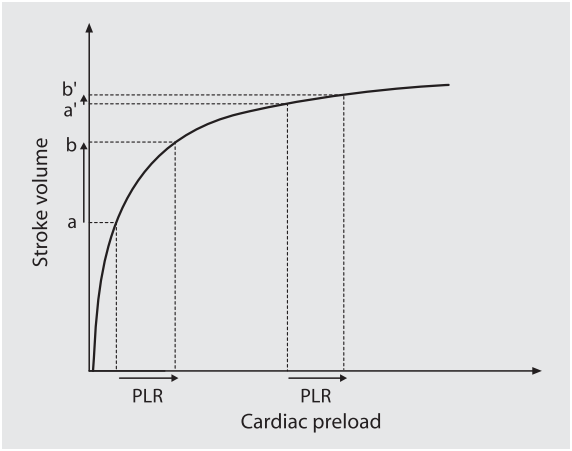


**Fig. 2.** Passive leg raising (PLR) acts like a 'self-volume challenge'. Passive leg raising allows estimation of which part of the Frank-Starling curve the patient's heart is working on. If the increase in cardiac preload induced by passive leg raising induces significant changes in stroke volume (from a to b), the heart can be supposed to work on the initial part of the curve and the patient will likely respond to fluid infusion. Conversely, if the same changes in cardiac preload during passive leg raising do not significantly change stroke volume (from a′ to b′), the heart is likely preload independent and fluid should not be administered.

pulse pressure was of poor specificity for predicting fluid responsiveness [11] and this has been confirmed by others [24].

Since passive leg raising induces a transient increase in cardiac preload, it is considered as another method for predicting the part of the Frank-Starling relationship on which the patient's heart is actually working: On the initial, steep part of the curve, where passive leg raising may induce large changes in cardiac output, or on the upper, flat part, where passive leg raising is supposed not to induce any change in cardiac output (Fig. 2). The issue has thus emerged as to which estimate of cardiac output or stroke volume is most accurate at assessing the effects of passive leg raising. Due to the short-term effects of passive leg raising, a real-time cardiac output monitoring technique would be particularly appropriate for rapidly tracking the transitional hemodynamic changes related to passive leg raising. In this regard, the automatic, semi-continuous measurement of cardiac output by thermodilution is not suitable for assessing the effects of passive leg raising.

## Effects of Passive Leg Raising on Arterial Pulse Pressure

Boulain et al. [5] performed passive leg raising in critically ill patients with acute circulatory failure who were sedated and receiving mechanical ventilation. These authors observed that passive leg raising-induced increases in arterial pulse pressure – taken as a surrogate of stroke volume – correlated significantly with the changes in cardiac index induced by subsequent fluid loading. However, the correlation between the passive leg raising-induced changes in pulse pressure and the volume-induced changes in cardiac index was only fair, maybe because the arterial pulse pressure is far from the best estimate of stroke volume. Indeed, pulse pressure depends not only on stroke volume, but also on arterial compliance and is influenced by complex propagation/reflection of the arterial waveform along the arterial tree.

## Effects of Passive Leg Raising on the Aortic Blood Flow

Another beat-by-beat estimate of stroke volume can be provided by esophageal Doppler. By means of a small-caliber probe located in the esophagus, this minimally invasive monitoring device measures the blood velocity in the descending thoracic aorta.

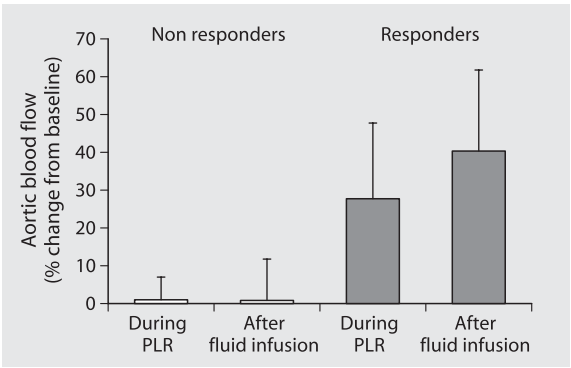


**Fig. 3.** Prediction of fluid responsiveness by passive leg raising (PLR) and esophageal Doppler in the critically ill. In 71 critically ill patients with acute circulatory failure, the aortic blood flow was measured by esophageal Doppler during a passive leg raising test and after fluid infusion [11]. In patients who did not respond to fluid infusion (non-responders, n = 34, open bars), passive leg raising did not alter the aortic blood flow significantly before fluid infusion. Conversely, in patients who responded to fluid infusion by an increase in aortic blood flow ≥ 15% (responders, n = 37, full bars), the fluid-induced changes in aortic blood flow were preceded by a significant increase in aortic blood flow during passive leg raising.

