

# Elevated Intra-Abdominal Pressure in Acute Decompensated Heart Failure

## A Potential Contributor to Worsening Renal Function?

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- Objectives** This study sought to determine whether changes in intra-abdominal pressure (IAP) with aggressive diuretic or vasodilator therapy are associated with improvement in renal function in acute decompensated heart failure (ADHF).
- Background** Elevated IAP ( $\geq 8$  mm Hg) is associated with intra-abdominal organ dysfunction. There is potential for ascites and visceral edema causing elevated IAP in patients with ADHF.
- Methods** Forty consecutive patients admitted to a specialized heart failure intensive care unit for management of ADHF with intensive medical therapy were studied. The IAP was measured using a simple transvesical technique at time of admission and before removal of the pulmonary artery catheter.
- Results** In our study cohort (mean age  $59 \pm 13$  years, mean left ventricular ejection fraction  $19 \pm 9\%$ , baseline serum creatinine  $2.0 \pm 0.9$  mg/dl), the mean baseline IAP was  $8 \pm 4$  mm Hg, with 24 (60%) patients having elevated IAP. Elevated IAP was associated with worse renal function ( $p = 0.009$ ). Intensive medical therapy resulted in improvement in both hemodynamic measurements and IAP. A strong correlation ( $r = 0.77$ ,  $p < 0.001$ ) was observed between reduction in IAP and improved renal function in patients with baseline elevated IAP. However, changes in IAP or renal function did not correlate with changes in any hemodynamic variable.
- Conclusions** Elevated IAP is prevalent in patients with ADHF and is associated with impaired renal function. In the setting of intensive medical therapy for ADHF, changes in IAP were better correlated with changes in renal function than any hemodynamic variable. (J Am Coll Cardiol 2008;51:300-6) © 2008 by the American College of Cardiology Foundation

Despite recent medical advances, the pathophysiology of acute decompensated heart failure (ADHF) remains poorly understood, particularly regarding the cardiorenal interactions. In many cases of heart failure, coexisting renal dysfunction may complicate the treatment course. In addition, therapies that alleviate congestion, such as loop diuretics, which remain a mainstay of the therapeutic armament against heart failure, can worsen renal insufficiency and may even increase mortality (1,2).

There has been increasing interest in measuring intra-abdominal pressure (IAP) in critically ill patients because elevated IAP has been associated with intra-abdominal organ dysfunction (3,4). The compliance of the abdominal

wall generally limits the increases in IAP as abdominal girth increases. However, once a critical volume is reached, compliance of the abdominal wall decreases abruptly. Further distention beyond this critical IAP results in a rapid increase in abdominal pressure and resultant organ dysfunction (5,6). Recently, during the second World Congress on Abdominal Compartment Syndrome, medical critical care specialists defined a normal IAP to be between 5 and 7 mm Hg in critically ill adults, an elevated IAP to be  $\geq 8$  mm Hg, and intra-abdominal hypertension (IAH) to be  $\geq 12$  mm Hg (7).

It has been recognized over the past century that elevated IAP can directly lead to renal compromise in the setting of abdominal compartment syndrome or other surgical conditions involving visceral edema (4-6). However, data regarding measurements of IAP in patients admitted with ADHF are lacking despite the potential for a substantial part of ADHF patients to present with ascites, visceral edema, and

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impaired renal function. The primary goal of our study was to test the hypothesis that IAP is commonly elevated in patients admitted with ADHF. The secondary goal was to investigate whether intensive medical therapy can reduce IAP and whether reduction in IAP may lead to corresponding improvement in renal function.

## Methods

**Patient population.** We prospectively enrolled consecutive patients ages 18 years or older with symptomatic heart failure (New York Heart Association functional class III to IV) who underwent a right heart catheterization and were admitted to the Cleveland Clinic heart failure intensive care unit for intensive medical therapy between November 1, 2006, and May 31, 2007. Subjects who met the following inclusion criteria were enrolled into the study: 1) markedly impaired systolic left ventricular function defined by left ventricular ejection fraction  $\leq 30\%$ , and 2) elevated filling pressures, as defined by pulmonary capillary wedge pressure (PCWP)  $\geq 18$  mm Hg and right atrial pressure  $\geq 8$  mm Hg. Exclusion criteria included: 1) patients on artificial ventilation; 2) patients who had undergone abdominal or thoracic surgery within the last 3 months; 3) patients without a Foley catheter; and 4) patients on renal replacement therapy. The Cleveland Clinic Institutional Review Board approved this project. Informed consent was obtained as part of the treatment and all invasive procedures during hospitalization, and was documented in the patient charts according to protocol and Cleveland Clinic policy.

