의학석사 학위 논문

Respiratory Variations in Electrocardiographic R-wave Amplitude during Hypovolemia by Inferior Vena Cava Clamping in Patients undergoing Liver Transplantation

간 이식 수술 중 하대정맥 결찰 후 발생하는

저혈량증에서 심전도 R-파 진폭의 호흡성 변화

울산대학교 대학원

의학과

박희선

Respiratory Variations in Electrocardiographic R-wave Amplitude during Hypovolemia by Inferior Vena Cava Clamping in Patients undergoing Liver Transplantation

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이 논문을 의학석사 학위 논문으로 제출함

2017년 12월

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Abstract

Introduction

The change of intraventricular volume could reflect electrocardiographic (ECG) R-wave amplitude and cardiopulmonary interaction induced by mechanical ventilation may generate respiratory variation in R-wave amplitude**.** The aim of this study was to examine whether the respiratory variation in ECG lead II R-wave amplitude(RDII) as dynamic index could predict intravascular volume status following inferior vena cava (IVC) clamping which induce to acute decrease in cardiac output and stroke volume undergoing liver transplantation (LT).

Materials and methods

We retrospectively investigated RDII before and after IVC clamping in 35 LT recipients. RDII was compared with other hemodynamic parameter from arterial waveform pressure analysis including cardiac output (CO_{FT}), cardiac index (CI_{FT}), stroke volume variation (SVV), pulse pressure variation (PPV), and stroke volume (SV_{FT}), and those from Swan-Ganz catheter including stat mode of continuous cardiac output and cardiac index (CCO_{stat}, CCI_{stat}), SV, stroke volume index

before and after IVC clamping. We also compared RDII and other hemodynamic parameters in low (≤0.05μg/kg/min) and high (>0.05μg/kg/min) norepinephrine groups. A receiver operating characteristic (ROC) curve analyses with area under the curve (AUC) was used to assess the cutoff value of RDII for predicting cardiac output decrease >25%.

Results

After IVC clamping, CO_{FT} and CCO_{stat} significantly decreased (P=0.005 and 0.004, respectively) while RDII significantly increased (P=0.005). The cutoff value and AUC of RDII predicting a decrease in CO_{FT} decrease > 25% were 38.9% and 0.867 with a specificity 70%, a sensitivity of 100% (95% confidence interval 0.706 – 0.958, P< 0.0001). RDII showed low sensitivity and specificity to predict CCO_{stat} decrease > 25% (AUC 0.532, 95% confidence interval 0.348 – 0.710, P=0.806). Furthermore, RDII changed significantly at low norepinephrine group, but not at high dose of norepinephrine (P=0.027 and 0.860 respectively).

Conclusion

We demonstrated that RDII could predict changes in cardiac output and have the possibility to be a

noninvasive dynamic parameter in patients with hemodynamic fluctuation.

Keywords: respiratory variation**,** electrocardiographic variation, R-wave amplitude

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Introduction

Dynamic preload indices are useful methods of fluid management for patients who are undergoing surgery or treated in intensive care units (ICU). Respiratory variations in stroke volume (stroke volume variation, SVV)[1], arterial pulse pressure (pulse pressure variation, PPV)[2] are well known dynamic indices that reflect intraoperative volume status and fluid responsiveness. To acquire these values, invasive arterial catheterization and specific devices are needed. However not every patient in operating room or ICU may need an arterial line and the invasive monitoring may not be always available.

Electrocardiogram (ECG), as noninvasive monitoring, can routinely be applied at bedside in operating room and ICU. Since "Brody effect" which QRS amplitude changed according to volume status of left ventricle presented in 1956[3], several literatures suggested that ECG morphology may reflect cardiac preload[4]. Like SVV or PPV, heart-lung interaction induced by mechanical ventilation will generate respiratory variation in ECG lead II R-wave amplitude (RDII) which may be used in the assessment of intravascular volume status noninvasively.

In liver transplantation surgery, inferior vena cava clamping causes to decrease venous return and cardiac output. There is no report that the ability of RDII to predict a decrease in cardiac output following IVC clamping induced acute central hypovolemia. We therefore examined to validate RDII reflect the change of cardiac output or stroke volume.

Materials and methods

This retrospective study involved thirty-five patients who underwent elective liver transplantation surgery at Asan Medical Center from June 2016 to January 2017. We excluded patients with arrhythmia which could not measure exact R wave such as atrial fibrillation, atrial flutter and left bundle branch block.