Simultaneous measurement – or estimation – of the aortic diameter at the same level allows calculation of the aortic blood flow, which has been demonstrated to correlate with cardiac output [25]. Moreover, esophageal Doppler monitoring devices are able to reliably track changes in cardiac output with various hemodynamic interventions [23, 26, 27].

In 71 mechanically ventilated patients with acute circulatory failure, we measured aortic blood flow during passive leg raising and during a subsequent fluid infusion of 500 ml saline [11]. In 37 patients, fluid infusion induced an increase in aortic blood flow greater than 15%, defining a positive fluid response. In these volume responders, a passive leg raising maneuver performed before fluid infusion significantly increased aortic blood flow by 28 ± 21% while passive leg raising did not alter aortic blood flow in non-responders (Fig. 3). Moreover, an increase in aortic blood flow greater than 10% during passive leg raising allowed a positive fluid response (increase in aortic blood flow ≥ 15%) to be predicted with a sensitivity of 97% and a specificity of 94% [11]. Our results were confirmed by a recent similarly designed study [28]. In our study, the passive leg raising-induced changes in arterial pulse pressure were of poorer predictive value for fluid responsiveness than the passive leg raising-induced changes in aortic blood flow, suggesting that the latter is a more direct estimate of cardiac output; if passive leg raising increased pulse pressure by ≥ 12%, the ensuing response to volume expansion could be predicted with a sensitivity of 60% and a specificity of 85% only and the receiver operating characteristic (ROC) curve analysis confirmed the superiority of aortic blood flow over pulse pressure for assessing the hemodynamic response to passive leg raising.

Importantly in our study, we specifically identified a subgroup of patients with spontaneous ventilator triggering or with arrhythmias [11]. As expected, the respiratory variation of arterial pulse pressure, which was also calculated, was not reliable for predicting the hemodynamic response to volume in this category of patients (see above). In contrast, the response of aortic blood flow to passive leg raising remained an excellent predictor of fluid responsiveness [11] in this subgroup of patients with inspiratory efforts or arrhythmias. Moreover, passive leg raising exerts its effects on cardiac preload over a period that includes numerous respiratory, and many more cardiac, cycles. Thus, passive leg raising appears to be able to resolve the crucial problem of predicting fluid responsiveness in the large population of patients who are arrhythmic or who do not receive deep sedation.

### Effects of Passive Leg Raising on Cardiac Output Measured by Pulse Contour Analysis

The automatic analysis of the contour of the systemic arterial waveform provides a beat-by-beat estimation of stroke volume. Since it is a more direct estimate of cardiac output than pulse pressure, it has been logically tested for measuring the hemodynamic effects of passive leg raising with a view to assessing fluid responsiveness. As observed with esophageal Doppler, the passive leg raising-induced increase in the pulse contour cardiac index reliably predicted a positive response to fluid loading. In a preliminary clinical study, we observed that an increase in cardiac index $\geq 12\%$ during passive leg raising predicted fluid responsiveness (defined by a fluid-induced increase in cardiac index $\geq 15\%$) with a sensitivity of 70% and a specificity of 92% [29]. Interestingly, all the patients in this study had spontaneous breathing activity or arrhythmias, which confirms the particular interest of passive leg raising in such conditions. Whether other techniques measuring beat-by-beat stroke volume and cardiac output, such as echocardiography or thoracic bioimpedance, could similarly predict fluid responsiveness by observing the effects of passive leg raising is quite likely but remains to be demonstrated.

## Conclusion

It has now been well demonstrated that the main hemodynamic effect of passive leg raising is to increase cardiac preload by shifting blood from the lower limbs toward the intrathoracic compartment. Passive leg raising can be used at the bedside as a 'self volume-challenge', reversible and easy-to-perform. This very simple postural maneuver has been demonstrated to be a valuable tool for predicting fluid responsiveness: the response of estimates of stroke volume to a short passive leg raising maneuver is correlated to the response of cardiac output to a subsequent fluid administration. Thus, passive leg raising can be considered as one of the tools of the functional hemodynamic monitoring concept [18]. Interestingly, this dynamic method remains fully reliable in patients with spontaneous triggering of the ventilator or with arrhythmias, conditions where prediction of fluid responsiveness cannot be provided by the respiratory variation of hemodynamic signals.

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