Postoperative Behavior of Renal Functional Reserve in Patients Undergoing Cardiac Surgery

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Background

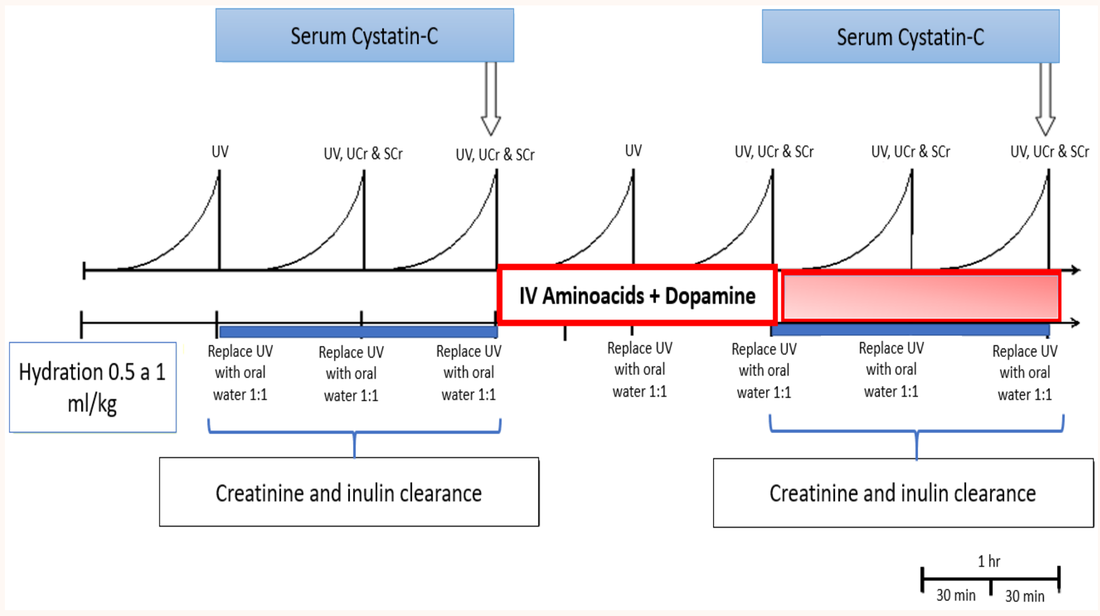
Renal Functional Reserve (RFR) evaluates the ability to increase Glomerular Filtration Rate (GFR) after a physiologic or pathologic stimulus. Recent publications have addressed the relation between baseline RFR and postoperative acute kidney injury (AKI).

Objectives

To describe the immediate postoperative behavior of RFR; and to evaluated potential biochemical surrogates for RFR.

Patients and methods

A prospective, longitudinal cohort study including adult patients with GFR between 30 and 90 ml/min undergoing elective cardiac valve surgery. GFR was assessed on day -1, +1 and +3 of cardiac surgery by inulin clearance before and after an intravenous infusion of amino acids and dopamine as a stimulus for renal reserve.

