Impact of Dopamine Infusion on Renal Function in Hospitalized Heart Failure Patients

Essay
Submitted for Partial Fulfillment of Master Degree
in Intensive Care

Presented By
Emad El Din Mohammed Abd El Naby
M.B.B.CH

Supervised by
Prof. Dr. Ahmed Abd El Aala El Shawarby
Professor of Anaesthesia and Intensive Care
Faculty of Medicine- Ain Shams University

Dr. Ahmed Mohammed Shafik
Assistant Professor of Anaesthesia and Intensive Care
Faculty of Medicine- Ain Shams University

Dr. Sahar Mohammed Talaat
Lecturer of Anaesthesia and Intensive Care
Faculty of Medicine- Ain Shams University

Faculty of Medicine
Ain Shams University
2013

سورة البقرة الآية: ٣٢

Acknowledgement

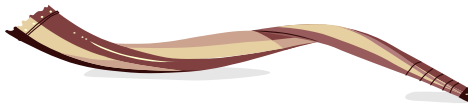
First of all, great thanks to God who enabled us to complete this work.

No words can express my deepest appreciation and profound respect to **Prof. Dr. Ahmed Abd El Aala El Shawarby,** *Professor of Anaesthesia and Intensive Care, Ain Shams University, for his continuous guidance, support and constructive criticism through the work. He has generously devoted much of his time and his effort for planning and supervision of this study.*

Also, my profound gratitude to **Dr. Ahmed Mohammed Shafik,** *Assistant Professor of Anaesthesia and Intensive Care, Ain Shams University, for his kind supervision and support. It was great honor to work under his supervision.*

Also, my profound gratitude to **Dr. Sahar Mohammed Talaat,** *Lecturer of Anaesthesia and Intensive Care, Ain Shams University for her kind supervision and support. It was great honor to work under her supervision.*

Lastly, I don't forget my father and my mother, the best helper for me, their full support, prayers and wishes were a great motive to accomplish this work. My deepest gratitude to them and thanks will never appreciate what I owe them.



Emad El Din Mohammed

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List of Abbreviations

Abb.	Meaning
AC	Adenyl cyclase
ACE	Angiotensin converting enzyme
ACEis	Angiotensin-converting enzyme inhibitors
ACRS	Acute cardiorenal syndrome
ACS	Acute coronary syndromes
ADHERE	Acute decompensated heart failure national registry
ADHF	Acute decompensated heart failure
AF	Atrial fibrillation
AHF	Acute heart failure
AKI	Acute kidney injury
AMI	Acute myocardial infarction
AR	Adrenergic receptor
ARBs	Angiotensin receptor blockers
AT II	Angiotensin II
AT1R	Angiotensin type 1 receptor
AT2R	Angiotensin type 2 receptor
BEST	Beta-blocker Evaluation of Survival Trial
BNP	Brain natriuretic peptide
BP	Blood pressure
BUN	Blood urea nitrogen
BW	Body weight
CABG	Coronary artery bypass grafting
CAD	Coronary artery disease

Abb.	Meaning
CAMP	Cyclic adenosine monophosphate
CBC	Complete blood count
CHF	Congestive heart failure
CKD	Chronic kidney disease
CK-MB	Creatine kinase MB
CO	Cardiac output
COPD	Chronic obstructive pulmonary disease
CPO	Cardiogenic Pulmonary Edema
Cr	Creatinine
CRF	Corticotrophin-releasing factor
CVP	Central venous pressure
CXR	Chest radiograph
CysC	Cystatin C
DA	Dopamine
DIG	Digitalis Investigation Group
DVT	Deep venous thrombosis
ECG	Electrocardiogram
ED	Emergency department
EF	Ejection fraction
eGFR	Estimated glomerular filtration rate
EMPHASIS	The Eplerenone in Mild Patients Hospitalization And Survival Study
EPHESUS	Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study
ERPF	Effective renal plasma flow

Abb.	Meaning
ESC	European Society of Cardiology
ESCAPE	Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness
ETT	Endotracheal tube
FF	Filtration fraction
FFAs	Free fatty acids
FIRST	Flolan International Randomized Survival trial
GFR	Glomerular filtration rate
GPCRs	G protein-coupled receptors
HDF	High-dose furosemide
HF	Heart Failure
HR	Heart rate
IABP	Intra-aortic balloon pump
IAP	Intra-abdominal pressure
ICU	Intensive care unit
IV	Intravenous
JVD	Jugular venous distention
JVP	Jugular venous pressure
KIM-1	Kidney injury molecule-1
LDFD	Low-dose furosemide combined with low-dose dopamine
L-DOPA	L-dihydroxyphenylalanine
LMWH	Low molecular weight heparin
LV	Left ventricular