General anesthesia was performed in a standardized manner of our institution. Anesthesia was induced with thiopental, midazolam, fentanyl and esmeron and was maintained with the use of 4-5 vol% desflurane, 50% oxygen/air, and continuous infusion with fentanyl and esmeron. Mechanical ventilation was performed without positive end-expiratory pressure, using a constant tidal volume of 6-8 ml/kg and a constant end-tidal carbon dioxide tension of 30-35 mmHg.

Five-lead electrocardiography was applied and invasive radial arterial pressure was measured. The arterial line was connected to the Vigileo-FloTrac^{TM} system (Edwards Lifescience, Irvine, CA, USA) and the transducer was leveled at the mid-axillary line. We obtained cardiac output (CO_{FT}), cardiac index (CI_{FT}), stroke volume (SV_{FT}) as indicator for fluid status and stroke volume variation

(SVV) through arterial pulse wave analysis. A 7.5F pulmonary arterial catheter (PAC) (Swan-Ganz CCOmbo CCO/SvO2/CEDV, Edwards Lifesciences, Irvine, Calif, United States), which was inserted via a 9F introducer sheath into the internal jugular vein, was advanced to a wedged position under the guidance of a pressure curve. The pulmonary artery catheter was connected to a Vigilance monitor (Edwards Lifesciences) which provides stat mode in which the measurements is updated every 54s. The stat mode provided continuous cardiac output (CCO_{stat}), as well as for stat continuous cardiac index (CCI_{stat}). Stroke volume (SV), stroke volume index (SVI), central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) were also recorded.

The beat-to beat data were continuously recorded with a 1000-Hertz sampling rate, beginning 1 minute before the induction of anesthesia and continuing until the end of surgery. The collection of data was conducted with the use of Windaq software (DATAQ Instruments, Akron, OH, USA), after which the data were stored in a database of the operating room. Vital sign data were selected for approximately 10 minutes back and forth around IVC clamping (Figure 1) and these data were reviewed by visual inspection. The segments containing signal loss, or noise were discarded.

Windaq files were transferred to data analysis software LabChart®6 pro (version6.1.3, ADInstruments, Colorado Springs, CO, USA) to analyze beat-to beat data. Then lead II R-wave amplitude was measured from the automatic method and the accuracy was checked via manual measurement. Corresponding period of radial arterial pressure was chosen for analysis of pulse pressure variation. Pulse pressure variation (PPV) was calculated manually as described by its authors[2] :

Pulse pressure (PP) variation = (PPmax-PPmin)/ [(PPmax +PPmin)/2], where PPmax and PPmin are the maximal and minimal PP which were determined over the same respiratory cycle. Like PPV calculation, Respiratory RDII values were recorded, analyzed offline, and calculated as follows[5]:

RDII (%) = 100 x (maximum R- minimum R)/ [(maximum R + minimum R)/2]

All measurement and calculation were repeated on three consecutive respiratory cycles and averaged for statistical analysis.

These hemodynamic measurements were repeated on three consecutive respiratory cycles, and averaged for statistical analyses.

