Fragmented QRS and myocardial performance index in nephrotic syndrome

To the Editor,

The recent report on "Fragmented QRS and myocardial performance index in nephrotic syndrome" published is very interesting (1). Özkan et al. (1) concluded that the "determination of fQRS in patients with nephrotic syndrome (NS) in surface ECG, an easily accessible technique, can be used as a parameter in the prediction of myocardial functions." In fact, it is already known that "fQRS may be useful in identifying patients at higher cardiac risk with larger areas of ischemic jeopardized or necrotic myocardium (2)." Hence, the similar finding among patients with NS is not a surprising finding. Nevertheless, an important factor to be concerned in the patient with NS is the medication. In the case of long-term use of steroid, the effect on the QRS can be expected (3), and this might decrease the utility of fragmented QRS detection. In the present report (1), the use of steroid is not mentioned well, and its effect on the diagnostic property of fragmented QRS is an interesting issue to be discussed.

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Author`s Reply

To the Editor,

We evaluated the presence of fQRS in patients with nephrotic syndrome and the relation between fQRS and myocardial functions in our study and showed an association between the presence of fQRS and myocardial performance in this patient group in published August 2014 in The Anatolian Journal of Cardiology (1). In addition, we determined that the presence of fQRS is significantly correlated with proteinuria. The demographic data for patients with or without fQRS are shown in Table 1. This also includes steroid use and other immunosuppressive drugs. As Table 1 shows, the only significant difference in demographic and laboratory parameters was between proteinuria levels, while there was no difference in terms of presence of fQRS in patients using steroids. Since there was no significant difference, the effect of the use of cyclosporine (2), which has been shown to affect myocardial functions and steroid use were not included in the discussion in order to avoid confusion.

Various studies have assessed the use of steroids and particularly long-term use on myocardial functions. One such study by Sali et al. (3) showed that continuous administration of prednisone to mdx mice initially improves skeletal muscle strength, but further therapy results in deterioration of muscle strength and cardiac function, associated with enhanced cardiac fibrosis. Another study was cited by the authors (4). However, to the best of our knowledge, there are no studies showing an association between the presence of fQRS and long-term steroid use.

In conclusion, levels of steroid use in patients with or without fQRS are given in the table, and no significant difference was determined.

This subject was therefore not included in the discussion.

In light of our patient numbers, we do not think it would be right to make any deductions on this subject.

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Contrast nephropathy in patients with well-preserved renal function

To the Editor,

We read with great interest the article by Yıldız et al. (1), "entitled "Relationship between brain natriuretic peptide, microalbuminuria, and contrast-induced nephropathy in patients with acute coronary syndrome," published in the September issue of The Anatolian Journal of Cardiology 2014; 14: 505-10, investigating the relationship among contrast-induced nephropathy (CIN), microalbuminuria, and brain-natri-

uretic peptide (BNP). Their main finding was that the incidence of CIN was not significantly different between patients with microalbuminuria and those without (4 out of 25 patients vs 26 out of 145 patients, p=NS). Among patients with microalbuminuria, the level of microalbuminuria was not different between those who developed CIN and those who did not. In addition, among those without microalbuminuria, the level of BNP was not different between patients with CIN and those without it. Although the authors did not express the mean creatinine level of the overall population, we can estimate from the data that it was around 0.9 mg/dL, with an approximate range of 0.1 to 1.7 mg/dL. Approximately 17% of the patients developed CIN, which is very high, considering the relatively well-preserved renal function of the study population. This is because of the definition of CIN. The application of a 25% or 0.5-mg/dL increase in serum creatinine for the definition of CIN in patients with well-preserved function is vague. For example, a patient with a baseline creatinine of 1.0 mg/dL is considered to have CIN if he had a creatinine level of 1.5 mg/dL after contrast administration. Another patient with a baseline creatinine level of 0.7 mg/dL is also denoted to have CIN if he had a post-contrast creatinine level of 0.9 mg/dL. These two patients are in the same basket of CIN. Thus, it is critical to consider this when reaching a conclusion from a study. We suggest an acute contrast-agent-induced reduction in renal function as an increase in serum creatinine concentration of at least 0.5 mg per deciliter after administration of the contrast agent in patients with relatively well-preserved renal function (2-5). Such an increase may be important, because it can increase the duration of hospitalization (2).

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Author`s Reply

To the Editor,

We would like to thank the authors of the letter for their interest and criticism about our study, published in the September issue of The Anatolian Journal of Cardiology 2014; 14: 505-10 (1). They suggested an acute contrast-agent-induced reduction in renal function as an increase in serum creatinine concentration of at least 0.5 mg per deciliter after administration of the contrast agent, instead of a 25% increase in serum creatinine concentration from the baseline value, in patients with relatively well-preserved renal function. This recommendation seems to be sensible at first glance but in fact can actually lead to misinterpretations.

