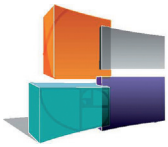
Anaesth Crit Care Pain Med 38 (2019) 147–152



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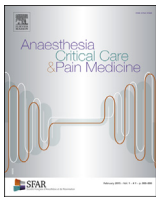

Original Article

Effect of fluid challenge on renal resistive index after major orthopaedic surgery: A prospective observational study using Doppler ultrasonography

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ARTICLE INFO

Article history:
Available online 21 April 2018

Keywords:
Renal resistive index
Fluid challenge
Acute kidney injury

ABSTRACT

Background: A postoperative renal resistive index (RRI) > 0.70 has the best threshold to early predict acute kidney injury (AKI). The response of RRI to a postoperative fluid challenge (FC) is unknown. The aim of our study was to assess the impact of a FC on RRI in suspected hypovolaemia patients after orthopaedic surgery.
Design: In this single-centre observational study, we prospectively screened 156 patients in the recovery room after having undergone a hip or knee replacement.
Interventions: Forty-six patients with a RRI > 0.70 and requiring FC were included. RRI and cardiac output (CO) were measured before and immediately after a fluid challenge with 500 mL of isotonic saline. A decrease in RRI > 5% was considered significant (renal responders).
Results: Overall, FC resulted in a consistent decrease in RRI (from 0.74 [0.72–0.79] to 0.70 [0.68–0.73], $P < 0.01$). Thirty-four patients (74%) showed a significant decrease in their RRI (from 0.74 [0.73–0.79] to 0.69 [0.67–0.72], $P < 0.05$, versus non-responders: from 0.73 [0.72–0.75] to 0.72 [0.71–0.79], P = NS). CO increased equally among renal responders and non-responders ($P = 0.56$). No correlation was found between changes in RRI and CO ($r^2 = 0.04$; $P = 0.064$). AKI was more common in renal non-responders (7/12) than in responders (3/34, $P = 0.001$).
Conclusions: After major orthopaedic surgery, a FC can decrease RRI in suspected hypovolaemia patients at risk of postoperative AKI, but the changes are not correlated to changes in CO. Decreases in RRI were associated with better renal outcome.

1. Introduction

Postoperative acute kidney injury (AKI) has been shown to be associated with significant morbidity and mortality [1,2]. For example regarding long-term risk for cardiovascular events, AKI is associated with an elevated risk of major events, particularly heart failure and acute myocardial infarction [3]. These findings highlight the need for screening and management of patients with postoperative AKI. However, its early diagnosis still remains challenging in daily practice insofar as serum creatinine (Cr) rise occurs after renal damage. Some studies suggest that measuring renal resistive index (RRI) in the post anaesthesia care unit (PACU) could predict the development of AKI after cardiac and orthopaedic surgery [4–6]. Postoperative RRI (i.e. performed in the post anaesthesia care unit) takes into account not only the preexisting condition but also changes induced during the intraoperative phase. It has been shown that in a population at risk of AKI, a RRI > 0.70 had a good sensitivity and specificity (0.94 and 0.71 respectively) with an ROC curve area of 0.86 to predict the development of AKI 1 or 2 days before the rise of Cr [5]. In critically ill patients, changes in renal haemodynamic observed by Doppler ultrasonography during a fluid challenge (FC) are controversial. While some authors found that FC was not effective in improving (i.e. decreasing) RRI in patients with septic shock [7], a decrease of this last can be observed before urine output increases in adult patients with acute circulatory failure [8]. To our knowledge, the effects of a FC on Doppler-based RRI have never been studied in the perioperative period.

The primary objective of this study was to determine if a FC in suspected hypovolaemia patients would be able to decrease RRI

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https://doi.org/10.1016/j.accpm.2018.04.008
2352-5568/

after major orthopaedic surgery. Secondary objectives were to assess if changes in RRI would be correlated with changes in cardiac output (CO) and be associated with a better renal outcome.

2. Materials and methods

This prospective observational study was approved by the local research ethics board (Comité d'Éthique Recherche, CHU Toulouse, Chairperson Dr Jean-Marie Conil, registration number 29-0512, July 2011) and carried out according to the declaration of Helsinki. All patients gave oral informed consent.