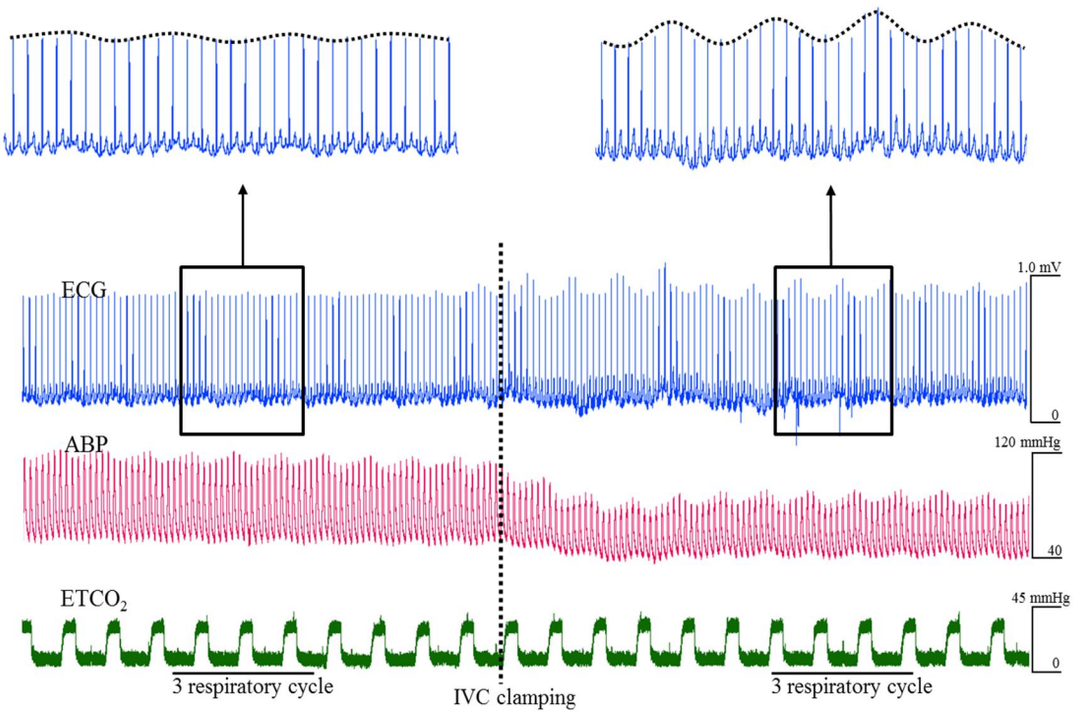

Fig 1. After inferior vena cava(IVC) clamping, arterial blood pressure decreased sharply. We selected the period of 3 consecutive respiratory cycles before and after IVC clamping and measured respiratory variations in electrocardiographic lead II R-wave amplitude.

Statistical Analysis

Demographics and hemodynamic data are expressed as mean and standard deviation or number (percentage). Based on the preliminary study of respiratory of lead II R amplitude (n=15), the sample size was determined to detect a projected difference of 10% (23 ms) in PTT before and after pressure decrease with an SD of 30 ms (a type I error of 0.05 and a power of 0.9). It was calculated that 33 patients were required. Expecting a dropout rate, we aimed at enrolling 35 patients. All data are presented as means ± SDs, unless otherwise indicated.

The paired t test or the signed-rank test was used for comparisons between the hemodynamic values before and after IVC clamping, and the t test or the rank-sum test was used for comparisons between groups in each individual phases. Receiver operator characteristic (ROC) curve were calculated for predicting decrease of CO_{FT} or CCO_{stat} > 25%**.** The area under the curve (AUC) values of ROC curve also was calculated. All data analyses were performed with the use of SPSS version 22 (SPSS Inc, Chicago, Ill, United States), and MedCalc version 13.1.1 (MedCalc Software, Mariakerke, Belgium). A value of $P < 0.05$ was considered statistically significant.

Results

The baseline demographic data and intraoperative norepinephrine use are listed in Table 1. The changes of hemodynamic variable and RDII before and after IVC clamping are listed in Table 2. RDII, SVV and PPV were significantly increased after IVC clamping (Table 2 and figure2). CO_{FT}, CI_{FT}, and SV_{FT} derived from arterial pressure waveform analysis were significantly decreased after IVC clamping. CCO_{stat}, CCI_{stat} and SV obtained from Swan-Ganz catheter showed also similar results.

Receiver operating characteristics curve analysis showed that RDII and other parameter to predict CO_{FT} and CCO_{stat} decrease > 25% (Table 3 and Figure 3). RDII cutoff value of 38.9% accurately predicted CO_{FT} decrease > 25%, with a specificity 70%, a sensitivity of 100%. On the other hand, RDII cutoff value of 84.2% predicted CCOstat decrease >25%, with a specificity 21.9%, a sensitivity of 100%. Another ROC analysis assessing the ability of RDII to predict PPV or SVV increase > 25% and rest of variables decrease > 25% is presented in Table 4.

At that time of IVC clamping, most patients received continuous norepinephrine infusion. The

patients were divided into two groups according to whether the continuous norepinephrine infusion dose > 0.5 μg/kg/min or not (Table 5). RDII, SVV and PPV changed significantly when low-dose norepinephrine were used, but not during a high dose of norepinephrine.

Table 1. Demographic and clinical characteristics of the 35 patients studied

Characteristics	
Patient characteristics and comorbidities	
Sex, Male (%)	24 (65.7%)
Age (years)	52.8 ± 9.3
Weight (kg)	75.3 ± 33.4
Height (cm)	155.4 ± 32.3
Cardiovascular disease[a]	4 (11.4%)
Hypertension	5 (14.2%)
Diabetes mellitus	12 (34.3%)
Child-Pugh score	8.4 ± 2.3
MELD[b] score	15.5 ± 6.9
Disease	
Viral hepatitis-related ESLD[c]	21 (60%)
Alcoholic cirrhosis	6 (17.1%)
Others	8 (22.9%)
Echocardiographic findings	
Left ventricle mass index (g/㎡)	89.4 ± 20.2
Left ventricular ejection fraction (%)	65.0 ± 5.4
Left ventricle internal dimension at end diastole (mm)	50.8 ± 8.1
Left ventricular posterior wall thickness at end diastole (mm)	8.6 ± 1.1
Interventricular septal dimension at end diastole (mm)	8.4 ±1.8
E/E' ratio[d]	9.0 ± 2.4
Intraoperative Norepinephrine use (%)	31 (88.6%)

