The Egyptian Journal of Hospital Medicine (April 2023) Vol. 91, Page 3913-3919

Can Passive Leg Raising Predict Fluid Responsiveness in Intra-Abdominal Hypertension Mechanically Ventilated Surgical Critically Ill Patients

Mohammed Abdel Monem Saeed[1], Tarek Osman AbdelAzim[2], Deyaa Mohamed Ibrahim[3], Alaa Mohamed Hussein[4]

Departments of [1]Critical Care Medicine and [2]General surgery, Faculty of Medicine, Helwan University, Egypt
Department of [3]Critical Care Nursing, Faculty of Nursing, Helwan University, Egypt
Department of [4]Clinical Pharmacist, Faculty of Pharmacy, Helwan University, Egypt
***Corresponding author:** Mohammed Abdel Monem Saeed, **Mobile:** (+20) 01099292999, **E-Mail:** mohammedicu1@gmail.com

ABSTRACT

Background: The fluid responsiveness of patients who are mechanically ventilated is evaluated using the reversible fluid loading technique known as passive leg raising.

Objective: This study purpose to determine if intra-abdominal hypertension, which reduces venous return, affected the ability of passive leg raising to detect fluid responsiveness in critically ill patients.

Patient and methods: Our study is a prospective study done at the critical care Medicine Department of Helwan University Faculty of Medicine on 400 mechanically ventilated patients with a pulse pressure variation greater than 12%. The esophageal Doppler was used to continually monitor the stroke volume. The bladder pressure was used to calculate intra-abdominal pressure. Fluid loading with 500 ml of saline was conducted after a passive leg-raising exercise and a return to baseline, with hemodynamic parameters recorded at each stage.

Results: Four hundred patients were divided into two groups based on their response to passive leg raising: responders (at least a 12% increase in stroke volume) and non-responders (all patients in both groups were responders to volume loading). Two hundred patients responded to passive leg raising (50%), whereas the non-responders (i.e. false negatives) were similarly 50%. At baseline, the non-responders to passive leg raising had considerably greater median intra-abdominal pressure than the responders to passive leg raising (20 [5.5]- vs 10 [4.5], respectively, with a p-value < 0.001).

Conclusion: Passive leg raising has false negatives are caused by intra-abdominal pressures more than 12 mmHg. In severely sick ventilated patients, intra-abdominal pressure (IAP) should be assessed before doing passive leg raising.

Keywords Intra-abdominal pressure, Passive leg raising, Mechanical Ventilation.

INTRODUCTION

Fluid treatment is the first-line therapy in critically sick hypotensive patients because insufficient fluid supply is harmful [(1)]. Passive leg raising increased stroke volume was used to forecast fluid response in individuals who are extremely ill [(2)]. Although multiple studies [(2, 3)], indicated that passive leg raising had good sensitivity in septic and postoperative patients, the IAP levels of the patients who were included in these studies were not disclosed.

Intra-abdominal hypertension (IAH) lowered venous return, which reduced cardiac output [(4-6)]; this is due to constriction induced by increased IAP of the inferior vena cava [(4)]. The foundation of the passive leg raising movement is the recruitment of splanchnic and leg blood [(7)]. The ICU common pathophysiology of intra-abdominal hypertension affects this maneuver's capacity to identify fluid responsiveness [(8)].

This study purpose to determine if intra-abdominal hypertension, which reduces venous return, affected the ability of passive leg raising to detect fluid responsiveness in critically ill patients.

PATIENT AND METHODS

Our study was performed on four hundred patients who were responders to volume expansion in the period from April 2020 till October 2022 in the Critical Care Department Faculty of Medicine Helwan University, in the general intensive care unit after approval of the ethical committee.

We included patients in our trial with circulatory failure, were receiving mechanical ventilation while sedated, and had a pulse pressure variation (Δpp) of greater than 12%. We also chose to track intra-abdominal hypertension.

Blood pressure during systole of less than 90 mmHg indicates circulatory failure, chronic lactic acidosis, or a requirement for vasoactive medications.

The tidal volume for each trial subject was kept to a minimum of 8 ml/kg. These were the specific criteria we used to exclude people: Exclusions include those who are under the age of 18, women who are pregnant, those who have a known mitral or aortic valve disease, those who have deep vein thrombosis or are wearing elastic compression stockings, and those who have severe acute respiratory distress syndrome that prevents them from finishing the PLR.