2.1. Patients

All patients undergoing total hip arthroplasty (primary or revision) or total knee arthroplasty (primary or revision) and scheduled for a fluid challenge (FC) in the PACU because suspected hypovolaemia were eligible for inclusion. The decision to administer a FC was ordered by the physician in charge and was based on the presence of at least one clinical sign of inadequate tissue perfusion as well as the absence of congestive heart failure. Clinical signs of inadequate tissue perfusion were defined as follows: systolic arterial pressure (SAP) $\leq$ 90 mmHg (or a decrease of at least 40 mmHg from the preoperative baseline in patients with a history of hypertension); mean arterial pressure (MAP) $\leq$ 65 mmHg; urine output $\leq$ 0.5 mL/kg per hour for at least 1 hour; tachycardia (heart rate $>$ 100/min); symptoms of inadequate tissue perfusion such as prolonged capillary refill $>$ 3 seconds or mottled skin; arterial lactate level $>$ 2 mmmol/L. Cardiac arrhythmias, agitation or confusion, tachypnoea (RR $>$ 35/min) or respiratory failure and poor echogenicity were considered exclusion criteria. A visual analogue pain scale $<$ 3/10 and an oxygen saturation $>$ 96% (if necessary with oxygen therapy) were needed before RRI measurements [5].

2.2. Ultrasound measurement

The RRI measurements were preceded by a formal two hours training session (30 minutes of theoretical instruction and 1 hour and 30 minutes of practical teaching at the bedside) with a trained radiologist. All echo measurements were made with a HD11XE ultrasound system (Philips Medical System; Bothell, WA) by the two authors (P.M. and C.F.) using a 5–1 MHz pulsed-wave Doppler probe (''cardiac'' probe). The kidney was visualised with 2D echo. The interlobar arteries (adjacent to medullary pyramids) located by colour Doppler were then insonated using a 2 to 4 mm pulsed Doppler gate [9]. Waveforms were optimised for measurement using the lowest pulse repetition frequency without aliasing (to maximize waveform size), and the highest gain without obscuring background noise. Three to five reproducible waveforms from one of the two kidneys (right kidney in most of the patients) were obtained, and RRIs from these waveforms were calculated as RRI = (peak systolic velocity – end diastolic velocity)/peak systolic velocity. RRIs were then averaged to arrive at mean RRI values [10–12]. The intra-observer and inter-observer variabilities for the RRI measurements have been calculated as part of a previous study (2.1% $\pm$ 2.6 and 2.9% $\pm$ 2.7 respectively) [5].

The CO was measured using transthoracic echocardiography (TTE). All measurements were performed at end expiration as previously described [13,14].

2.3. Study design

All suspected hypovolaemia patients scheduled for a FC in the PACU were screened, regardless of their risk for AKI. Afterward patients with a RRI $\leq$ 0.70 were excluded from the study (please see flow diagram in Fig. 1).

The investigators who measured RRI were blinded to the patient characteristics and the change in CO. An intravenous bolus of 500 mL of isotonic saline 0.9% was then administered within 15 minutes [7].

The following data were recorded for each patient: age, gender, diabetes, arterial hypertension, arteriosclerosis, chronic heart failure and preoperative serum creatinine (Cr). Intraoperative data that were recorded included the type and length of surgery, type of anaesthesia, volume and type of iv. fluid and amount of intraoperative blood loss. RRI and CO measurements were performed in the PACU before and immediately after FC. Two distinctive groups were analysed according to the change in RRI after FC: renal responders and non-responders. A renal responder was defined as having a decrease of at least 5% of the RRI (corresponding to approximately twice the intra-observer and inter-observer variabilities) [5].

As part of the routine practice at our institution, the Cr value was measured before the operation (baseline) and then daily each morning, until a peak Cr value was reached. Urine output was measured every 6 hours for 48 hours. AKI was defined according to the Acute Kidney Injury Network classification either as an increase in Cr value by $\geq$ 26.4 μmol/L (0.3 mg/dL) or an increase to $\geq$ 150% from baseline or as a urine output of less than 0.5 mL/kg/hr for 6 hours or more.