Data are expressed as the mean ± standard deviation or number (percentage). [a] Including prior myocardial infarction, angina pectoris, left ventricular dysfunction, and peripheral arterial occlusive disease. [b] MELD : Model for End-Stage Liver Disease [c] ESLD: end stage of liver disease. [d] Ratio of early transmitral flow velocity to early diastolic velocity of the mitral annulus

Table 2. Comparison of RDII and other variables during IVC clamping (n=35)

	Before	**After**	**P value**
	mean ± SD	mean ± SD	
MBP (mmHg)	82.4 ± 10.1	79.0 ± 9.0	0.085
HR (bpm)	84.5 ± 12.6	87.5 ± 14.9	0.006
CVP (mmHg)	11.6 ± 3.4	10.7 ± 3.7	0.197
PCWP (mmHg)	19.9 ± 5.0	16.3 ± 4.5	0.001
Variables from arterial pressure waveform analysis†			
SVV (%)	6.8 ± 3.6	9.3 ± 5.2	0.002
CO_{FT} (L/min)	6.4 ± 1.7	5.8 ± 1.7	0.005
CI_{FT} (L/min/m^2)	3.7 ± 0.8	3.4 ± 0.8	0.003
SV_{FT} (mL/beat)	76.6 ± 24.3	69.7 ± 26.3	0.014
Variables from Swan-Ganz catheter			
CCO_{stat}‡(L/min)	7 ± 2.0	6.2 ± 2.1	0.004
CCI_{stat}‡(L/min/m^2)	4.1 ± 1.0	3.6 ± 1.0	0.002
SV*(mL/beat)	86.1 ± 27.1	77.4 ± 28.7	< 0.001
SVI* (mL/beat/m^2)	48.2 ± 15.5	44.7 ± 13.3	0.058
PPV (%)	6.9 ± 4.4	11.3 ± 5.5	< 0.001
RDII (%)	11.4 ± 6.1	14.3 ± 8.6	0.005

†n=34, ‡n= 32 (before), 34(after), *n=33, MAP, mean arterial pressure; HR, heart rate; CVP, central venous pressure; PCWP, pulmonary wedge pressure; SVV, stroke volume variation; CO, cardiac output; CI, cardiac index; SV, stroke volume; CCO, continuous cardiac output; CCI, continuous cardiac index; SVI, stroke volume index; PPV, pulse pressure variation; RDII: respiratory variation of lead II R-wave amplitude

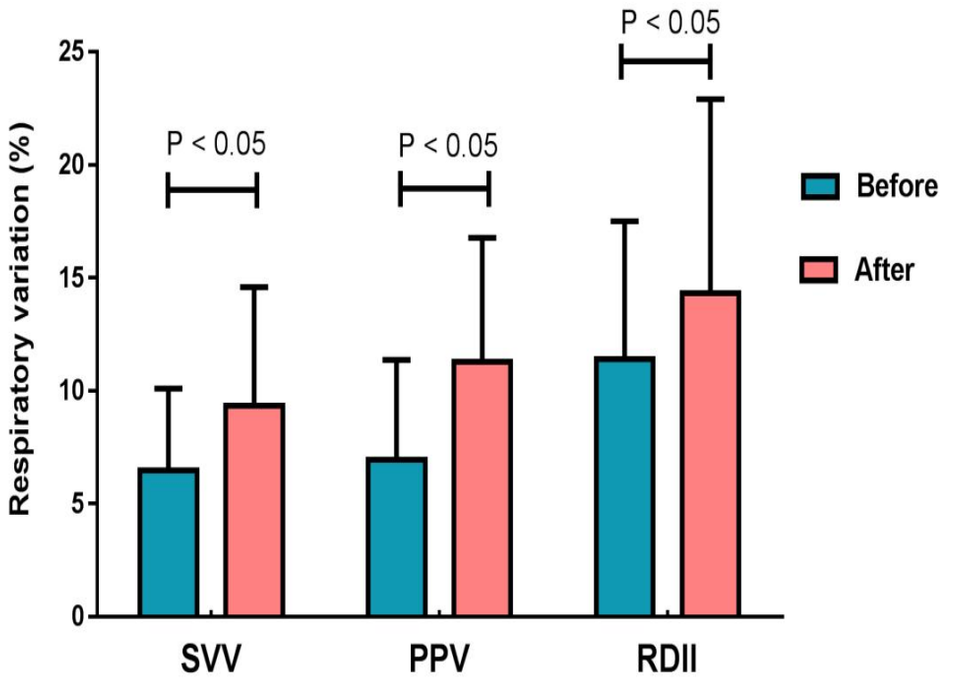

Fig 2. The change of variation of R-wave amplitude in lead II (RDII), stroke volume variation (SVV) or pulse pressure variation (PPV) before and after inferior vena cava clamping.