Measurements:

At baseline, demographic information and ventilator parameters (tidal volume, plateau pressure (P_{plat}), and end expiratory pressure) were collected (such as age, gender, weight, height, body mass index, and the simplified Acute Physiology Score II).

A trained researcher who was uninformed of the clinical data orally implanted an esophageal Doppler probe into each patient (Cardio Q: Gamida Eaubonne, France).

Received: 05/11/2022
Accepted: 06/01/2022

To achieve the best signals for descending aorta blood velocity, the optimal probe location was modified.

The programme continuously measured the aortic blood flow and automatically determined the average stroke volume over 10 seconds. Before the trial began, the stroke volume was checked, and 400 patients had their stroke volume assessed twice by skilled fixed investigators.

The mean difference between the two values was computed and divided by that value. The monitor's algorithm (Philips Intellivue Mp 70; Philips, Suresnes, France) was used to automatically determine the pulse pressure fluctuations (Δpps) [9, 10].

Heart rate, mean arterial pressure, systolic pressure, and diastolic pressure were all measured.

After injecting 25 ml of saline and zeroing the transducer at the midaxillary line, the IAP was measured in the supine position using the bladder pressure in accordance with the standards of the worldwide Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome [11].

Study Design:

Study protocol included only surgical postoperative patients with abdominal surgery and Δpp > 12% who fulfilled the inclusion criteria which is base 1 up right 45º position underwent passive leg raising maneuver (PLR) then returned back to base 2 upright 45º again then underwent volume expansion by 500 ml saline where patients were classified into responders to volume expansion and Non-responders to volume expansion, non-responders to volume expansion were not analyzed (false positives of Δpp) and were excluded forty cases from the study as they were non-responders to volume expansion which started by four hundred & forty patients; then the responders to volume expansion were classified into responders to passive leg raising (PLR+) and non-responders to passive leg raising (PLR-), then retrospective comparison between the two groups were done.

At baseline, IAP, ventilator data, and demographic information were all measured. Each of the four consecutive phases involved the measurement of hemodynamic parameters. The patient was placed in a semi-recumbent position for the initial measurements (the "base 1" condition) (i.e. with the body at an angle of 45º to bed plane).

About a minute after the legs were raised, second measurements were collected at the aortic blood flow peak of the legs had been lifted to a 45-degree angle by an automated bed elevation mechanism (in the "PLR" condition).

Third, the restoration of the baseline (in the semi-recumbent position) following PLR monitoring (in the "base 2" condition).

Fourth, Following VE, measurements were taken using 500 ml of saline over a 30-minute interval (in the volume expansion [VE] condition).

Patients were labeled as false positives for Δpp and were excluded from analysis if their between base 2 and VE, the stroke volume did not increase by at least 12%. Considering the increase in SV brought on by PLR, patients were subsequently split into two groups: PLR responders (PLR+) and non-responders (PLR-).

Retrospective comparisons between the two groups (PLR+ and PLR-) were made. A 12% rise in SV was thought to be clinically meaningful [12]. Passive leg raising non-responders (PLR-) were believed to be a false negative for PLR, mechanical ventilation parameters were not changed during the study.

Ethical consent:

The study was authorized by Helwan University's Ethical Institutional Review Board. All study participants provided written informed permission after being informed of our research's goals. The Declaration of Helsinki for human beings, which is the international medical association's code of ethics, was followed during the conduct of this study.

Statistical analysis

SPSS version 20 for Windows® (IBM SPSS Inc, Chicago, IL, USA). The median and interquartile range serve as representations for continuous variables. The qualitative parameters were noted in terms of quantity and percentage. A Mann-Whitney U test was used to compare the PLR+ and PLR- groups, and a Wilcoxon rank-sum test was employed to evaluate the change in data from one stage to the next. The threshold for significance was set at a P-value of 0.05 or less. In order to calculate the area under the curve $\pm$ SEM for intra-abdominal pressure, a receiver-operating characteristic was used. The intra-abdominal pressure cut off value for false negative PLR prediction with the greatest sensitivity and specificity was determined using the best accuracy and best positive probability ratio.

To identify patient characteristics that were independently associated with PLR non-response, a stepwise multivariate logistic regression was used. The odds ratio and their 95% confidence intervals (95% CIs) were computed, with the exception of intra-abdominal pressure, for which the threshold value was established using the receiver-operating characteristic curve. For quantitative data, a threshold was established using the population's median value.

