analyses were performed using the Student
analysis was used to determine the relationship between continu-
tion. Sixty patients were divided randomly into two groups that
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After one month, blood samples for measuring plasma ALD
were collected simultaneously from the aortic root (AO) and
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The subjects were 60 consecutive patients with CHF (left
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After one month, blood samples for measuring plasma ALD
were collected simultaneously from the aortic root (AO) and
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were performed as previously reported (4,5).

All results are expressed as mean values ± SEM. Univariate
analyses were performed using the Student t test. Linear regression
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uous variables. A p value < 0.05 was regarded as significant.

Table 1. Clinical Characteristics of Patients in the Furosemide and Torasemide Groups at Randomization

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ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; NS = not significant; NYHA = New York Heart Association.

There was no difference in patient characteristics between the
two groups (Table 1). In the torasemide group, the mean dose of
torasemide was 10.1 ± 0.7 mg/day (range, 4 to 16 mg/day).

One month after randomization, there were no differences in
hemodynamic parameters. In the furosemide group, the plasma ALD
level in the CS was significantly lower than that in the AO (73.1 ± 10.0 pg/ml vs. 56.9 ± 6.5 pg/ml; p < 0.001). In contrast, there was
no difference in plasma ALD levels between the AO and the CS in
the torasemide group (85.4 ± 10.5 pg/ml vs. 83.1 ± 11.6 pg/ml) (Fig.
1). Plasma procollagen type III aminoterminal peptide in the CS
concentration was significantly lower in the torasemide group than
that in the furosemide group (0.52 ± 0.03 U/ml vs. 0.67 ± 0.06
U/ml; p < 0.05). There was a significant negative correlation between
the dose of torasemide and the transcardiac gradient (AO–CS) of
ALD in the torasemide group (r = −0.56, p < 0.01).

We evaluated the transcardiac extraction of ALD as a potential
marker of ALD action in the heart (4,5) in patients with CHF. In
the furosemide group, the plasma ALD level in the CS was
significantly lower than that in the AO. In contrast, there was
no difference in plasma ALD level between the AO and the CS in
the torasemide group. The transcardiac gradient (AO–CS) of ALD
and the extraction ratio of ALD in the AO were significantly lower
in the torasemide group than those in the furosemide group.

Plasma procollagen type III aminoterminal peptide concentration,
a biochemical marker of fibrosis, was significantly lower in the
torasemide group than in the furosemide group. These findings
may indicate that unlike furosemide, torasemide has an ALD
receptor antagonist in the heart. Experimental studies indicate that
torasemide inhibits the binding of ALD to its receptor in the
cytoplasmic fraction of rat kidney (2,3). Moreover, the serum
potassium level was significantly higher in the torasemide group
than in the furosemide group in a large series (i.e., TORIC study).

Torasemide Inhibits Transcardiac Extraction of Aldosterone in Patients
With Congestive Heart Failure

To the Editor: Loop diuretics such as furosemide and torasemide
are important for the symptomatic treatment of congestive heart
failure (CHF). Recently, in the TORasemide In Congestive Heart
Failure (TORIC) study, torasemide had a more beneficial effect on
the mortality and morbidity of patients with CHF than furosemide
(1). The mechanism by which torasemide provides a greater
benefit than furosemide remains unknown, but experimental
studies indicate that torasemide may inhibit the binding of
aldosterone (ALD) to its receptor in the cytoplastic fraction of the
rat kidney (2,3). Therefore, in the present study, we evaluated the
transcardiac extraction of ALD, as a potential marker of ALD
action in the heart (4,5), in torasemide therapy compared with
furosemide therapy in the treatment of patients with CHF.

The subjects were 60 consecutive patients with CHF (left
ventricular ejection fraction <45%). Patients receiving spironolac-
tone were excluded. Informed consent was obtained from all
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Taken together with our results, the mechanism of the beneficial effect of torasemide in comparison with those of furosemide in the TORIC study may be due to the presence of mineralocorticoid receptor blocker in torasemide.

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Letters to the Editor

Depression and Heart Failure

We read with great interest the recent study by Gottlieb et al. (1) and its related editorial (2) about the important problem of depression in outpatients with heart failure (HF) evaluating its prevalence and the impact of age, race, and gender. We would like to add two comments.

First, we believe that the duration of evolution of HF symptoms is a simple and important parameter that should have been taken into account. A depression is likely to occur with time when the patient realizes the true chronic aspect of its disease, its severity, and the overall limitation in physical activity and well-being. The number of hospitalizations across the years may also play a role, and these aspects of the disease might have been included in the prediction model of Gottlieb et al. (1).

Second, the diagnosis of depression should particularly be addressed when one considers the implantation of a cardioverter-defibrillator (ICD). Although the device is usually well accepted despite fear of being shocked, patients with an ICD have high levels of anxiety and depressive symptoms (3). The ICD shocks might have the potential to cause psychological disorders. This may become a relatively frequent problem considering the preliminary results of the Sudden Cardiac Death-Heart Failure Trial (SCD-HeFT) in which treatment with an ICD compared with placebo and amiodarone was associated with a reduction in all-cause mortality in a large population of patients with heart failure of all etiologies and with a left ventricular ejection fraction <35% (4).

Thus, considering the potential association among ICD placement, electrical shock from the ICD, and depression, we should probably collect information regarding this association in the evaluation of such patients.

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