**Drug titration protocols.** The hemodynamic goals and pharmacologic approach to intravenous therapy in the specialized heart failure intensive care unit have been previously described (8). Briefly, optimal hemodynamic response was defined as a decrease in PCWP to  $\leq 18$  mm Hg, decrease in central venous pressure (CVP) to  $\leq 8$  mm Hg, and improvement in cardiac index (CI) to  $\geq 2.2$  l/min/m<sup>2</sup>, all while maintaining mean arterial pressure (MAP)  $> 65$  mm Hg. To achieve the hemodynamic goals, most patients were treated according to standard protocols developed in our intensive care unit with intravenous loop diuretics in combination with vasodilators (i.e., nitroprusside) and/or inotropic agents while continuing previous therapies with angiotensin-converting enzyme inhibitors, beta-blockers, and spironolactone as tolerated. Loop diuretics were given as a continuous infusion with or without an initial bolus at the discretion of the attending physician.

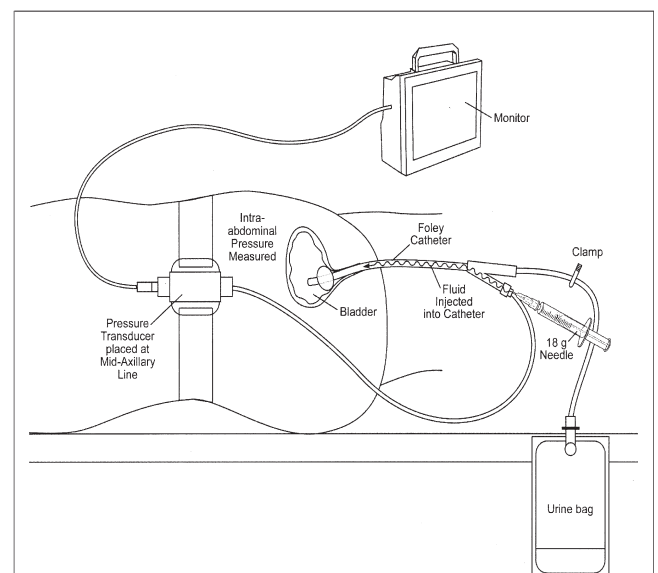
**IAP measurement.** Clinical examination of the abdomen and/or the abdominal perimeter is not an accurate indicator of IAP (9,10). To obtain a precise IAP value, the pressure is measured with the transvesical method (11,12). Briefly, IAP is measured via a standard Foley catheter, which is connected with a pressure transducer placed in-line with the iliac crest at the midaxillary line (Fig. 1). The Foley catheter is flushed with a maximal instillation volume of 50-ml sterile saline via the aspiration port of the Foley catheter

with the drainage tube clamped to allow a fluid-filled column to develop up into the bladder. Installation of more volume can lead to bladder distention, which can be uncomfortable to the patient and lead to increased intravesical pressure. This could thus give rise to a falsely high IAP measurement. A pressure transducer is then inserted in the aspiration port, and the pressure is measured. The IAP is expressed in mm Hg and is measured at end-expiration in the supine position, ensuring that abdominal muscle contractions are absent. In this study, the IAP is measured on admission before drug initiation and before removal of the pulmonary artery catheter (36  $\pm$  12 h later). The interobserver and intraobserver variabilities of IAP measurements were compared in 30 consecutive IAP measurements, and were found to be 5% and 4%, respectively.