RESULTS

All demographic data and mechanical ventilation settings were non-significant statistically.

Table (1): Demographic and Mechanical ventilation data

		Non-responders to leg raising	Responders to leg raising	Test of sig.		
		N (%) Mean ± SD	N (%) Mean ± SD	Value	P Value	Sig.
Sex	Male	113 (56.5%)	113 (56.5%)	$X^2 = 0$	1	NS
	Female	87 (43.5%)	87 (43.5%)			
AGE		48.78 ± 9.09	48.82 ± 9.02	t = -0.04	0.969	NS
Height		170.06 ± 6.01	79.98 ± 5.84	t = 0.1	0.918	NS
Weight		80.13 ± 5.99	79.8 ± 5.84	t = 0.25	0.800	NS
BMI		23.57 ± 1.8	23.54 ± 1.84	t = 0.17	`0.868	NS
APACHE II		7.16 ± 1.49	7.15 ± 1.53	t = 0.03	0.974	NS
MV Setting	VT	641 ± 47.93	639.8 ± 46.75	t = 0.25	0.800	NS
	P.P	22.59 ± 1.7	22.6 ± 1.66	t = 0.06	0.953	NS
	PEEP	5.0 ± 1.2	5.0 ± 1.1	t =-0.05	9.966	NS

All hemodynamics parameters were non-significant statistically when compared between the two groups, except the intra-abdominal pressure which was significant statistically.

Table (2): Hemodynamic characteristics at baseline (Base 1 Step)

Total population (n = 400)		Non-Responders to passive leg raising maneuver (n = 200)	Responders to passive leg raising maneuver (n = 200)	P-value	Sig.
Stroke volume (m l)	60.42 ± 2.25	60.12 ±3.25	60.74 ±2.76	0.9	NS
Pulse pressure variation %	16.07 ± 1.51	16.04 ± 1.98	16.1 ± 1.34	0.8	NS
Heartrate (Beat/min)	106 ± 3.06	106 ± 3.02	106 ± 3.09	0.442	NS
Mean arterial Blood pressure (MAP)	68 ± 1.9	68 ± 2.21	68 ± 1.73	0.6	NS
Systolic Blood pressure (SBP)	95 ± 2.2	95 ± 1.92	95±3.12	0.8	NS
Diastolic Blood Pressure (DBP)	55 ± 2.4	55 ± 1.67	55 ± 3.1	0.7	NS
Central venous pressure, mmHg	11 ± 1.72	11 ± 2	11 ± 1.47	0.8	NS
Cardiac output L/min	6.1 ± 1.85	6.1 ± 1.9	6.1 ± 1.73	0.8	NS
Intra-abdominal pressure	15	22	8	<0.001	S

All parameters were expressed in mean and standard deviation where the comparison between all the hemodynamics parameters in the base 1 to PLR were all non-significant statistically, where comparing the same parameters between Base 2 and volume expansion were all significant statistically.

Table (3): Hemodynamics parameters for Non-responders to the passive leg raising maneuver at each step and in between steps comparison

	Base 1	**Passive leg raising maneuver**	**p-value**	**S**	**Base 2**	**VE volume expansion**	**p-value**	**S**
Stroke volume (ml)	60.12±3.25	61.33±2.69	0.7	NS	59.71±2.11	75.54±3.49	<0.001	S
Pulse pressure variation %	16.04±1.98	16.06±2.04	0.75	NS	15.04±1.3	16.28±1.37	<0.00l	S
Heart rate (Beat/min)	106±3.02	106±2.33	0.9	NS	106±2.18	95±3.62	<0.00l	S
Mean arterial Blood pressure (MAP)	68 ±2.21	68 ± 2.22	0.9	NS	70 ± 1.64	78 ± 3.45	<0.001	S
Systolic blood pressure (SBP)	95±1.92	95 ±1.57	0.9	NS	96±2.77	110±5.83	<0.001	S
Diastolic blood pressure (DBP)	55±1.67	55±2.2	08	NS	55±2	66±2.72	<0.00l	S
Central venous pressure (mmHg)	11±2	11.5±0.97	0.7	NS	12 ±1.85	17.5±1.66	<0.001	S
Cardiac output (L/min)	6.1±1.9	6.2±1.63	0.6	NS	6.0 ± 1.73	6.8±1.6	<0.001	S

All parameters were expressed in mean and standard deviation where the comparison between all the hemodynamics parameters in the base 1 to passive leg raising maneuver were all significant statistically in favor of the good response to passive leg raising maneuver, also all parameters when compared between base 2 and volume expansion, were all statistically significant expressing good response to volume expansion.