2.4. Sample size projection

A literature search did not reveal a previous study evaluating the response of RRI in suspected hypovolaemia patients after major orthopaedic surgery. Taking into account previous findings [5], a minimum sample size was estimated as follows. Assuming a RRI $>$ 0.70 in the selected patient population and a standard deviation of 0.05, and using a test with 0.05 type I error and 0.1 type II error, at least 24 patients with a renal response were needed to detect a 5% decrease in RRI after fluid challenge. Estimating that half of the selected patients would have a renal response after fluid challenge (a search did not reveal a previous study evaluating the rate of patients with a renal response to fluid challenge in this setting), at least 48 patients with RRI $>$ 0.70 were thus needed to detect a 5% decrease in RRI after fluid challenge. Assuming that half of the hypovolemic patients would have a RRI $>$ 0.70 in the PACU, at least 96 patients were thus needed for RRI measurement. Estimating that about 30% of the patients would have exclusion criteria, at least 138 hypovolemic patients in PACU would need to be screened for inclusion.

2.5. Statistical analysis

Data distribution was verified using a Kolmogorov–Smirnov test. Continuous variables are presented as median [interquartile range, IQR] or mean $\pm$ standard deviation (SD) as appropriate. Qualitative data were given as number and percentage. The non-parametric Mann–Whitney U test was used for the comparison of continuous variables between groups (renal responders versus non-responders) and the Wilcoxon rank–sum test was used to compare continuous variables obtained before and after the FC. Categorical variables were compared with the Fisher's exact test or Chi^2 test. The correlation between relative changes of RRI and variations of CO was estimated using linear regression. To test the abilities of ΔRRI (i.e. change from before to after fluid challenge, %) and RRI after fluid challenge to predict AKI, areas under the ROC curves [area under the curve (AUC) = 0.5: no better than chance, no prediction possible; AUC = 1.0: best possible prediction] were calculated and compared using the Hanley–McNeil test. Statistical analysis was carried out