**Data collection and variable definitions.** Data were collected by 3 experienced heart failure cardiologists. The following additional data were recorded: demographic characteristics, medical history, medical treatment, implanted pacemaker and implantable cardioverter defibrillator device information, and echocardiographic data. In all patients,

### Abbreviations and Acronyms

<b>ADHF</b> = acute decompensated heart failure
<b>CI</b> = cardiac index by Fick equation
<b>CVP</b> = central venous pressure
<b>FG</b> = renal filtration gradient
<b>GFR</b> = glomerular filtration rate
<b>IAH</b> = intra-abdominal hypertension
<b>IAP</b> = intra-abdominal pressure
<b>MAP</b> = mean arterial pressure
<b>PCWP</b> = pulmonary capillary wedge pressure



**Figure 1** Transvesical Method for Measuring Intra-Abdominal Pressure

Bedside technique using conventional Foley catheter filled with saline and connected to a hemodynamic monitor.

serum creatinine levels on admission and before removal of the pulmonary artery catheter were recorded. Estimated glomerular filtration rate (GFR) was calculated using the abbreviated Modification of Diet in Renal Disease study formula:  $GFR \text{ for men} = 186 \times (\text{serum creatinine } [\mu\text{mol/l}] \times 0.0113) - 1.154 \times \text{age (years)} - 0.203$ . For women the result was multiplied by 0.742 and for African Americans by 1.210.

**Hemodynamic assessment.** Complete hemodynamic assessment was performed in all patients before the start of intensive medical therapy, and again before removing the pulmonary artery catheter. The CVP and PCWP were assessed at end-expiration with a balloon-tipped catheter at steady state with the patient in a supine position. The CI was determined by calculation using the Fick equation through sampling of a mixed central venous blood gas measurement taken in the pulmonary artery while assuming standard metabolic rates. The MAP was calculated as  $(\text{systolic blood pressure} + 2 \times \text{diastolic blood pressure})/3$ . The abdominal perfusion pressure was determined by following equation:  $\text{MAP} - \text{IAP}$  (12). The renal filtration gradient (FG) is the mechanical force across the glomeruli and can be estimated as: glomerular filtration pressure minus proximal tubular pressure (12). In the presence of elevated IAP, proximal tubular pressure may be assumed to equal IAP, and thus glomerular filtration pressure can be estimated as:  $\text{MAP} - \text{IAP}$ . The FG was therefore calculated as:  $\text{MAP} - 2 \times \text{IAP}$ .

**Statistical analysis.** All data are expressed as mean  $\pm$  SD for continuous data and as percent ratio for categorical data. Univariate comparisons of these variables were performed between baseline and follow-up variables and between patients with normal versus elevated IAP ( $\geq 8$  mm Hg) using SPSS for Windows, release 11.5 (SPSS Inc., Chicago, Illinois). A paired and unpaired *t* test for continuous data and Pearson correlation coefficients were used for appropriate comparisons. Statistical significance was set at a two-tailed probability level of  $<0.05$ .

## Results

**Baseline characteristics and medical treatment.** A total of 40 patients met eligibility criteria and were enrolled in the study. Baseline characteristics and treatment during admission of the patients stratified according to IAP  $\geq 8$  mm Hg ( $n = 24$ ) and IAP  $< 8$  mm Hg ( $n = 16$ ) are summarized in Table 1. All patients were classified as New York Heart Association functional class III or IV. Mean left ventricular ejection fraction was similar between the 2 patient groups, as was the percentage of patients with moderate to severe right ventricular systolic dysfunction (38% in both groups). There were no statistically significant differences in medical therapy on admission or during intensive medical therapy between the 2 patient groups.

No patient complained of abdominal discomfort on admission or during treatment. The median length of