Table (4): The hemodynamics parameters for responders to passive leg raising maneuver at each step and in between steps comparison

	Base 1	Passive leg raising maneuver	p-value	S	Base 2	VE volume expansion	p-value	S
Stroke volume (ml)	60.2±74.76	67.41±1.76	<0.001	S	57.41± 1.73	73.78±.2.7	<0.001	S
Pulse pressure variation %	16.1±1.34	14.2±1.39	<0.001	S	16.09±1.37	10.08±1.38	<0.00l	S
Heart rate (Beat/min)	106±3.09	96±2.78	<0.001	S	73±2.01	98±2.45	<0.00l	S
Mean arterial Blood pressure (MAP)	68 ±1.73	80 ± 2.6	<0.001	S	73 ± 2.01	85 ± 4.26	<0.001	S
Systolic blood pressure (SBP)	95±3.12	107 ±4.78	<0.001	S	103±3.68	118±4.51	<0.001	S
Diastolic blood pressure (DBP)	55±3.1	67±4.25	<0.001	S	63±3.51	72±4.29	<0.00l	S
Central venous pressure (mmHg)	11±1.47	67±4.25	<0.001	S	63 ±2.14	18±1.64	<0.001	S
Cardiac output (L/min)	6.1±1.73	6.3±1.91	<0.001	S	6.0 ± 1.83	6.8±1.6	<0.001	S

Figure (1) receiver operating characteristic curve of intra-abdominal pressure (IAP) to detect non-responders to passive leg raising maneuver.

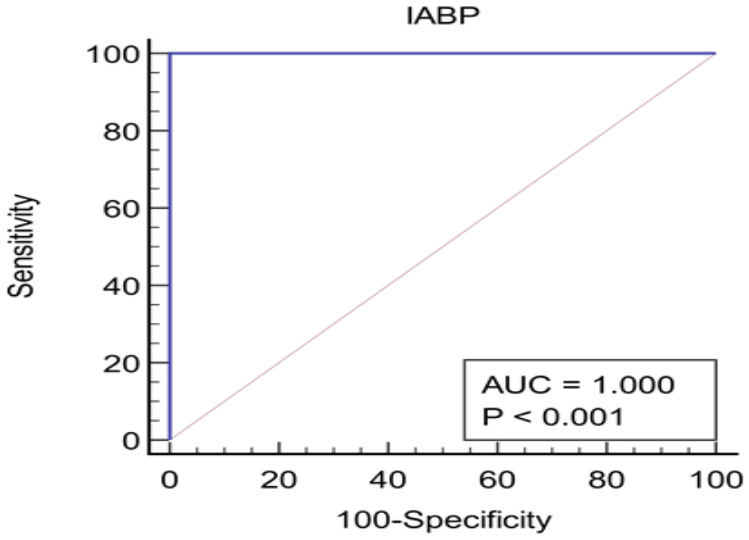

Figure (1): ROC curve of IABP to detect non-responders

	AUC	95% CI	p value	sig.	Cutoff point	Sensitivity	Specificity	PPV	NPV
IABP	1	0.991 to 1.000	<0.0001	S	**≥12**	100%	100%	100%	100%

Receiver operating characteristic curve of intra-abdominal pressure (IAP) with cut off value of ≥ 12 mmHg to prevent responding to passive leg raising maneuver, with 95% CI 0.991 – 1.000 , p-value < 0.0001, sensitivity 100%, specificity 100%., positive predictive value 100% and negative predictive value 100%.

DISCUSSION

In our study we decided to monitor IAP and to do volume expansion in surgical mechanically ventilated patients. Fifty percent false negative cases were evident when applying the passive leg raising maneuver. We found that intra-abdominal pressure of ≥ 12 mmHg can precisely identify these false negative results to passively lifting your legs. A fluid loading that is reversible technique is the passive leg raise movement [(2, 7, 12-15)].

The thoracic compartment receives venous blood from the legs and the splanchnic compartment when patients are subjected to the passive leg raising procedure (PLR), lifting the legs at a 45-degree angle while keeping the body horizontal is how you do this [(7)]. This increase in venous return can increase stroke volume so long as both ventricles contract along the steep portion of the Frank-Starling curve (and cardiac output).