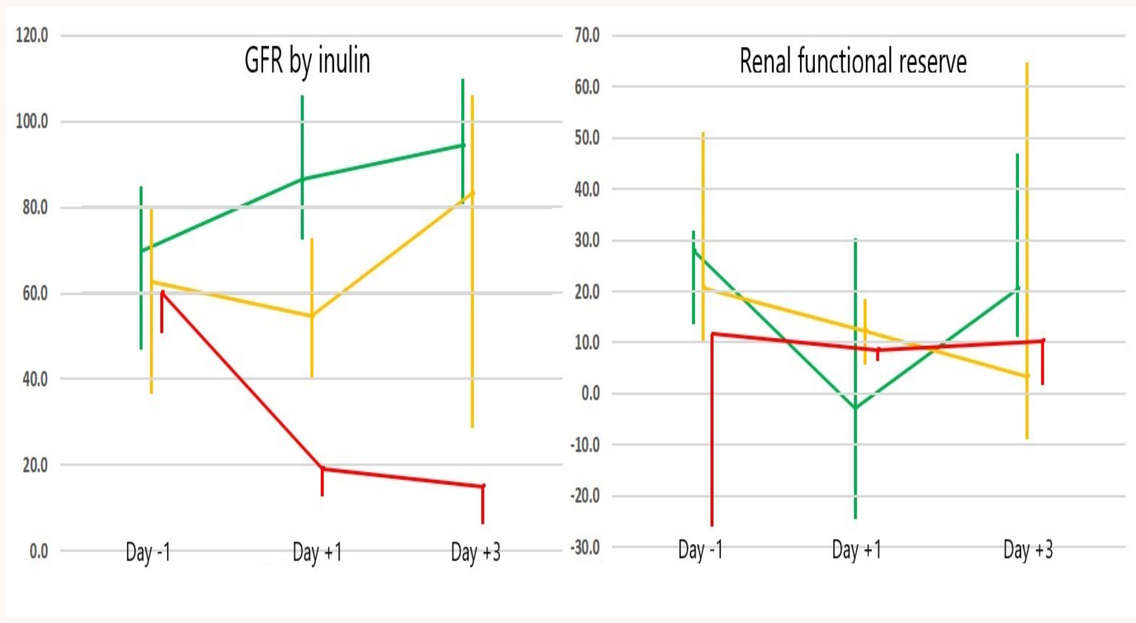


Results

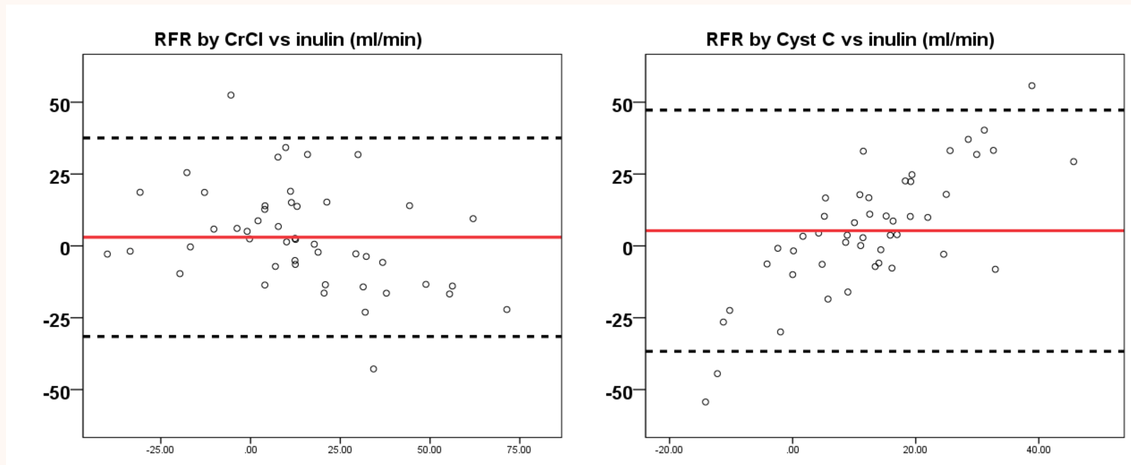
Eighteen patients were included. Seven (38.9%) developed AKI and only 2 of them had severe and persistent AKI. None required renal replacement therapy.

Variables	Total N=18	AKI n=7	Non AKI n=11	P
Female	9 (50)	1 (14.3)	8 (72.7)	0.025
Age	60 (46 - 64.5)	62 (39 - 66)	57 (47 - 61)	0.888
Body Surface	1.7 (1.5 - 1.7)	1.7 (1.7 - 1.8)	1.5 (1.4 - 1.7)	0.054
Diabetes	7 (38.9)	4 (57.1)	3 (27.3)	0.22
Hypertension	4 (22.2)	1 (14.3)	3 (27.3)	0.48
Aortic stenosis	7 (38.9)	3 (42.9)	4 (36.4)	0.58
Prior cardiac surgery	8 (44.4)	6 (85.7)	2 (18.2)	0.009
Heart failure NYHA III	9 (50)	4 (57.1)	5 (45.5)	0.5
EuroScore	3.3 (1 - 6.2)	7.7 (3.6 - 9.4)	2 (0.8 - 4.1)	0.001
Baseline SCr	0.8 (0.7 - 0.9)	0.9 (0.9 - 1)	0.8 (0.7 - 0.8)	0.004
Cystatin C	1.1 (0.9 - 1.2)	1.1 (0.9 - 1.2)	1 (0.8 - 1.2)	0.325
Reintervention	3 (16.7)	3 (42.9)	0 (0)	0.043
Death	1 (5.6)	1 (14.3)	0 (0)	0.38

In patients with no AKI, GFR is increased by using its RFR (green line). With transient AKI, RFR lead to no increment in postoperative GFR on day +1 (orange line). In patients with severe persistent AKI, a lower baseline RFR available showed a significant drop in GFR (red line).

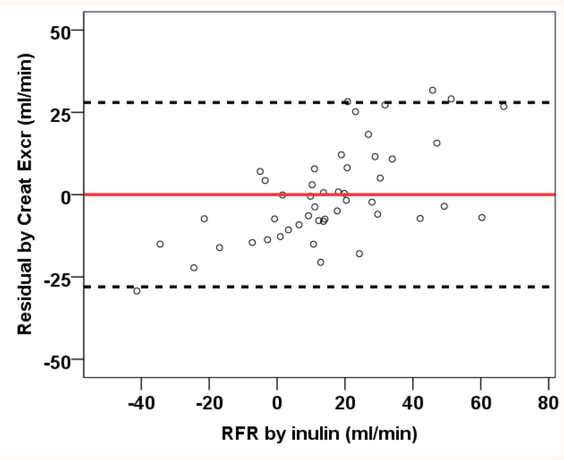


Rho coefficients in comparison to RFR by inulin were: 0.210 (p=0.131) for serum creatinine, 0.309 (p=0.024) for cystatin C, 0.574 (p<0.001) for short creatinine clearance (CrCl), and 0.525 (p<0.001) for short creatinine excretion.



Bland-Altman analysis showed a difference of 5.3 ml/min for RFR by Cyst C vs inulin, and 3.0 ml/min for RFR by CrCl vs inulin, but with width confidence intervals (-36.7 to 47.3 ml/min for Cyst C, and -31.5 to 37.6 ml/min for CrCl). However, creatinine excretion estimating RFR showed a deviation of 0.0 (-28.0 to 28.0) ml/min.

Regression analysis equation to estimate RFR by inulin was 11.8+5.6*CrExcr(mcg/kg/min); R^2 0.615, F 73.369, p <0.001.



Conclusions

Postoperative behavior suggests the use of RFR as a mechanism of compensation (kidney adaptation) in patients who do not develop clinical AKI. Short creatinine excretion measurement should be evaluated as a potential surrogate for changes in GFR including the estimation of RFR.

Acknowledgments:

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