**Table 1** Baseline Patient Characteristics According to IAP  $\geq 8$  mm Hg and IAP  $< 8$  mm Hg

	Patients With IAP $\geq 8$ mm Hg (n = 24)	Patients With IAP $< 8$ mm Hg (n = 16)	p Value
<b>Demographics and vital statistics</b>			
Mean age (yrs)	58 $\pm$ 11	61 $\pm$ 14	NS
Men (%)	67	62	NS
Weight (kg)	94 $\pm$ 23	82 $\pm$ 17	NS
Height (cm)	177 $\pm$ 10	172 $\pm$ 7	NS
Hypertension (%)	70	47	0.04
Hyperlipidemia (%)	46	53	NS
Diabetes (%)	37	34	NS
Smoking (%)	30	20	0.05
Previous CABG (%)	35	33	NS
ICD/CRT-D (%)	63	60	NS
Idiopathic dilated (%)	62	68	NS
Ischemic (%)	38	32	NS
Ejection fraction (%)	19 $\pm$ 8	21 $\pm$ 12	NS
Hemoglobin (g/dl)	11 $\pm$ 2	11 $\pm$ 2	NS
<b>Medication on admission (%)</b>			
Beta-blockers	62	56	NS
ACE inhibitors/ARB	62	62	NS
Spirololactone	46	44	NS
Loop diuretic	96	100	NS
<b>Parenteral medication during admission (%)</b>			
Loop diuretics	82	86	NS
Nitroprusside	45	46	NS
Dobutamine	24	26	NS
Milrinone	31	27	NS

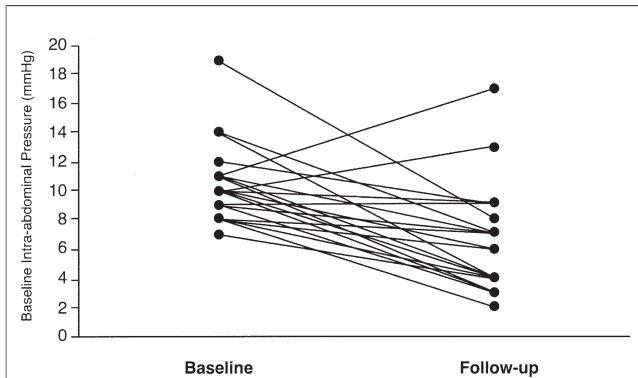
Values are mean  $\pm$  standard deviation or n (%).

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; CABG = coronary artery bypass graft; CRT-D = cardiac resynchronization therapy with defibrillator; ICD = implantable cardioverter-defibrillator.

treatment (from baseline to follow-up, defined as change in all measurements) was  $36 \pm 10$  h and  $36 \pm 14$  h in the patients with normal IAP and elevated IAP, respectively ( $p = 0.9$ ).

**IAP measurements.** In the overall cohort, the mean IAP at baseline was  $8 \pm 4$  mm Hg, which improved to a mean of  $5 \pm 3$  mm Hg after medical therapy ( $p < 0.001$ ). The mean IAP in the cohort of patients with elevated IAP ( $n = 24$ ) on admission was  $10 \pm 2$  mm Hg, which was also significantly reduced at follow-up to  $6 \pm 3$  mm Hg ( $p < 0.001$ ) (Fig. 2). Four (10%) patients presented with IAH on admission, and they too had a significant decrease in IAP at follow-up ( $15 \pm 3$  mm Hg to  $7 \pm 2$  mm Hg,  $p < 0.001$ ). Only 3 patients who presented with elevated IAP had an increase of IAP at follow-up. No urinary tract infection or abdominal discomfort was seen in any patient during the treatment period.

**Hemodynamic and renal variables at baseline and follow-up.** Table 2 illustrates the hemodynamic measurements on admission and after intensive medical therapy in all patients, as well as for the subgroup of patients with elevated IAP and