Contrast-induced nephropathy (CIN) is most commonly defined as acute renal failure occurring within 48 hours after exposure to intravascular radiographic contrast material that is not attributable to other causes (2). Ideally, the impairment of renal function should be measured by serial creatinine clearance, but because this step may not be practical or cost-effective in many centers, most of the literature describes the use of isolated measurements of serum creatinine levels, even though this parameter may be less sensitive in reflecting subtle early changes in renal function and may be slower to reach maximal sensitivity than creatinine clearance.

The rate of incidence of contrast-induced nephropathy as a complication of radiographic diagnostic and interventional studies varies markedly, depending on the definition used and on other variables, such as the type of radiology procedure performed, the dose and type of contrast agent administered, the differing patient populations in regard to number and type of risk factors, and the length of patient follow-up. An overall incidence of 14.5% was recently quoted in a large epidemiologic study, which is close to the rate in our study (defined as >25% increase in serum creatinine levels over baseline in the first 5 days), but rates may vary from 0% to 90%, depending on the presence of risk factors, most notably chronic renal insufficiency, diabetes mellitus, and high contrast volume administered (3-5). In our study, approximately 17% of the patients developed CIN as you marked and considered very high in the relatively well-preserved renal function of the study population. Baseline creatinine level is very important, as you indicated, but it is not the only factor that facilitates the development of contrast nephropathy.

Our study population was a heterogeneous group that consisted of patients with different diagnoses; for example, the study population had 74 diabetic patients. The incidence of CIN among patients with diabetes has been reported to be 9%-40% (3). Also, our study population was under the stress of ACS, in contrast to patients who had a diagnostic angiography, and most of our study patients had a coronary intervention, which extends the duration of coronary angiography with the usage of more contrast media compared to diagnostic coronary angiography.

In summary, even apparently small decreases in renal function can lead to excessive mortality rates, independent of other risk factors, given that small rises in serum creatinine levels actually represent a significant decline in GFR. So, in light of this fact, contrast-induced nephropathy has become most commonly defined as "a 25% increase in serum creatinine concentration from the baseline value or an absolute increase of at least 0.5 mg/dL (44.2 µmol/L), which appears within 48 hours after the administration of radiographic contrast media and is maintained for 2-5 days" (5).

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Additional diagnostic parameter for coronary artery disease during exercise test: Heart rate recovery

To the Editor,

We read with great interest the manuscript written by Akyüz et al. (1), entitled "Heart rate recovery may predict the presence of coronary artery disease," in the June issue of Anatolian Journal of Cardiology 2014;14:351-6. In that study, they investigated whether post-exercise first-minute abnormal heart rate recovery (HRR1) helps to predict the presence and severity of coronary artery disease (CAD) (1). They found that abnormal HRR, which was defined as ≤21 beats in the sitting position during the first minute of the recovery period, had moderate sensitivity and low specificity for predicting the presence of CAD. However, abnormal HRR was not predictive of the severity of CAD.

HRR after graded exercise is one of the commonly used parameters to reflect autonomic activity. Abnormal HRR might be attributable to a defect in sympathetic withdrawal, parasympathetic reactivation, or both. Because these changes correlate with an increased risk of death, it has been hypothesized that an abnormal HRR would similarly predict increased mortality. Chaitman et al. (2) showed that the mechanism of increased mortality associated with abnormal HRR might be related more to autonomic dysfunction than to the presence or extent of CAD. On the other hand, Kizilbash et al. (3) suggested that blunted HRR was associated with several risk factors of CAD. In addition, Gera et al. (4) found that abnormal HRR was also associated with a high prevalence of CAD, left ventricular dysfunction, and composite high-risk myocardial perfusion imaging findings. In concordance with the basic findings of the study by Akyüz et al. (1), they also suggested that abnormal HRR alone, noted on stress testing, might warrant further evaluation for suspected CAD. When this relationship of abnormal HRR with CAD is taken in an opposite way, there are studies supporting this relationship. It has been shown that various programs that have been performed to control underlying CAD or rehabilitation of a CAD patient improve HRR. Tsai et al. (5) found that patients who were enrolled in a cardiac rehabilitation program after undergoing coronary artery bypass graft surgery had significantly higher HRR values compared to the control group.

In conclusion, although HRR and CAD prediction are and will further be a topic of hot debate, such an index, which can very easily be obtained during exercise stress test, can be used as a diagnostic parameter, in addition to the more commonly used parameters, including ST-segment depression, typical chest pain, or hypotensive response.

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To the Editor,

We would like to thank the authors for their comments on our original investigation published in the Anatolian Journal of Cardiology 20142014;14:351-6. (1). We defined abnormal heart rate recovery (HRR)