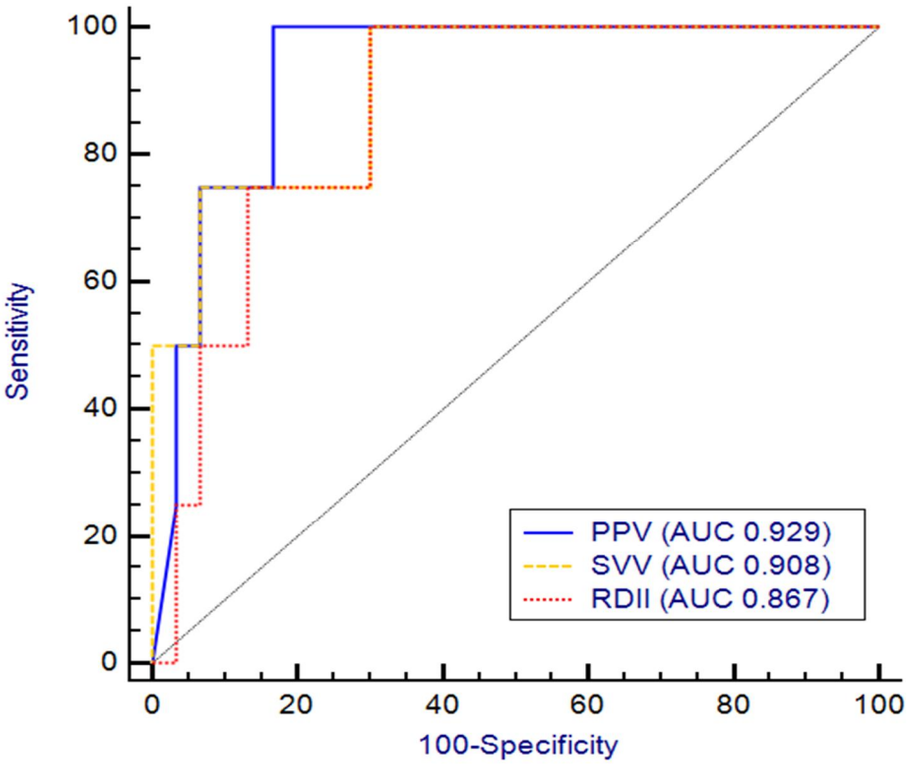

Fig 3. Ability of variation of R-wave amplitude in lead II (RDII), stroke volume variation (SVV) or pulse pressure variation (PPV) to predict a cardiac output decrease> 25% (receiver operating characteristics curves).

Table 3. Receiver operating characteristic curve analysis assessing the abilities parameter to predict cardiac output 25% decrease

		Cutoff	Sensitivity (%)	Specificity (%)	AUC	Standard error	95% CI	Z statistic	P value
CO from arterial pressure waveform analysis									
ΔCO_{FT}	RDII	38.9%	100	70	0.867	0.075	0.706 – 0.958	4.884	<0.0001
	SVV	62.7%	100	70	0.908	0.076	0.759 - 0.980	5.335	<0.0001
	PPV	109%	100	83	0.929	0.048	0.787 - 0.989	8.875	<0.0001
	CVP	-25.3%	75	77	0.650	0.162	0.468 - 0.805	0.924	0.356
	PCWP	-36.7%	75	90	0.742	0.176	0.563 - 0.876	1.376	0.169
CO from Swan-Ganz catheter									
ΔCCO_{stat}	RDII	84.2%	100	22	0.532	0.130	0.348 - 0.710	0.246	0.806
	SVV	19.3%	98.5	50	0.731	0.097	0.545 - 0.871	2.368	0.018
	PPV	36.9%	83.3	42	0.574	0.151	0.387 - 0.746	0.490	0.624
	CVP	16.3%	50	92	0.513	0.199	0.331 - 0.693	0.065	0.949
	PCWP	-53.9%	33.3	96	0.609	0.143	0.421 - 0.776	0.760	0.448