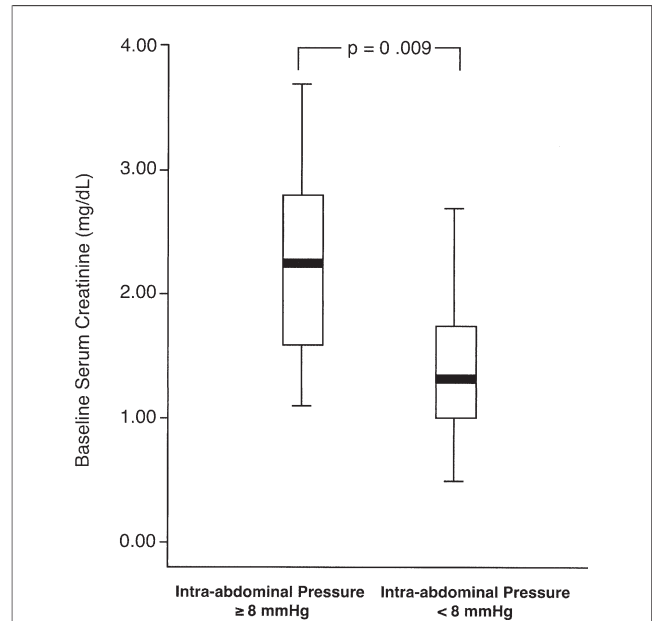


**Figure 2** Change in IAP in Patients With Baseline Elevated Intra-Abdominal Pressure

Noted overall trend of reduction in intra-abdominal pressures (IAP) in patients with IAP at  $\geq 8$  mm Hg at baseline.

normal IAP. Overall MAP, CI, PCWP, CVP, and abdominal perfusion pressure were comparable between the 2 cohorts at baseline and at follow-up. All patients had signs of impaired hemodynamics with elevated right-sided and left-sided filling pressures, which significantly improved after parenteral administration of vasodilators, diuretics, and/or inotropic therapy.

Patients with elevated baseline IAP or IAH had higher serum creatinine levels at baseline ( $2.3 \pm 1.0$  mg/dl vs.  $1.5 \pm 0.8$  mg/dl,  $p = 0.009$ ) and at follow-up ( $1.8 \pm 0.8$  mg/dl vs.  $1.3 \pm 0.9$  mg/dl,  $p = 0.04$ ) compared with those who had a normal IAP at baseline. As shown in Figure 3, IAP was related to impaired renal function. The renal filtration gradient was statistically lower at baseline in the patients with elevated IAP versus those with normal IAP ( $56 \pm 14$  mm Hg vs.  $65 \pm 10$  mm Hg,  $p = 0.03$ ). Furthermore, in those with elevated IAP at baseline, there was an average increase of renal filtration gradient from  $56 \pm 14$  mm Hg to  $64 \pm 12$  mm Hg ( $p = 0.01$ ) that paralleled with an improvement in mean GFR ( $40 \pm 21$  ml/min to  $49 \pm 23$  ml/min,  $p = 0.003$ ) as well as in serum creatinine ( $2.3 \pm 1.0$  mg/dl to  $1.8 \pm 0.8$  mg/dl,  $p = 0.01$ ).



**Figure 3** Baseline Serum Creatinine Level and IAP

Box and whisker plot with median, quartiles, and extremes for serum creatinine levels for all patients with intra-abdominal pressure (IAP)  $< 8$  mm Hg and  $\geq 8$  mm Hg at baseline. Note that there is a statistically higher serum creatinine level in patients with a higher IAP.

**Relation between changes in IAP, hemodynamic variables, and renal function.** Table 3 illustrates the relationship between changes in hemodynamic variables and renal function changes in all patients and in those with elevated IAP. Although there was a significant reduction in right-sided and left-sided filling pressures together with an improved cardiac index, these hemodynamic improvements did not correlate with improvements in renal function or IAP. Changes in IAP (either an increase or a decrease) after intensive medical therapy correlated with changes in renal function ( $r = 0.77$ ,  $p < 0.001$ ), and this only in patients with elevated IAP (Fig. 4A). Patients who had an increase in IAP at follow-up also had a deterioration of their renal

**Table 2** Hemodynamic Variables on Admission and Time of Pulmonary Artery Catheter Removal in All Patients and Stratified According to IAP  $\geq 8$  mm Hg and IAP  $< 8$  mm Hg