Therefore, it is possible to assess how the heart's load reliance changes when passive leg raising (PLR) increases stroke volume. Numerous studies have shown that passive leg raising (PLR) can distinguish between patients who respond to volume expansion (VE) with fluid and those who do not in patients who need mechanical ventilation [(2,3)], those who breathe spontaneously [(14, 15)], and those who have arrhythmias [(2)]. The patients' intra-abdominal pressure values were not stated in any of these articles, nonetheless, various studies in the literature have demonstrated how this parameter affects cardiac output and venous return [(4-6,16-21)]. When IAP surpasses right atrial pressure, the abdominal inferior vena cava collapses and a vascular water fall develops, reducing venous return. **Takata and colleagues** proved this and introduced the novel notion of "abdominal vascular zone Conditions" [(4)].

PLR depends on venous blood flowing into the intrathoracic compartment from the legs and splanchnic veins; IAH induced inferior vena cava compression likely precluded a "endogenous venous return" effect.

The pulse pressure variation did not diminish with passive leg raising in the passive leg raising negative group either, confirming the idea that there was no increase in venous return.

Intra-abdominal hypertension is described as a prolonged or recurrent pathologic rise in intra-abdominal pressure of 12 mm Hg or greater by the World Society of Abdominal Compartment Syndrome [(10)]. In the case of animal models, the threshold of intra-abdominal pressure that reduces stroke volume has variably been reported as 20 mmHg, 25 mmHg [(19-21)], and 30 mmHg [(5,16)]. Pneumo-peritoneum was used to maintain the intra-abdominal pressure at 10, 12, or 14 mmHg in further animal and human investigations [(6, 22, 23)].

Despite the fact that ICUs tend to employ a comparable range of pressure levels, none of these research observed or discussed the impact of intra-abdominal pressures between 12 and 24 mmHg [(24)].

In our investigation, passive leg raising (PLR) false negative results occurred in around 50% of cases, which is higher than previously reported in the literature.

This criterion of 8% is lower than our advised limit of 12%, which might account for the divergence in the findings. In twenty-two critically sick patients, **Lafanechère *et al.*** [3] found that a negative reaction to passive leg raising (PLR-) resulted in an increase in cardiac output of at least 8% predicted fluid responsiveness with a 90% sensitivity.

Monnet *et al.* [2] discovered that a lower threshold value could be utilised to identify between patients who reacted to volume expansion and those who did not, with 97% sensitivity, using a passive leg raise-induced 10% increase in cardiac output in 71 critically sick patients. Only non-surgical patients, whose prevalence of intra-abdominal hypertension was likely lower, were evaluated by later writers [8,24].

On the other hand, 100 percent of our patients received surgery. Therefore, the larger frequency of intra-abdominal pressure may also help to explain our high rate of false negative findings. The intra-abdominal pressure in 79% of our patients was less than 12 mmHg. Also, compared to previously reported and published research, we had a greater prevalence of intra-abdominal hypertension. **Malbrain *et al.*** [24] discovered that 58.8% of general ICU patients in their one-day multicenter trial had intra-abdominal hypertension.

The ICU patients in the same research had an IAP of ≥ 15 mmHg in 28.9% of cases. In our investigation, 40 patients were removed because they despite a hydration infusion, he did not respond pulse pressure fluctuation of more than 12%. This indicator, which serves as a stand-in for fluctuations in stroke volume, has been demonstrated to reliably a fluid responsiveness prediction. **Michard *et al.*** [25] showed a sensitivity of 94% and a specificity of 96%, and several additional research supported these findings [26].

Right ventricular dysfunction patients have previously shown that ICU patients typically exhibit this index's false positive [27].

Additionally, the pulse pressure fluctuation in a group of individuals with intra-abdominal hypertension or abdominal compartment syndrome never been carefully studied [28].

It was shown that intra-abdominal hypertension causes a more pronounced rise in pulse pressure variation (Δpp), and that volemia-related alterations are not the only cause of this variation. This conclusion was made by **Duperret *et al.*** [21]**.**

Although in cases of intra-abdominal hypertension a threshold value of 20.5% was needed to evaluate fluid responsiveness, pulse pressure variation (Δpp) can reliably predict fluid responsiveness. **Renner *et al.*** [19] came at this conclusion using an animal model of intra-abdominal hypertension.