AUC: area under the curve, SE: standard error, CI: confidence interval, SVV, stroke volume variation; CO, cardiac output; CI, cardiac index; SV, stroke volume; CCO, continuous cardiac output; CCI, continuous cardiac index; SVI, stroke volume index; PPV, pulse pressure variation; RDII: respiratory variation of lead II R-wave amplitude

Table 4. ROC curve analysis assessing the abilities R amplitude variation (RDII) predicting other variables of 25% change

	Cutoff	Sensitivity (%)	Specificity (%)	AUC	Standard error	95% CI	Z statistic	P value
Variables from arterial pressure waveform								
CO_{FT} 25%	38.9	100	70	0.867	0.075	0.706 - 0.958	4.884	<0.0001
CI_{FT} 25%	38.9	100	70	0.867	0.075	0.706 - 0.958	4.884	<0.0001
SV_{FT} 25%	38.9	83.3	71.4	0.774	0.094	0.598 - 0.899	2.924	0.004
SVV 25%	-8.1	83.3	50	0.656	0.098	0.474 - 0.810	1.596	0.111
PPV 25%	20.3	58.3	81.8	0.701	0.109	0.523 - 0.843	1.596	0.111
Variables from Swan-Ganz catheter								
CCO_{stat} 25%	84.2	100	21.9	0.532	0.130	0.348 - 0.710	0.246	0.806
CCI_{stat} 25%	38.9	66.7	69.6	0.623	0.116	0.435 - 0.788	1.060	0.289
SV 25%	-0.1	83.3	40.7	0.494	0.129	0.316 - 0.673	-0.048	0.962
SVI 25%	-15.9	100	21.4	0.543	0.145	0.361 - 0.717	0.295	0.768

AUC: area under the curve, SE: standard error, CI: confidence interval, SVV, stroke volume variation; CO, cardiac output; CI, cardiac index; SV, stroke volume; CCO, continuous cardiac output; CCI, continuous cardiac index; SVI, stroke volume index; PPV, pulse pressure variation; RDII: respiratory variation of lead II R-wave amplitude

Table 5. Comparisons of ΔRII and other variables according to the dose of continuous norepinephrine infusion during IVC clamping

	Low dose ≤0.05 μg/kg/min or none) (n=17)			High dose (>0.05 μg/kg/min) (n=18)		
	Before	After	P value	Before	After	P value
Variables from arterial pressure waveform analysis†						
SVV (%)	5.3 ± 3.4	7.3 ± 3.9	0.045	7.6 ± 3.6	11.4 ± 5.7	0.210
CO_{FT} (L/min)	6.3 ± 1.2	5.8 ± 1.8	0.084	6.4 ± 1.8	5.8 ± 1.6	0.028
CI_{FT} (L/min/m^2)	3.7 ± 0.8	3.4 ± 0.8	0.082	3.7 ± 0.9	3.4 ± 0.9	0.015
SV_{FT} (mL/beat)	90.1 ± 28.5	82.0 ± 29.5	0.101	76.5 ± 26.3	69.2 ± 29.3	0.077
Variables from Swan-Ganz catheter						
CCO_{stat}‡ $(L/min)_t$	7.2 ± 2.4	6.5 ± 2.5	0.118	6.8 ± 1.6	5.9 ± 1.6	0.007
CCI_{stat}‡ (L/min/m^2)	4.2 ± 1.2	3.8 ± 1.1	0.057	3.9 ± 0.8	3.5 ± 0.8	0.012
SV* (mL/beat)	90.1 ± 28.5	82.0 ± 29.5	0.020	82.3 ± 26.0	72.8 ± 27.1	0.007
SVI* (mL/beat/m^2)	51.9 ± 13.8	47.3 ± 13.1	0.020	44.8 ± 16.6	42.2 ± 13.5	0.428
PPV	5.6 ± 3.2	10.3 ± 4.9	<0.0001	8.3 ± 5.1	12.2 ± 6.0	0.013
RDII	10.5 ± 5.4	13.9 ± 7.5	0.027	12.1 ± 6.8	14.8 ± 9.8	0.860