	All Patients (n = 40)			Patients With IAP $\geq 8$ mm Hg (n = 24)			Patients With IAP $< 8$ mm Hg (n = 16)		
	Baseline	Follow-Up	p Value	Baseline	Follow-Up	p Value	Baseline	Follow-Up	p Value
MAP (mm Hg)	75 $\pm$ 13	73 $\pm$ 11	NS	78 $\pm$ 14	75 $\pm$ 11	NS	72 $\pm$ 11	72 $\pm$ 11	NS
CVP (mm Hg)	15 $\pm$ 7	11 $\pm$ 6	$< 0.001$	16 $\pm$ 7	13 $\pm$ 7	$< 0.001$	13 $\pm$ 6	9 $\pm$ 5	0.05
PCWP (mm Hg)	22 $\pm$ 6	17 $\pm$ 4	$< 0.001$	22 $\pm$ 5	17 $\pm$ 3	$< 0.001$	23 $\pm$ 8	18 $\pm$ 5	0.03
CI (l/min/m <sup>2</sup> )	2.1 $\pm$ 0.9	2.6 $\pm$ 0.7	$< 0.001$	2.1 $\pm$ 0.7	2.7 $\pm$ 0.7	$< 0.001$	2.2 $\pm$ 1.0	2.5 $\pm$ 0.6	0.05
IAP (mm Hg)	8 $\pm$ 4	5 $\pm$ 3	$< 0.001$	10 $\pm$ 2	6 $\pm$ 3	$< 0.001$	3 $\pm$ 1	3 $\pm$ 1	NS
APP (mm Hg)	68 $\pm$ 12	69 $\pm$ 11	NS	68 $\pm$ 13	69 $\pm$ 11	NS	68 $\pm$ 11	69 $\pm$ 11	NS
FG (mm Hg)	61 $\pm$ 13	64 $\pm$ 11	NS	56 $\pm$ 14	64 $\pm$ 12	0.01	65 $\pm$ 10	66 $\pm$ 11	NS
Creatinine (mg/dl)	2.0 $\pm$ 0.9	1.6 $\pm$ 0.9	0.002	2.3 $\pm$ 1	1.8 $\pm$ 0.8	0.01	1.5 $\pm$ 0.8	1.3 $\pm$ 0.9	NS
GFR (ml/min)	50 $\pm$ 35	61 $\pm$ 44	$< 0.001$	40 $\pm$ 21	49 $\pm$ 23	0.003	63 $\pm$ 46	77 $\pm$ 58	NS

APP = abdominal perfusion pressure; CI = cardiac index; CVP = central venous pressure; FG = renal filtration gradient; IAP = intra-abdominal pressure; MAP = mean arterial pressure; PCWP = pulmonary capillary wedge pressure.



**Table 3** Relation of Changes in IAP or Serum Creatinine With Hemodynamic Variables Stratified According to All Patients and in Patients With IAP  $\geq 8$  mm Hg

	All Patients (n = 40)				Patients With IAP $\geq 8$ mm Hg (n = 24)			
	Changes in IAP		Changes in sCr		Changes in IAP		Changes in sCr	
	r	p Value	r	p Value	r	p Value	r	p Value
Changes in MAP (mm Hg)	0.17	NS	-0.26	NS	0.02	NS	-0.37	NS
Changes in CVP (mm Hg)	0.05	NS	-0.23	NS	0.16	NS	-0.09	NS
Changes in PCWP (mm Hg)	-0.01	NS	-0.23	NS	0.01	NS	-0.17	NS
Changes in CI (l/min/m <sup>2</sup> )	0.01	NS	0.08	NS	-0.03	NS	0.11	NS

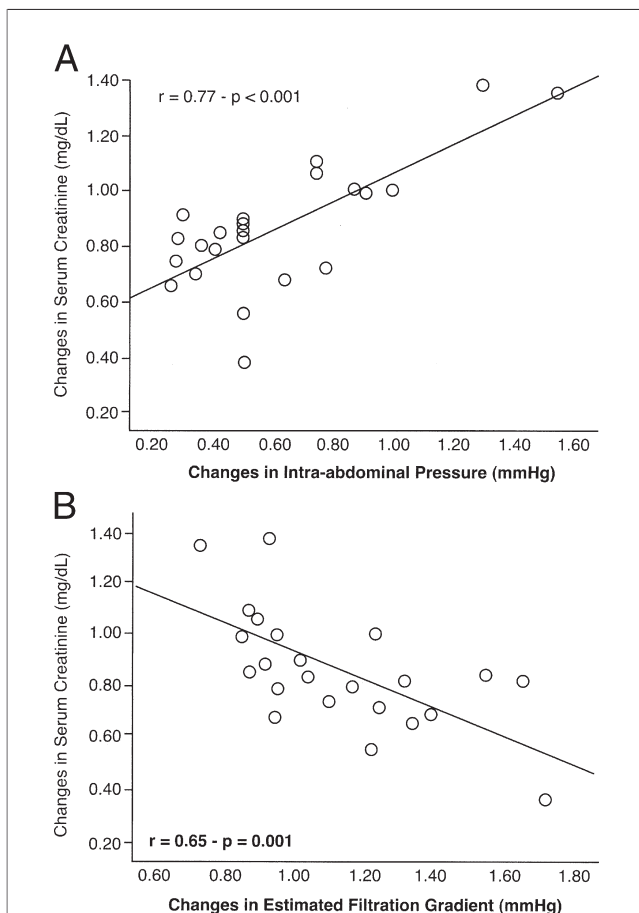
NS = nonsignificant; r = correlation coefficient; sCr = serum creatinine; other abbreviations as in Table 2.