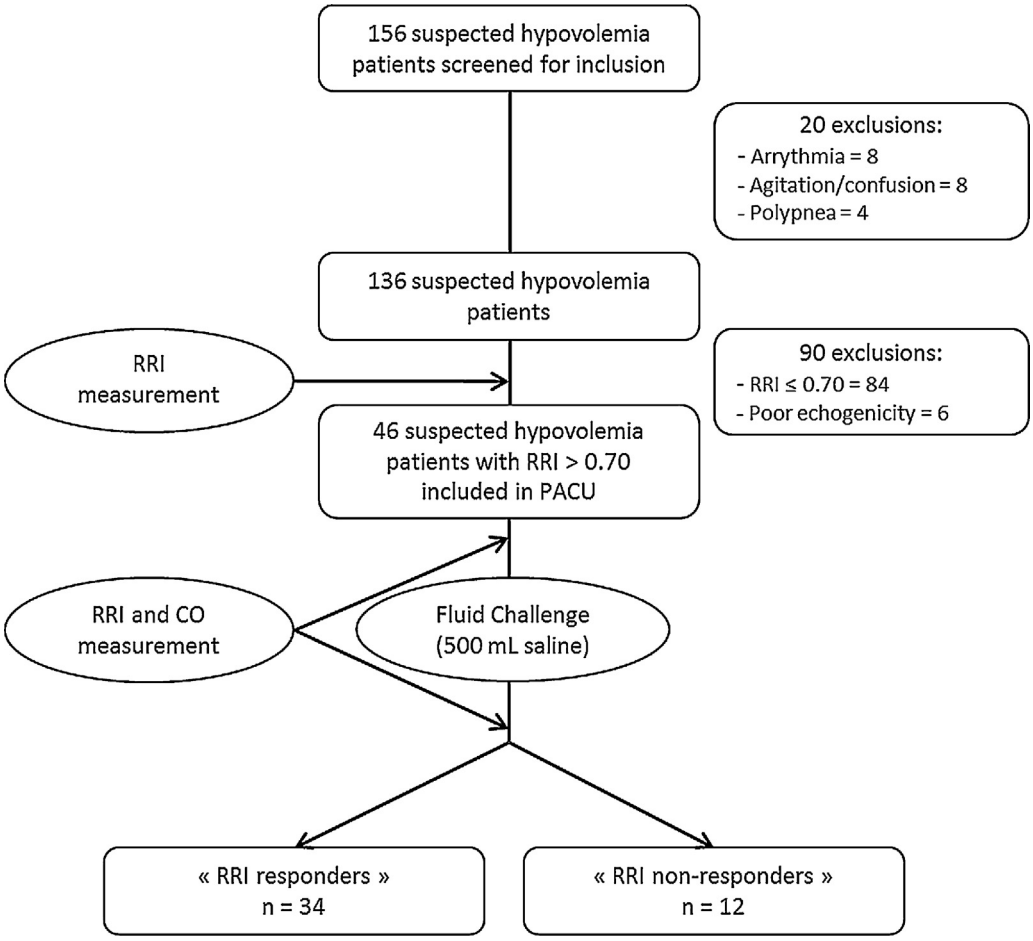

Fig. 1. Flow Chart. RRI: renal resistive index; CO: cardiac output; PACU: post anaesthesia care unit.

using MedCalc Statistical Software, version 12.6.1 (MedCalc Software bvba, Ostend, Belgium; 2013). A p-value of less than 0.05 was considered statistically significant.

3. Results

3.1. Patient characteristics

The study design and the patient flow diagram are shown in Fig. 1. One hundred and fifty six patients undergoing FC in PACU after total hip or total knee arthroplasty were screened for inclusion. Eight patients were excluded because of arrhythmias, 8 for confusion/agitation and 4 for tachypnoea. A renal Doppler was then performed in the remaining 136 patients. Of those, 6 patients were excluded because no good image could be obtained and 84 were excluded for a RRI $\leq$ 0.70. Thus, a total of 46 patients fulfilled criteria for inclusion and completed the study.

3.2. Primary objective

Overall, fluid administration resulted in a consistent decrease in RRI: from 0.74 [0.72–0.79] before fluid challenge to 0.70 [0.68–0.73] after fluid challenge ($P < 0.01$) (Fig. 2). The ΔRRI (i.e. change from before to after fluid challenge, %) was −6.8 [−8 to −3.8].

By focusing on the two subgroups of patients (separated according to their change in RRI after FC), RRI decreased among 34 patients (74%) after the FC (renal responders, $P < 0.05$). The renal responders and non-responders were comparable in terms of demographic data (Table 1) and intraoperative characteristics (Table 2).

3.3. Secondary objective

The FC resulted in a significant stroke volume (SV) increase and heart rate decrease in renal responders and non-responders (Table 3) without changing the mean arterial pressure.

No significant difference was noticed for RRI before FC between renal responders and non-responders (0.74 [0.73–0.79] vs. 0.73 [0.72–0.75]; $P = 0.32$). After FC, RRI was lower in renal responders compared to non-responders (0.69 [0.67–0.72] vs. 0.72 [0.71–0.79]; $P < 0.001$) (Fig. 3). No correlation was found between RRI variation and CO variation induced by FC ($r^2 = 0.04$; $P = 0.064$).

Ten subjects went on to showed signs of AKI. AKI was more common in non-responders compared to responders ($n = 7$, 58% vs. $n = 3$, 9%; $P = 0.001$), but none of the patients suffered permanent kidney injury, or needed dialysis. Using the AKIN classification, 8 patients were classified as AKIN stage 1 and 2 patients were classified as AKIN stage 2. RRI after fluid challenge and ΔRRI detected postoperative AKI with similar mean areas under the ROC curves: 0.817 for RRI after fluid challenge (95% confidence interval (CI) between 0.675 and 0.915, $P < 0.0001$) and 0.790 for ΔRRI (95% CI between 0.645 and 0.896, $P = 0.003$), $P = 0.8$ for the pairwise comparison of ROC curves (Fig. 4). The optimal threshold value given by ROC analysis was 0.703 for RRI after fluid challenge (90% sensitivity, 66.7% specificity, 2.7 LR+ and 0.15 LR−) and −3.8% for ΔRRI (70% sensitivity, 88.9% specificity,