†n=34, ‡n= 32 (before), 34(after), *n=33†

Discussion

The major findings of this study are that RDII can predict the change of CO_{FT} by IVC clamping during liver transplantation and this index may not be sufficient to predict a decrease in CCO_{stat}. The idea of the use of RDII as dynamic index is based on "Brody effect" and cardio-pulmonary interaction on ECG induced by positive pressure ventilation. The "Brody effect" said that an increased blood volume in ventricular cavity induces an increase in R-wave amplitude. It is due to the electrical inhomogeneity remote from the heart and its effect on the transmission of myocardium depolarization to the body surface. In patients with mechanical ventilation, positive-pressure inspiration makes cyclic changes in vena-cava blood, pulmonary artery and aorta blood flow. As a result, right ventricular stroke volume decreases during inspiratory phase and these changes lead to decrease in left ventricular output only a after two or three beats[6]. There was a literature that a preload-dependent patient in mechanical ventilation support showed which respiratory variations in ECG R-wave amplitude reflect LV preload dependent variables[4]. These heart-lung interaction and cyclic change to arterial pulse pressure (PPV) and stroke volume(SVV)

can be used to assess fluid responsiveness over three or more breathes[7].

Actually RDII have correlation with SVV or PPV that RDII is a reliable parameter to estimate the changes in intravascular volume status in preload dependent status[5,8,9]. One of these studies conducted in pig with hemorrhage, while other reports performed in human who did not rapidly change in their intravascular volume. We used the period of IVC clamping during liver transplantation for this study. Occlusion of the vena cava can lead to reduce venous return with subsequent decrease in cardiac output and eventually result in preload-dependent status. At this central hypovolemic status, CO_{FT}, CI_{FT} and SV_{FT} decreased approximately 9% while RDII, SVV and PPV significantly increased 39%, 73% and 115% respectively. In contrast, CVP, one of the static parameter, did not significantly change. The magnitude of changes in SVV and PPV were greater than in RDII (Table 2, Figure 2). These findings were consistent with a previous literature by Giraud et al[8] which carried out an experiment that made hypovolemic and normovolemic state induced by hemorrhage and re-transfusion of blood to pig. In that study, the magnitude of change in PPV was greater than those of RDII. They explained that the reason is supposed to be due to the

arterial elastance and catecholamine effect on PPV and SVV[8,10].

However, there is a report that RDII showed lower sensitivity than PPV to detect changes in low volume (approximately 300 ml) of mobilization in stroke volume[11]. Soltner et al[11] reported RDII could detect of preload dependent patients when a change of stroke volume greater than 12% is achieved. Because preload-independent heart will not experience large respiratory variations in cardiac volume and, consequently, the R wave amplitude, will not show large variations[5,9,12]. Which is the reason the authors used a cutoff value of 25%.

Our ROC analysis indicated RDII is superior to CVP and PCWP to predict the cardiac output change. Another implication of present study is that RDII can predict the cardiac output decrease >25% (AUC = 0. 867) similar to other variables (SVV and PPV). Unfortunately RDII could not predict variables from Swan-Ganz catheter. Although we selected stat mode of CCO and CCI data, the time delays in the response of the pulmonary artery catheter was difficult to reflect rapid alterations of the hemodynamic state[13] .

One of confounding factors of this study may be the use of vasopressor agents. The characteristics

of patient undergoing liver transplantation with hyper-dynamic circulation, as well as blood loss and massive intravascular fluid shifts, need a continuous use of vasopressor infusion[14]. Such agents can affect arterial tone and right ventricular function. Unfortunately most patients (31/35) in this study received norepinephrine infusion at that time of IVC clamping. Thus we divided all patients into two groups according to whether the continuous norepinephrine infusion dose> 0.5 μg/kg/min or not. Then comparing each variables, RDII, SVV and PPV varied significantly when low-dose norepinephrine were used, but not during a high dose of norepinephrine. These discrepancy can be attributed to the administration of catecholamine affected PPV and SVV both of which were reduced[15,16]. On the other hand, RDII is theoretically not directly affected by the administration of catecholamine. The use of norepinephrine may mask real intravascular volume deficit so RDII could not show significant change in a high dose of agent. Furthermore IVC clamping generally induces severe hemodynamic instability, the anesthesiologists will do rapid fluid infusion such as crystalloid, colloid or blood transfusion in order to replace the intravascular volume. Patients with high dose of norepinephrine would have been in more hemodynamic instability status, so such

interventions above mentioned would have been more massive. These factors would have contributed to the inconsistent of the results in our study.

There are some technical problems in using ECG as a hemodynamic parameter. The ECG baseline wandering or noises can disturb the exact measurement of R amplitude. Furthermore manual measurement of ECG may not be accurate. If technical problems could be solved and automated software could be developed, it would be a useful noninvasive method to predict intravascular volume status and fluid responsiveness. There was a trial to compare an automated method using Matlab (Matlab R2011a, MathWorks Inc., Natick, MA, USA) with a manual determination of R-wave amplitude[12].