function. The 1 patient who initially presented with a normal IAP and had an increase of IAP at follow-up (from 3 to 8 mm Hg) also showed a corresponding worsening of renal function from baseline to follow up. No differences in hemodynamic profile or therapeutic regimen were noticed between patients in whom IAP increased during treatment compared with those in whom it did not. There was a significant negative correlation between changes in FG and changes in creatinine ( $r = -0.65$ ,  $p = 0.001$ ) (Fig. 4B).

## Discussion

There are several key findings in this hypothesis-generating study. First, patients with advanced heart failure presenting with ADHF have a high prevalence of elevated IAP despite the absence of overt abdominal symptoms. Second, elevated IAP is associated with more impaired renal function. Third, improvement in renal function after medical therapy is associated with a reduction of IAP, yet bears no relationship with changes in hemodynamic measurements. Fourth, measurement of IAP is simple, safe, inexpensive, and reproducible with equipment readily available in the clinical setting. Our clinical observations raise the possibility that increased IAP might contribute, in part, to the renal dysfunction commonly observed in patients with ADHF. Although the mechanism is unclear, both reduced renal perfusion and increased renal vein pressure (and thus increased renal pressure) might be a consequence of increased IAP.

Elevated IAP among critically ill patients has predominantly been described in the surgical and critical care literature in scenarios involving abdominal catastrophes (3,4,13). As the pathophysiology of IAP becomes better understood, the importance of IAP measurements in the diagnosis and management of elevated IAP and IAH has evolved. The abdomen can be considered a closed box with both rigid (costal arch, spine, and pelvis) and flexible (abdominal wall and diaphragm) walls, thus the IAP measured at one point may be assumed to represent the IAP throughout the abdomen (14,15). However, it is important to remember that clinical judgment and physical examination are far from accurate in estimating IAP (9,16). The transvesical IAP pressure measurement depends on the wall of the bladder functioning as a transducing membrane without imparting any additional pressure from its own musculature, allowing it to act as a passive reservoir (17,18). Although a substantial number of ADHF patients present with ascites and visceral edema, both potential causes of elevated IAP, no reports in the literature have studied the prevalence and potential role of elevated IAP in ADHF patients. As shown by our study, elevated IAP in patients presenting with ADHF is common (60%), with a smaller proportion (10%) demonstrating IAH, which was not detected on routine history and physical examination. None of the patients in this study presented with

**Figure 4** Changes in Renal Parameters and Changes in IAP

Relationship between changes in renal function and changes in intra-abdominal pressure (IAP) (A) and estimated renal filtration gradient (B) in patients with IAP  $\geq 8$  mm Hg at baseline.

abdominal discomfort as a subjective sign of elevated IAP, which shows that this phenomenon is often, if not always, asymptomatic in ADHF patients. One explanation might be that the fluid build-up in these patients is often gradual over weeks, and thus the increase in IAP may also be slow and insidious. Lowering of the IAP was likely caused by mobilization of fluid from the third space through a combination of aggressive diuretic, vasodilator, and/or inotropic therapy. Successful intensive medical therapy, as evidenced by a reduction in right-sided and left-sided filling pressures together with improved cardiac output, coincided with the observed reduction in IAP in most patients. However, no correlation between changes in any hemodynamic variable or alterations in IAP was observed. This potentially explains why patients with improved hemodynamics may subsequently develop worsening renal function after aggressive therapy during ADHF admission if there is persistent elevation of IAP. Our data corroborate this hypothesis because all patients whose renal function deteriorated during treatment had an increase in IAP at follow-up, however, with improved hemodynamics.