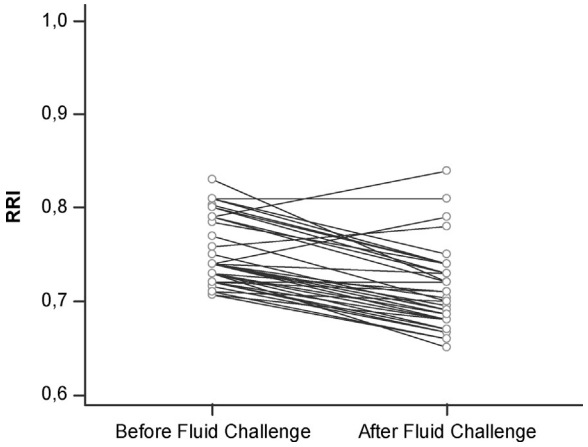

Fig. 2. RRI variations induced by Fluid Challenge in the overall population of patients with suspected hypovolemia and a high RRI ($P < 0.01$ when compared to before fluid challenge).

Table 1
Demographic characteristics.

	RRI responders $n = 34$	RRI non-responders $n = 12$	P
Sex			
Female	14 (41)	4 (33)	0.63
Male	20 (59)	8 (67)	
Age (years)	66 [63.7–77.5]	77.5 [67–82]	0.74
BMI (kg/m^2)	24.8 [22.8–27.1]	25.9 [24.1–29.5]	0.16
Diabetes	11 (32)	2 (17)	0.29
Arterial hypertension	11 (32)	8 (67)	0.9
Arteriosclerosis	13 (38)	4 (33)	0.76
Chronic cardiac failure	5 (15)	2 (17)	1.0
Smoking	11 (32)	3 (25)	0.63
Preoperative serum creatinine (μmol/L)	78 [55–95]	70 [60–83]	0.24
Preoperative GFR (mL/min/1.73m^2)	85 [60–94]	78 [63–84]	0.32
Chronic kidney disease (CKD-EPI) Stage 1/2/3A/3B/4/5	0/2/3/2/0/0	0/1/3/2/1/0	0.1

RRI: renal resistive index; BMI: body mass index; GFR: glomerular filtration rate; Creatinine clearance was defined according to the CKD-EPI formula. Data are expressed as median [IQR] or n (%).

6.3 LR+ and 0.34 LR−). The Cr peaked in all subjects within the first four postoperative days.

4. Discussion

This study is the first to assess the effect of a FC on RRI in suspected hypovolaemia patients with a high RRI after hip and knee replacement and suggests that a FC could decrease RRI in 74% of those patients and be associated with a decreased incidence of AKI.

It has been shown that measuring RRI could allow early detection of patients at risk of developing AKI [6] in contrast to the serum Cr and urine output which are relatively late indicators of AKI. This early detection may thus allow timely interventions such as haemodynamic optimisation or avoidance of medications with potential nephrotoxicity. The role of haemodynamic optimisation (i.e. a FC) in improving (i.e. decreasing) RRI and ultimately preventing the development of AKI is still a matter of debate. A previous study found that a FC was not effective in decreasing RRI in patients with septic shock [7] and it has been postulated that this finding may be due to the particular pathophysiology linked to sepsis (i.e. the renal resistance primarily reflecting local microcirculatory changes mediated by renal endothelium) [15]. Investigators have already tested other strategies to decrease RRI in septic patients for example the use of catecholamine infusions [16].

It is surprising that the RRI decrease in our study was not correlated with the changes in CO. In other words, the evolution of the cardiac output after fluid challenge does not allow us to predict the evolution of the RRI. This point underlines the complexity of the regulation of the renal perfusion. For example, in the setting of circulatory failure, the increase in CO induced by a FC is probably not uniformly distributed among the peripheral circulation. In addition, there may not be a direct relationship between RRI and renal perfusion and the relationships between the various factors that affect the Doppler-derived renal arterial waveform (i.e. vascular compliance, vascular resistance, and heart rate) and renal perfusion are still being explored [12,17]. Previous studies have shown that RRI is a reliable marker of renal arteriosclerosis cause by essential hypertension. In this setting, RRI appears to be strongly associated with creatinine clearance and it increases in patients with hypertensive end-organ damage [18]. The renal impact of fixed impairment of vascular compliance in hypertensive patients may help to explain why known hypertension was 2 times higher in our renal non-responders compared to renal responders patients (although this difference did not reach statistical significance potentially because of small sample size).

