Errata

In the article "Renal and Hormonal Effects of Systemic Nitric Oxide Inhibition in Patients With Congestive Heart Failure and in Healthy Control Subjects" by Bech et al (J Card Fail 2013;19:776-785), a number of standard deviations were cited incorrectly throughout the article. Because these errors appear throughout the article, the full and corrected version of the article has been posted online along with this erratum.

DOI of original article: http://dx.doi.org/10.1016/j.cardfail.2013.10.001
http://dx.doi.org/10.1016/j.cardfail.2014.04.003

1071-9164/$ - see front matter

Renal and Hormonal Effects of Systemic Nitric Oxide Inhibition in Patients With Congestive Heart Failure and in Healthy Control Subjects

J.N. BECH, MD, PhD, J. STARKLINT, MD, PhD, H. BENTZEN, MD, PhD, O. NYVAD, MD, AND E.B. PEDERSEN, MD, DrSci

Holstebro, Denmark

ABSTRACT

Background: The significance of basal renal nitric oxide (NO) availability in the regulation of renal perfusion and sodium excretion in human congestive heart failure (CHF) has not been described previously.

Methods and Results: We studied the effects of acute systemic NO synthesis inhibition with N^G-monomethyl-L-arginine (L-NMMA) in 12 patients with CHF and 10 healthy control subjects (CON) in a randomized placebo-controlled study. Effect parameters were renal plasma flow (RPF), renal vascular resistance (RVR), glomerular filtration rate (GFR), urine sodium excretion and plasma levels of vasoactive hormones. L-NMMA was associated with a significant decrease in RPF (CON-LNMMA: $-13 \pm 5\%$ [$P = .014$]; CHF-LNMMA: $-17 \pm 6\%$ [$P = .017$]) and a profound increase in RVR in both CHF and CON (CON-LNMMA: $+26 \pm 7\%$ [$P = .009$]; CHF-LNMMA: $+37 \pm 10\%$ [$P = .005$]). Significant decreases in sodium excretion were found in both CHF-LNMMA and CON-LNMMA. Relative changes from baseline were not statistically different between CHF-LNMMA and CON-LNMMA. After L-NMMA, RPF values correlated inversely with plasma aldosterone in CHF-LNMMA ($P = .01$). L-NMMA induced an increase in A-type natriuretic peptide (ANP) only in CHF-LNMMA ($+18 \pm 6\%$; $P = .035$), which correlated significantly with basal ANP levels ($P = .034$).

Conclusions: There was no difference in the renal response to L-NMMA in CHF vs CON, suggesting that the impact of NO on renal perfusion and sodium excretion is maintained in stable CHF. We suggest that NO influences the release of ANP during high levels of atrial stretch in CHF. (*J Cardiac Fail 2014;20:376.e15–376.e24*)

Key Words: Nitric oxide, N^G-monomethyl-L-arginine, renal plasma flow, ANP, aldosterone.

Congestive heart failure (CHF) is generally accompanied by increased peripheral resistance and increased sodium and water retention by the kidneys. Patients with CHF usually develop a disproportionate decrease in renal blood flow and glomerular filtration rate (GFR), leading to increased renal vascular resistance (RVR) and increased filtration fraction.[1] Renal failure may develop due to severely compromised tissue perfusion and intense neurohormonal activation, including increased activity of the renin-angiotensin system, endothelin-1, and the sympathetic nervous system. The presence of severe sodium and water retention and overt renal failure during CHF is associated with a poor prognosis regarding survival.[2] Nitric oxide (NO) is an important endothelium-derived vasodilator which exerts a continuous vasodilating tone in the systemic and renal circulations.[3] In the kidneys, NO is a strong promoter of natriuresis by acting on a number of natriuretic mechanisms.[3] The activity and relative influence of the L-arginine (L-arg)–NO pathway during CHF has therefore attracted considerable interest, because decreased NO availability could be of pathophysiologic significance to the vascular and renal derangements observed during CHF.

A large number of studies of the peripheral vasculature in animals and humans have demonstrated impaired endothelial function during CHF thought to be caused by decreased NO availability.[4,5] However, other studies are in disagreement with this.[6] Regarding renal NO activity during CHF,

From the Department of Medical Research, Holstebro Hospital, Holstebro, Denmark and Aarhus University, Aarhus, Denmark.

Manuscript received July 12, 2013; revised manuscript received September 10, 2013; revised manuscript accepted October 2, 2013.

Reprint requests: J.N. Bech, MD, PhD, Dept of Medical Research, Holstebro Hospital, DK-7500 Holstebro, Denmark. Tel: +45 78436787. E-mail: jnbech@dadlnet.dk

See page 376.e23 for disclosure information.

1071-9164/$ - see front matter

http://dx.doi.org/10.1016/j.cardfail.2013.10.001