An inadequate renal filtration gradient has been identified as a key factor in the development of renal dysfunction (19,20). Patients with an acute exacerbation of advanced heart failure often present with a low systemic blood pressure and impaired cardiac index (21). Both factors may substantially reduce renal blood flow, the most important variable of the FG in patients presenting with congestive heart failure (22,23). Consequently, even small elevations in IAP lead to significant reductions in FG. Indeed, a statistically significant reduction of the FG was noticed in the patients who presented with elevated IAP compared with the patients with normal IAP, although both groups had a comparable reduction in cardiac index and mean systemic arterial blood pressure. Moreover, changes in FG were closely correlated with changes in renal function, thereby emphasizing the importance of an adequate FG. Impaired cardiac index and increased filling pressures seen in advanced heart failure patients with ADHF will further activate the renin-angiotensin and sympathetic nervous system, reduce nitric oxide in the endothelium, and induce inflammatory mediators, thus aggravating the hypoperfusion state of the glomeruli (24).

Elevated IAP may also lead to renal vein and ureter compression, further impairing renal function (4,25,26). Although impairment of venous return probably plays a role (27), it cannot by itself completely explain the manifestations of renal function improvement after normalizing the IAP, because the reduction of right- and left-sided filling pressures was not correlated with improved renal function. Instead, elevated IAP is transdiaphragmatically transmitted and may give rise to elevation of intrathoracic pressures (4,6). This may result in elevated pulmonary pressure, CVP, and capillary wedge pressure readings from the pulmonary artery catheter. In addition, increased IAP decreases venous return by obstructing the inferior vena cava blood flow in

the abdomen, decreasing cardiac output, and increasing the risk for peripheral edema and venous thrombosis (28–30). As a result, elevated IAP in ADHF patients makes preload assessment difficult and further compromises the already-impaired left ventricle. Furthermore, measured intravascular pressures are not reflective of intravascular volumes, and inappropriate diuretic admission might increase the risk of renal function worsening.

It is important to emphasize that although the degree of renal dysfunction was correlated with the degree of elevated IAP, there was a wide range of IAPs in relation to serum creatinine levels on presentation. Our findings regarding the relationship between changes in IAP and changes in renal function are primarily focused on those with elevated IAP at baseline. These findings corroborate the hypothesis that the response of IAP to treatment rather than the absolute level of IAP on admission is contributing to improved or worsening renal function.

**Study limitations.** There are several limitations in our observational series, including the relatively small sample size, the lack of any outcomes data on renal function after hospital discharge, the lack of any urinalysis, the adoption of the technique of IAP measurement used in the surgical literature, and the lack of physiological measurements (such as direct assessment of intraparenchymal renal pressures and renal blood flow) to fully explain the complex underlying pathophysiology. However, our observations are unique, because the prevalence of increased IAP in patients without obvious abdominal symptoms in ADHF has not been previously reported. Animal data have suggested that increased intraparenchymal pressure may not contribute to renal dysfunction (5). Although clinically not apparent, we did not routinely perform an abdominal ultrasound to confirm ascites as a contributing factor to the elevated IAP. Additionally, serial measurements of abdominal girth as a potential indicator of reduction in IAP during therapy were not performed. Further studies are necessary to better understand the exact pathophysiology underlying this cardioabdominal interaction and whether there is a cause-and-effect relationship between IAP and worsening renal function. The extent to which elevated IAP contributes to the observed renal dysfunction in our ADHF population requires further investigation.

## Conclusions

In patients admitted with advanced decompensated heart failure, we observed a high prevalence of elevated intra-abdominal pressure, which is associated with impaired renal function. A strong correlation between reduction in IAP and improvement in renal function was observed, although it was independent of hemodynamic changes. Measurement of IAP in patients with ADHF should be considered to assist in the management of these patients because changes in IAP with therapy are predictive of changes in renal function.

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