One third of patients with acute decompen-
sated heart failure also develop worsening kidney
function, and the management of these patients is
very challenging. This common clinical condition is
now termed “Cardiorenal Syndrome Type 1”. In this
issue of the Journal, Omar and Zedan reviewed
the subtypes, prevalence, pathophysiology, treat-
ment, and outcomes of this complex syndrome.1
The authors should be congratulated for their effort
to compile the current data on these syndromes-
for which the best management is still a mystery.

Among the five different subtypes of cardiore-
nal syndrome, cardiologists frequently help manage
patients with Type 1. These patients usually receive
high doses of diuretics or less commonly are treated
with venovenous ultrafiltration. Current heart failure
guidelines recommend ultrafiltration in these patients
as a Class IIa recommendation.2 However, limited
data are available comparing the safety and efficacy
of ultrafiltration with diuresis in acute heart failure
complicated with cardiorenal syndrome and conges-
tion. Recently, the CARRESS-HF trial demonstrated
that the best treatment for these patients was not
ultrafiltration.3 This randomized trial included 188 pa-
tients with acute decompensated heart failure and
worsening kidney function (0.3 mg/dl increase in cre-
atinine levels). The mean age of these patients was
68 years, 75% of the patients were men, and the av-
erage creatinine was 2 mg/dl. Patients received either
ultrafiltration therapy or aggressive diuresis with a
goal of 3-5 liters of urine output per day. Patients ran-
domized to ultrafiltration had higher rates of adverse
events, mainly kidney failure, bleeding complications,
and intravenous catheter related complications, than
patients randomized to the medical treatment arm
(72% vs. 57%, p=0.03). Aggressive diuretic treat-
ment was superior to ultrafiltration in bivariate primary
endpoints, including change in weight and change in
creatinine levels 96 hours after enrollment. Fur-
thermore, ultrafiltration worsened the kidney func-
tion more frequently than medical therapy in this trial.
This investigator driven, National Heart, Lung, and
Blood Institute funded trial is published online in the

The diuretic doses used in the pharmacologic
arm of the CARRESS-HF trial need to be highlighted.
The investigators suggested the use of significantly
higher doses of intravenous loop diuretics with or
without metolazone than is usual in clinical practice.
For instance, the suggested dose for a patient who
was on less than 80 mg of oral loop diuretic daily
before the cardiorenal syndrome was 160 mg intra-
venous loop diuretic per day. If the urine output for
this particular patient was less than three liters at 24
hours, the suggested dose was 320 mg intravenous
loop diuretic and 5 mg metolazone daily. If the urine
output was still less than three liters at the next 24
hours, the subsequent step was 560 mg intravenous
loop diuretic plus 10 mg of metolazone and the con-
sideration of dopamine, dobutamine, nitroglycerine,
or nesiritide depending on the systolic blood pres-
sure, symptoms, and ejection fraction. This strategy
reminded me of the term “boluresis” which was ini-
tially thought to be harmful by increasing the crea-
tinine and further worsening the kidney function. I
suggest all clinicians treating these patients should
be aware of the details on doses in the stepped phar-
macologic care arm of CARESS-HF trial (found in the
Supplementary Appendix of the original publication).

There is urgent need to find the best strat-
ey to manage acute cardiorenal syndrome. From
the CARESS-HF trial we learn that ultrafiltration
should not be recommended as first-line, routine
treatment for patients with Type 1 cardiorenal syn-
drome. I am anticipating that the next heart fail-
ure guidelines will consider the results of this study
and downgrade the recommendations for ultrafiltra-
tion in acute decompensated heart failure patients.
REFERENCES


Author Affiliation: Texas Heart Institute at St. Luke’s Episcopal Hospital, Department of Adult Cardiology, Baylor College of Medicine, Houston, TX 77030
Author Contact Information: cevik@bcm.tmc.edu
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