In conclusion, RDII could predict the change of cardiac output during IVC clamping in liver transplantation. Our results also confirmed the "Brody effect". Although there are still limitations of clinical application, ECG can be a reliable parameter to assess cardiac output and intravascular volume status as a noninvasive monitoring method and will also contribute to fluid management in operating room.

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국문 요약

연구 목적

중환자 치료와 전신 마취 중 환자 관리에 있어 적절한 심박출량 유지를 위하여 환자의 체액 상태를 정확히 파악하고 그에 따라 올바른 수액치료를 유지하는 것은 매우 중요하다. 이러한 환자의 체액 상태를 평가하기 위한 동적 지표로 동맥압의 변이 (pulse pressure variation, 이하 PPV)나 1회 심박출량 변이 (stroke volume variation, 이하 SVV)가 흔히 사용된다. 그러나 이 지표들은 동맥관 삽입이라는 침습적인 시술을 해야하고, 이를 분석하기 위한 별도의 장치도 필요하지만 실제로 모든 수술 환자에서 동맥관 거치를 필요로 하지 않는다. 반면 심전도는 비침습적이며 마취 중이나 중환자실에서 필수적으로 시행하는 감시장치이다. 1956년 Brody가 발표한 논문에 따르면 심전도 R파형의 진폭은 심실내 혈액 부피와 직접적인 연관이 있음이 알려져 있다. 따라서 본 연구에서는 간이식 수술 중 하대정맥을 결찰하여 혈관 용적이 감소 했을 때, 기계호흡에 따른 R파 진폭의 변화가 심박출량 변화를 예측할 수 있는지를 알아보고자 한다.

연구재료와 연구방법

생체 간이식을 받는 환자를 대상으로 하여, 수술 중 하대정맥 결찰 전후의 호흡에 따른 R파 진폭 변이(Respiratory variation in electrocardiographic lead II R wave amplitude, 이하 RDII)와 PPV, SVV, 심박출량 및 일회 심박출량 등을 포함한 변수들을 비교 및 분석한다.

연구결과

하대정맥 결찰 후, 동맥압 분석 및 Swan-Ganz 카테터를 통해 측정한 심박출량은 모두 유의하게 감소한 반면 (p=0.005, 0.004), RDII는 유의하게 증가하였다 (P=0.005). RDII의 동맥압 분석을 통한 심박출량 예측에 대한 수신자 조작 특성곡선 (Receiver Operating Characteristic Curve, ROC Curve)은 39% 이상의 RDII가 심박출량의 25% 감소를 특이도 70%, 민감도 100%로 예측할 수 있음을 보여주었다 (AUC=0.867, 95% 신뢰구간 0.706–0.958, p<0.0001). 그러나 RDII의 Swan-Ganz 카테터를 통한 심박출량 변화 예측에 대해서는 낮은 특이도 및 민감도를 보였다 (AUC=0.532, 95% 신뢰구간 0.348–0.710, P=0.806). 또한 하대정맥 결찰 시에 노르에피네프린 군을 용량에 따라 나누어서 비교

해보았다. 그 결과 저용량 노르에피네프린 (<0.05mcg/kg/min)을 사용한 군에서는 RDII 가 유의하게 증가하였지만, 고용량 (>0.05mcg/kg/min)군에서는 그렇지 못했다 (각각 p=0.027, 0.860).

고찰 및 결론

본 연구를 통하여, 수술 중 하대정맥 결찰로 인하여 저혈량증이 발생했을 때 RDII는 PPV, SVV 와 같이 증가하였고 이는 심박출량의 변화를 예측할 수 있음을 알 수 있었다. 그러나 본 연구는 간이식 수술 중 하대정맥 결찰이라는 과역동학적인 혈역학적 상황에서 진행되었고 노르에피네프린을 사용하거나 수액의 과도한 주입 및 수혈 등의 변수들이 연구 결과에 영향을 주었을 가능성이 있다. 또한 심전도 분석을 하는데 잡음과 신호 처리 방법 등의 기술적인 제한점이 있었다. 그러나 심전도 파형 분석을 이용한 비침습적인 동적 지표를 이용하여 환자의 체액 상태를 평가하고 적절한 수액 관리의 지표로 활용될 수 있는 가능성을 보여주었다. 앞으로 보다 다양한 상황에서의 R 파형 분석을 통하여 임상적 유용성에 대한 정확히 평가하고 이를 통해 실제 상황에서 활용하기 위한 추가적인 연구가 필요하겠다.

중심단어: 호흡변화, 심전도 변화, 심전도 R 파