Abb.	Meaning
LVEDP	Left ventricle end diastolic pressure
LVH	Left ventricle hypertrophy
MAP	Mean arterial blood pressure
MI	Myocardial infarction
MR	Mitral regurgitation
MSNs	Medium spiny neurons
NAG	N-acetyl-beta-D-glucosaminidase
NCC	Sodium chloride co-transporter
NES	Nesiritide
NGAL	Neutrophil gelatinase-associated lipocalin
NHE1	Sodium hydrogen exchanger type 1
NHE3	Sodium hydrogen exchanger type 3
NIV	Non-invasive Ventilation
NSTEMI	Non ST-segment elevation myocardial infarction
NTG	Nitroglycerin
NTP	Nitroprusside
NT-pro BNP	N-terminal probrain natriuretic peptide
NYHA	New York Heart Association classification classes for HF
PCG	Pressure in the glomerular capillary
PCI	Percutaneous coronary intervention
PCWP	Pulmonary capillary wedge pressure
PE	Pulmonary embolism
PLA2	Phospholipase A2
PLC	Phospholipase C

Abb.	Meaning
PND	Paroxysmal nocturnal dyspnea
PSF	Preserved systolic function
PTP	Proximal tubular pressure
RAAS	Renin angiotensin aldosterone system
RALES	Randomized Aldactone Evaluation Study
RARs	Rapidly adapting stretch receptors
RAS	Renin angiotensin system
RBF	Renal blood flow
ROS	Reactive oxygen species
RVR	Rapid ventricular response
RyR	Ryanodine receptor
SBP	Systolic blood pressure
SERCA	Sarcoplasmic reticulum calcium ATPase
SL	Sublingual
SNS	Sympathetic nervous system
SR	Sarcoplasmic reticulum
STEMI	ST-segment elevation myocardial infarction
SVR	Systemic vascular resistance
The UNLOAD trial	Ultrafiltration vs. Intravenous Diuretics for Patients Hospitalized with Acute Decompensated Heart Failure trial
UF	Ultrafiltration
UFH	Unfractionated heparin
VMAC	Vasodilation in the management of acute congestive HF

Abb.	Meaning
VO2	Peak oxygen consumption per unit time
VS	Vital signs
WRF	Worsening renal function

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Introduction

Acute HF is defined as “a rapid onset or change in the signs and symptoms of HF, resulting in the need for urgent therapy.” It may be either new onset HF or worsening of pre-existing HF and that cardiac dysfunction may be related to different causes, including acute coronary syndromes (ACS), valve dysfunction, arrhythmias, pericardial disease, and increased left ventricular (LV) afterload and that these different causes may interact ***(Dickstein et al., 2008).***

The majority of acute heart failure patients have worsening chronic heart failure; after initial management resulting in stabilization, they should no longer be considered acute but chronic heart failure ***(Dickstein et al., 2008).***

The most important pathophysiologic drive of heart failure is a reduction in cardiac output. This can be the consequence of decreased systolic function, impaired diastolic function, or a combination of both. The net result is the same: decreased cardiac output resulting in decreased renal perfusion ***(Dickstein et al., 2008).***

Worsening of renal function during hospitalization for acute decompensated heart failure occurs in more than one-third of hospitalized patients and is associated with prolonged hospital stay, higher in–hospital mortality, increased likelihood

of readmission, and increased mortality after discharge ***(Giamouzis et al., 2009).***

Importantly, new evidence suggests that renal failure in heart failure has a striking resemblance with chronic kidney disease in primary renal disease and not only includes functional changes such as decreased GFR, decreased erythropoietin production, calcium- phosphate metabolism disturbances, chronic inflammation, and vitamin D deficiency but also glomerular and tubular damage ***(Belonje et al., 2010).***

In recent years nephrologists and cardiologists have worked together in further elucidating this frequently occurring condition now termed as cardiorenal syndrome ***(Ronco et al., 2010).***

Both reduced renal perfusion and increased renal Venous pressure represent the hemodynamic interaction of reduced GRF in the cardiorenal syndrome in heart failure. This highlights the observation that the pivotal player in the pathophysiology of cardiorenal failure remains impaired hemodynamics, but that there are other so-called cardiorenal connectors such as renin angiotensin aldosterone system activity, inflammation, nitric oxide balance, sympathetic nervous system activity, reactive oxygen species and the presence of diabetes and hypertension that may modulate this