As a result, our forty false positives may have been caused by the threshold value being higher than 12% or by right ventricular dysfunction, as previously indicated [27]**,** in other investigations [28]. Due to the high intra-abdominal pressure of (14, 16, 18, 20, 22 and 24 mmHg) and the pulse pressure variation (Δpp) of 12%, 14%, and 16% in our forty eliminated false positive instances, both processes may be contributing to the false positivity.

One of the good impact of our study is that we had done our study on a large scale of surgical patients admitted in the ICU as a postoperative abdominal surgery patients which is different from the vast majority of studies done [3,7,14,29]. One of the drawbacks of our study is that we assessed intra-abdominal pressure in the supine position shortly before base 1, and we were unaware of the actual intra-abdominal pressure that prevented venous return in the passive leg raising posture. The World Society of Abdominal Compartment Syndrome Consensus recommends that intra-abdominal pressure be monitored consistently and uniformly each time [11].

Aortic cross-sectional areas were calculated with a normogram rather than measured when we evaluated stroke volume fluctuation using esophageal Doppler, which anybody may argue lacks accuracy [30].

However, even in studies that connected fluctuations in stroke volume with cardiac output, thermodilution was still substantially associated with the esophageal Doppler measurements of stroke volume [30-34].

Our study carried a good correlation between intra-abdominal pressure and its impact on passive leg raising maneuver in a large scale of critically ill postoperative mechanically ventilated patients.

CONCLUSION

Our study came to the conclusion that the passive leg raising procedure did not predict fluid response in critically sick surgical, mechanically ventilated patients with an intra-abdominal pressure of $\geq$ 12 mmHg.

Passive leg raising must be done with extreme caution and attention since critical care units frequently have high intra-abdominal pressure values. Therefore, measuring IAP in critically unwell patients is advised.

Supporting and sponsoring financially: Nil.
Competing interests: Nil.

REFERENCES

1. **Task Force of the American College of Critical Care Medicine, Society of Critical Care Medicine (1999):** Practice parameters for hemodynamic support of sepsis in adult patients in sepsis. Crit. Care Med., 27:639-660.
2. **Monnet X, Rienzo M, Osman D *et al.* (2006):** Passive leg raising predicts fluid responsiveness in the critically ill. Crit. Care Med., 34: 1402-1407.
3. **Lafanechere A, Pene F, Goulenok C *et al.* (2006):** Changes in aortic blood flow induced by passive leg raising predict fluid responsiveness in critically ill patients. Critical Care, 10: 132. doi: 10.1186/cc5044.
4. **Takata M, Wise R, Robotham J (1990):** Effects of abdominal pressure on venous return: Abdominal vascular zone conditions. J. Appl. Physiol., 69:1961-1972.
5. **Vivier E, Metton O, Piriou V *et al.* (2006):** Effects of increased intra-abdominal pressure on central