The decrease of RRI after fluid resuscitation was associated with a significant better renal outcome in this study. The determination and use of RRI could therefore serve as an important bedside parameter to allow implementation of different therapeutic strategies to improve renal outcome. However, none of the patients suffered from permanent kidney injury, or needed dialysis in our study and the clinical relevance of measuring RRI in our study population remains therefore uncertain. It will be necessary to develop, test and validate different treatment algorithms based on RRI in larger clinical trials that could take into account preexisting risk factors for the development of postoperative AKI.

Table 2
Intraoperative characteristics.

	RRI responders $n = 34$	RRI non-responders $n = 12$	P
Primary total hip arthroplasty	8 (23)	2 (17)	0.24
Revision total hip arthroplasty	16 (47)	8 (66)	0.08
Primary total knee arthroplasty	4 (12)	0	0.23
Revision total knee arthroplasty	6 (18)	2 (17)	0.15
Type of anaesthesia			
General anaesthesia	24 (70)	8 (67)	1.0
Spinal anaesthesia	10 (29)	4 (33)	
Length of anaesthesia (min)	150 [120–180]	180 [150–190]	0.32
Intraoperative blood loss (mL)	400 [400–600]	500 [350–625]	0.71
Intraoperative fluid administration (mL)	1500 [1375–1500]	1250 [1000–1500]	0.21

RRI: renal resistive index. Data are expressed as median [IQR] or n (%).

Table 3
Postoperative characteristics.

	RRI responders $n=34$	RRI non-responders $n=12$	P
MAP (mmHg)			
Before FC	81 [72–89]	79 [71–86]	0.82
After FC	84 [74–89]	78 [71–103]	0.32
HR (bpm)			
Before FC	85 [75–96]	83 [72–88]	0.86
After FC	72 [68–86]*	70 [67–78]*	0.96
SV (mL)			
Before FC	60 [48–66]	61 [52–71]	0.69
After FC	65 [56–72]*	71 [59–84]*	0.18
ΔSV (%)	11 [1.6–16]	14.3 [1–29.5]	0.23
RRI			
Before FC	0.74 [0.73–0.79]	0.73 [0.72–0.75]	0.32
After FC	0.69 [0.67–0.72]*	0.72 [0.71–0.79]	< 0.001
ΔRRI (%)	−8 [−9 to −7]	0 [−2.1–4.6]	< 0.001
Postoperative AKI	3 (9)	7 (58)	0.001

RRI: renal resistive index; MAP: mean arterial pressure; FC: fluid challenge; HR: heart rate; bpm: beat per minute; SV: stroke volume; RRI: renal resistive index; AKI: acute kidney injury; Δ: change from before to after fluid challenge. Data are expressed as median [IQR] or n (%).

* $P < 0.05$ when compared to before fluid challenge (within the group).

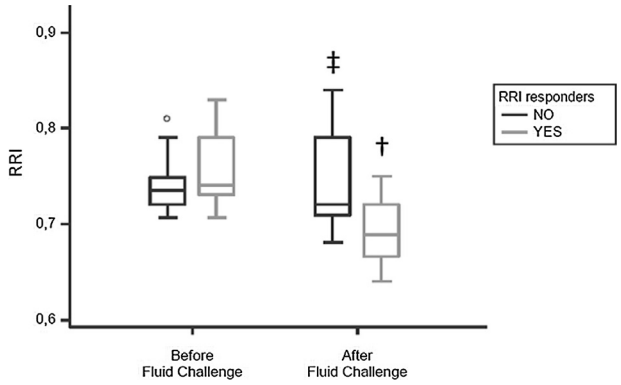

Fig. 3. RRI variations induced by Fluid Challenge. Box plot of RRI variations induced by fluid challenge. Median values shown as solid line within box of 25 and 75th percentile values. Whiskers represent range values. Single daggers mean different from before (within the group, $P < 0.05$). Double daggers mean different from RRI responders ($P < 0.01$). RRI: renal resistive index.

This study has some limitations. One of them is the monocentric design. Since ultrasonography is known to be operator dependent, these results have to be confirmed with other operators in other institutions. Second, among the 156 patients screened in this study, only 46 fulfilled criteria for inclusion and the observed results might therefore not be generalisable to all hypovolemic patients after knee and hip replacement. Indeed, the response of RRI to a fluid challenge in patients with a baseline postoperative RRI ≤ 0.70 remains unknown. Moreover, excluding patients with RRI ≤ 0.70 is highly questionable as they could account for nearly two-thirds of patients. Third, the ΔRRI chosen to distinguish renal responders and non-responders is debatable. Ultrasound techniques have potentially high variability, especially in non-trained operators. For example, the variability of values in Doppler echocardiography usually ranges from 5 to 10%. In this setting, results could have been different with another threshold value of ΔRRI. Fourth, one of the main limitations of the study is the lack of a clearly defined endpoint for fluid resuscitation (which was administered by the physician in charge of the patient). Indeed, we were not able to follow the patients with serial CO measurements to determine if the initial improvement in CO was indeed sustained. As such we cannot rule out that some of the patients had only a transient increase in CO (i.e. would have needed more than one fluid challenge) and that this may have impacted the lack of correlation between CO and RRI response. Finally, a large uncertainty remains regarding the use of this biomarker in clinical decision-making.

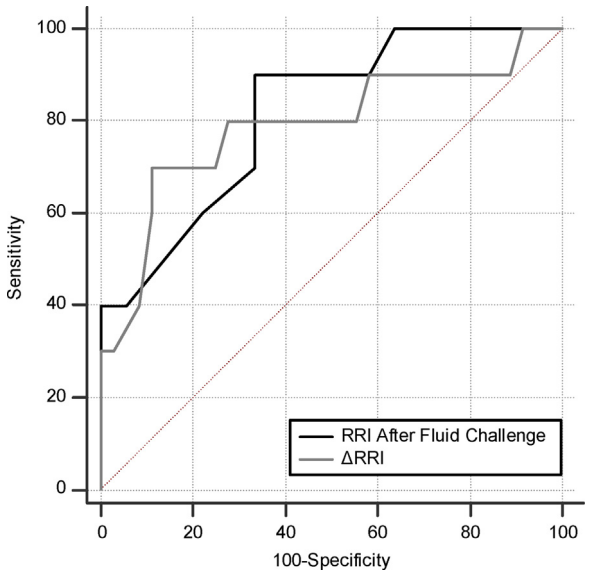

Fig. 4. ROC curves comparing the ability of ΔRRI (i.e. change from before to after fluid challenge) and RRI after fluid challenge to predict postoperative Acute Kidney Injury.

5. Conclusion

Our results show that in suspected preload-dependency patients with a high RRI in the recovery room after knee and hip replacement a fluid challenge can decrease RRI. This improvement when present was associated with a better renal outcome. Whether this better renal prognosis is related to fluid challenge itself remains unknown.

Funding sources

This work should be attributed to the Department of Anesthesiology and Intensive Care, Toulouse University Hospital, Toulouse, France. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Authors' contributions

PM and CF performed all measurements. PM and FF participated in the design of the study and wrote the manuscript. PM performed the statistical analysis. MK and VM participated in its design and coordination and helped to draft the manuscript. All authors read and approved the final manuscript.

Ethical statement

Written informed consent was obtained from all study patients according to the Declaration of Helsinki. The Methods section includes a statement that an IRB approved the recruitment of human subjects for this study.

All authors have read and approved the submission of this manuscript. The authors take full responsibility for the data, the analyses and interpretation, the conduct of the research and the right to publish any and all data.

The material in the manuscript has not been published and is not being considered for publication elsewhere, in whole or in part in any language.

Disclosure of interest

The authors declare that they have no competing interest.

Acknowledgements

None.

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