circulation. Br J Anaest., 96: 701-707

6. **Alfonsi P, Vieillard-Baron A, Coggia M *et al.* (2006):** Cardiac function during intraperitoneal CO2 insufflation for aortic surgery: A transesophageal echocardiographic study. Anesth. Analg., 102:1304-1310.
7. **Jabot J, Teboul J, Richard C *et al.* (2009):** Passive leg raising for predicting fluid responsiveness: Importance of the postural change. Intensive Care Med., 35:85-90.
8. **Malbrain M, Chiumello D, Pelosi P *et al.* (2005):** Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: A multiple-center epidemiological study. Crit. Care Med., 33: 315-322.
9. **Derichard A, Robin E, Tavernier B *et al.* (2009):** Automated pulse pressure and stroke volume variations from radial artery': Evaluation during major abdominal surgery. J Anaesth., 103:678-84.
10. **Cheatham M, Safcsak K (1998):** Intraabdominal pressure: A revised method for measurement. J. Am. Coll. Surg., 186:594-595.
11. **Malbrain M, Cheatham M, Kirkpatrick A *et al.* (2006):** Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. Intensive Care Med., 32:1722-1732.
12. **Vallée F, Fourcade O, De Sovres O *et al.* (2005):** Stroke output variations calculated by esophageal Doppler is a reliable predictor of fluid response. Intensive Care Med., 31: 1388-1393.
13. **Caille V, Jabot J, Belliard G *et al.* (2008):** Hemodynamic effects of passive leg raising: An echocardiographic study in patients with shock. Intensive Care Med., 34:1329-1245.
14. **Maizel J, Airapetian N, Lome E *et al.* (2007):** Diagnosis of central hypovolemia by using passive leg raising. Intensive Care Med., 33: 1133-1138.
15. **Lamia B, Ochagavia A, Monnet X *et al.* (2007):** Eclio-cardiographic prediction of volume responsiveness in critically ill patients with spontaneously breathing activity. Intensive Care Med., 33:1125-1132
16. **Kitano Y, Takata M, Sasaki N *et al.* (1999):** Influence of increased abdominal pressure on steady- state cardiac performance. J. Appl. Physiol., 86:1651-1656.
17. **Kashtan J, Green J, Parsons E *et al.* (1981):** Hemodynamic effect of increased abdominal pressure. J. Surg. Res., 30:249-255.
18. **Robotham J, Wise R, Bromberger-Barnea B (1985):** Effects of changes in abdominal pressure on left ventricular performance and regional blood flow. Crit. Care Med., 13:803-809.
19. **Renner J, Gruenewald M, Quaden R *et al.* (2009):** Influence of increased intra-abdominal pressure on fluid responsiveness predicted by pulse pressure variation and stroke volume variation in a porcine model. Crit. Care Med., 37:650-658.
20. **Valenza F, Chevallard G, Porro G *et al.* (2007):** Static and dynamic components of esophageal and central venous pressure during intra-abdominal hypertension. Crz.Y. Care Med., 35: 1575-1581.
21. **Duperret S, Lhuillier F, Piriou V *et al.* (2007):** Increased intra-abdominal pressure affects respiratory variations in arterial pressure in normovolaemic and hypovolaemic mechanically ventilated healthy pigs. Intensive Care AW., 33:163-171.
22. **Büachenene E, Machado S, Fonseca E *et al.* (2007):** Pulse pressure variation as a tool to detect hypovolaemia during pneumoperitoneum. Acta Anaesthesiol Scand., 51: 1268-1272.
23. **Tournadre J, Allaouchiche R, Cayrel V *et al.* (2000):** Estimation of cardiac preload changes by systolic pressure variation in pigs undergoing Pneumoperitoneum. Atca Anaesthesiol Scand., 44:231-235.
24. **Malbrain M, Chiumdlo D, Pelosi P *et al.* (2004):** Prevalence of intra-abdominal hypertension in critically ill patients: A multicentre epidemiological study. Intensive Care Med., 30:822-829.
25. **Michard E, Boussat S, Chemla D *et al.* (2000):** Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory' failure. J. Respir. Crit. Care Med., 162: 134-138.
26. **Marik P, Cavallazzi R, Vasu T *et al.* (2009):** Dy-namic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: A systematic review of the literature. Crit. Care Med., 37: 2642-2647.
27. **Mahjoub Y, Pila C, Friggeri A *et al.* (2009):** Assessing fluid responsiveness in critically ill patients: False-positive pulse pressure variation is detected by Doppler echocardiographic evaluation of the right ventricle. Crit. Care Med., 37:2570-2575.
28. **Malbrain M, de Laet I (2009):** Functional hemodynamics and increased intra-abdominal pressure: Same thresholds for differ conditions ..? Crit. Care Med., 37(2):781-3.
29. **Boulain T, Achard J, Teboul J *et al.* (2002):** Changes in BP induced by passive leg raising predict response to fluid loading in critically ill patients. Chest, 121:1245-1252.
30. **Singer M, Clarke J, Bennett E (1989):** Continuous hemodynamic monitoring by esophageal Doppler. Crit. Care Med., 17:447-452.
31. **Valtier B, Cholley B, Belot J *et al.* (1998):** Noninvasive monitoring of cardiac output in critically ill patients using transesophageal Doppler. Am. J. Respir. Crit. Care Med., 158: 77-83.
32. **Perrino A, Fleming J, LaMantia K (1991):** Transesophageal Doppler cardiac output monitoring: Performance during aortic reconstructive surgery. Anesth. Analg., 73: 705-710.
33. **Schmid E, Spahn D, Tornic M (1993):** Reliability of a new generation transesophageal Doppler device for cardiac output monitoring. Anesth. Analg., 77:971-979.
34. **Keyl C, Rodig G, Lemberger P *et al.* (1996):** A comparison of the use of transoesophageal Doppler and thermodilution techniques for cardiac output determination. Eur. J. Anesthesiol., 13:136-142.